

CONTEMPORARY NEUROPSYCHOLOGY SERIES

# Neuropsychological Rehabilitation

EDITORS

Chad A. Noggle  
Raymond S. Dean

ASSOCIATE EDITOR

Mark T. Barisa

# Neuropsychological Rehabilitation

## ***About the Editors***

**Chad A. Noggle, PhD, ABN**, is an Assistant Professor of Clinical Psychiatry and Chief of the Division of Behavioral and Psychosocial Oncology at Southern Illinois University–School of Medicine. He previously served as an Assistant Professor at both Ball State University and Middle Tennessee State University. Dr. Noggle holds a BA in Psychology from the University of Illinois at Springfield and completed his MA and PhD at Ball State University with specialization in Clinical Neuropsychology. He completed a 2-year postdoctoral residency at the Indiana Neuroscience Institute at St. Vincent’s Hospital with specialization in Pediatric and Adult/ Geriatric Neuropsychology. To date, Dr. Noggle has published more than 300 articles, book chapters, encyclopedia entries, and research abstracts and has made over 100 presentations at national and international conferences in Neuropsychology. He served as the lead editor of *The Encyclopedia of Neuropsychological Disorders* and both *The Neuropsychology of Psychopathology* and *The Neuropsychology of Cancer and Oncology*, the latter two representing additional volumes of the Contemporary Neuropsychology Series. He currently serves as a reviewer for a number of Neuropsychology journals and is a member of the Editorial Board for Applied Neuropsychology-Adult and Applied Neuropsychology-Child. Dr. Noggle is a Diplomate of the American Board of Professional Neuropsychology and a professional member of the American Psychological Association (APA; Divisions 5, 22, 38, 40), the National Academy of Neuropsychology, and the International Neuropsychological Society. He is also a Fellow of the American College of Professional Neuropsychology. Dr. Noggle is a licensed psychologist in both Illinois and Indiana. His research interests focus on both adult and pediatric populations, spanning psychiatric illnesses, dementia, PDDs, and neuromedical disorders.

**Raymond S. Dean, PhD, ABPP, ABN, ABPdN**, holds a BA degree in Psychology (magna cum laude) and an MS degree in Research and Psychometrics from the State University of New York at Albany. As a Parachek-Frazier Research Fellow, he completed a PhD in School/ Child Clinical Psychology at Arizona State University in 1978. Dr. Dean completed an internship focused on Neuropsychology at the Arizona Neuropsychiatric Hospital and postdoctoral work at the University of Wisconsin at Madison. Since his doctoral degree he has served in a number of positions and has been recognized for his work. From 1978–1980, Dr. Dean was an Assistant Professor and Director of the Child Clinic at the University of Wisconsin at Madison. During this time he was awarded the Lightner Witmer Award by the School Psychology Division of the American Psychological Association. From 1980–1981, he served as Assistant Professor of Psychological Services at the University of North Carolina at Chapel Hill. From 1981–1984, Dr. Dean served as Assistant Professor of Medical Psychology and Director of the Neuropsychology Internship at Washington University School of Medicine in St. Louis. During this same time, Dr. Dean received both the Outstanding Contribution Award from the National Academy of Neuropsychology and the Early Contribution Award by Division 15 of the APA. He was named the George and Frances Ball Distinguished Professor of Neuropsychology and Director of the Neuropsychology Laboratory at Ball State University and has served in this position since 1984. In addition, Dr. Dean served as Distinguished Visiting Faculty at the Staff College of the NIMH. Dr. Dean is a Diplomate of the American Board of Professional Psychology, the American Board of Professional Neuropsychology, and the American Board of Pediatric Neuropsychology. He is a Fellow of the American Psychological Association (Divisions: Clinical, Educational, School and Clinical Neuropsychology), the National Academy of Neuropsychology, and the American Psychopathological Association. Dr. Dean is a Past President of the Clinical Neuropsychology Division of the APA and the National Academy of Neuropsychology. He also served as Editor of the *Archives of Clinical Neuropsychology*, *Journal of School Psychology* and the *Bulletin of the National Academy of Neuropsychology*. Dr. Dean has published some 600 research articles, books, chapters and tests. For his work he has been recognized by awards from the National Academy of Neuropsychology, the

*Journal of School Psychology*, and the Clinical Neuropsychology Division of APA.

**Mark T. Barisa, PhD, ABPP**, is the Director of Neuropsychology Services at the Baylor Institute for Rehabilitation in Dallas, TX. He is a Diplomate of the American Board of Professional Psychology in Clinical Neuropsychology and is active in numerous professional organizations including AACN, NAN, and APA (Divisions 40 and 22). He has worked in a variety of clinical settings, including administrative positions, while maintaining teaching and training activities for predoctoral interns and postdoctoral residents. He is active in research and presents regularly at local, national, and international conferences on various topics related to Neuropsychology, Rehabilitation Psychology, and the business aspects of psychological practice. Dr. Barisa is the author of the text, *The Business of Neuropsychology: A Practical Guide* (2010). His research and clinical interests have focused on outcome measurement in clinical practice, differential diagnosis of dementia, functional correlates of neuropsychological data, and return-to-work issues following injury/illness.

.....  
CONTEMPORARY NEUROPSYCHOLOGY SERIES

# Neuropsychological Rehabilitation

## EDITORS

**Chad A. Noggle, PhD, ABN**

**Raymond S. Dean, PhD, ABPP, ABN, ABPdN**

## ASSOCIATE EDITOR

**Mark T. Barisa, PhD, ABPP**

  
SPRINGER PUBLISHING COMPANY  
NEW YORK

Copyright © 2013 Springer Publishing Company, LLC

All rights reserved.

No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without the prior permission of Springer Publishing Company, LLC, or authorization through payment of the appropriate fees to the Copyright Clearance Center, Inc., 222 Rosewood Drive, Danvers, MA 01923, 978-750-8400, fax 978-646-8600, [info@copyright.com](mailto:info@copyright.com) or on the Web at [www.copyright.com](http://www.copyright.com).

Springer Publishing Company, LLC

## 11 West 42nd Street

New York, NY 10036

[www.springerpub.com](http://www.springerpub.com)

*Acquisitions Editor:* Nancy S. Hale *Production Editor:* Joseph Stubenrauch *Composition:* Newgen Imaging

*ISBN:* 978-0-8261-0714-5

*e-book ISBN:* 978-0-82610715-2

13 14 15 / 5 4 3 2 1

The author and the publisher of this Work have made every effort to use sources believed to be reliable to provide information that is accurate and compatible with the standards generally accepted at the time of publication. The author and publisher shall not be liable for any special, consequential, or exemplary damages resulting, in whole or in part, from the readers' use of, or reliance on, the information contained in this book. The publisher has no responsibility for the persistence or accuracy of URLs for external or third-party Internet websites referred to in this publication and does not guarantee that any content on such websites is, or will remain, accurate or appropriate.

Library of Congress Cataloging-in-Publication Data Neuropsychological rehabilitation / editors, Chad A. Noggle, Raymond S. Dean ; associate editor, Mark T. Barisa.

p. ; cm.

Includes bibliographical references and index.

ISBN 978-0-8261-0714-5—ISBN 978-0-82610715-2 (e-book) I. Noggle, Chad A., editor of compilation. II. Dean, Raymond S., editor of compilation. III. Barisa, Mark T., editor of compilation.

[DNLM: 1. Brain Injuries—rehabilitation. 2. Brain Injuries—psychology. 3. Neurobehavioral Manifestations. 4. Recovery of Function. WL 354]

617.4'81044--dc23

2013000628

Special discounts on bulk quantities of our books are available to corporations, professional associations, pharmaceutical companies, health care organizations, and other qualifying groups. If you are interested in a custom book, including chapters from more than one of our titles, we can provide that

service as well.

**For details, please contact:**

Special Sales Department, Springer Publishing Company, LLC

11 West 42nd Street, 15th Floor, New York, NY 10036-8002

Phone: 877-687-7476 or 212-431-4370; Fax: 212-941-7842

E-mail: [sales@springerpub.com](mailto:sales@springerpub.com)

Printed in the United States of America by Bang Printing.

*To my wife and children for supporting me through yet another book. Your constant love and encouragement always sees me through—CAN*

*To my children with all my heart—RSD*

*To my wife, Mary, and my sons Brian and Zachary for their inspiration to try harder and reach higher—MTB*





## Contents

*Contributors*

*Preface*

Acknowledgments

Chapter 1: Historical Principles and Foundations of Neuropsychological Rehabilitation

*Karen K. Brewer-Mixon and C. Munro Cullum*

Chapter 2: Neurological Recovery and Plasticity

*Javan Horwitz and Natalie Horwitz*

Chapter 3: Utilizing a Developmental Perspective: The Influence of Age and Maturation on Approach

*Justin J. Boseck, Christopher M. McCormick, and Chad A. Noggle*

Chapter 4: Rehabilitation of Attention and Executive Function Deficits

*Kaaren Bekken and L. Lynn LeSueur*

Chapter 5: Rehabilitation of Aphasia

*Mark T. Barisa, Chad A. Noggle, David B. Salisbury, Katherine McKenzie Meredith, and Justin J. Boseck*

Chapter 6: Rehabilitation of Memory Deficits

*Renée R. Lajiness-O'Neill, Laszlo A. Erdodi, Alfred Mansour, and Amy Olszewski*

Chapter 7: Rehabilitation of Visuospatial Deficits

*Justin H. Ory, Mandi Wilkes-Musso, Alyse Ann Barker, Daniel Proto, and Wm. Drew Gouvier*

Chapter 8: Educational and Occupational Rehabilitation and Intervention

*Sarah C. Connolly, Eric E. Pierson, and Chad A. Noggle*

Chapter 9: Rehabilitation in Traumatic Brain Injury

*Carrie-Ann H. Strong and Jacobus Donders*

Chapter 10: Rehabilitation in Stroke

*Rene Hernandez-Cardenache and Douglas Johnson-Greene*

Chapter 11: Cognitive Rehabilitation Following Neurosurgical Intervention

*F. Scott Winstanley and Sherry T. Thrasher*

Chapter 12: Neuropsychological Assessment in Rehabilitation

*Jennifer P. Edidin and Scott J. Hunter*

Chapter 13: Utilization of Neuroimaging in Rehabilitation

*Shawn D. Gale and Ramona O. Hopkins*

Chapter 14: System and Family Support

*Margaret Semrud-Clikeman, Jesse Bledsoe, and Lisa Vroman*

Chapter 15: Practitioner Traits in Neurorehabilitation

*Pamela S. Klonoff*

Chapter 16: Transitioning in Neurorehabilitation

*Susan M. Rumble*

Chapter 17: Cross-Specialty Collaboration in Neuropsychological Rehabilitation

*Therese Meyer-Cox and Christine V. Paradee*

Chapter 18: Rehabilitative Psychopharmacology

*Anya Mazur-Mosiewicz and Chad A. Noggle*

Chapter 19: Telerehabilitation and Teleneuropsychology: Emerging Practices  
*Michael McCue and C. Munro Cullum*

*Index*



## Contributors

**Mark T. Barisa, PhD, ABPP**

Board Certified Clinical Neuropsychologist Department of Neuropsychology Baylor Institute of Rehabilitation Dallas, TX

**Alyse Ann Barker, MA**

Graduate Student  
Department of Psychology  
Louisiana State University  
Baton Rouge, LA

**Kaaren Bekken, PhD**

Clinical Neuropsychologist  
Private Practice  
Boston, MA

**Jesse Bledsoe, PhD**

Postdoctoral Fellow  
Department of Psychiatry  
Children's Hospital of Seattle Seattle, WA

**Justin J. Boseck, PhD**

Clinical Neuropsychology Resident Trinity Health Care Systems  
Minot, ND

**Karen K. Brewer-Mixon, PhD**

Assistant Professor in Psychiatry Department of Psychiatry  
University of Texas Southwestern Medical Center Dallas, TX

**Sarah C. Connolly, MA**

Graduate Student  
Department of Educational Psychology Ball State University  
Muncie, IN

**C. Munro Cullum, PhD**

Professor of Psychiatry and Neurology & Neurotherapeutics Department of Psychiatry  
University of Texas Southwestern Medical Center Dallas, TX

**Jacobus Donders, PhD, ABPP**

Chief Psychologist  
Department of Psychology

Mary Free Bed Rehabilitation Hospital Grand Rapids, MI

**Jennifer P. Edidin, PhD**

Evaluation Center for Learning Northfield, IL

**Laszlo A. Erdodi, PhD, MS**

Postdoctoral Fellow in Clinical Neuropsychology Department of Psychiatry  
Geisel School of Medicine/Dartmouth College Hanover, NH

**Shawn D. Gale, PhD**

Associate Professor

Department of Psychology and Neuroscience Center Brigham Young University  
Provo, Utah **Wm. Drew Gouvier, PhD**

Professor of Psychology

Department of Psychology

Louisiana State University

Baton Rouge, LA **Rene Hernandez-Cardenache, PsyD**

Clinical Neuropsychologist

Assistant Professor of Psychiatry and Neuropsychology Department of Psychiatry and Behavioral Sciences

Miller School of Medicine, University of Miami Miami, FL

**Ramona O. Hopkins, PhD**

Professor

Psychology Department and Neuroscience Center Brigham Young University

Provo, Utah **Javan Horwitz, PsyD**

Clinical Neuropsychologist

Department of Extended Care & Rehabilitation VA Northern Indiana Health Care System Marion, IN

**Natalie Horwitz, MA**

Carmel Neuropsychology Services, P.C.

Carmel, IN

**Scott J. Hunter, PhD**

Associate Professor of Psychiatry & Behavioral Neuroscience Associate Professor of Pediatrics Director of  
Neuropsychology

The University of Chicago

Department of Psychiatry & Behavioral Neuroscience—Section of Child & Adolescent Psychiatry Chicago,  
IL

**Douglas Johnson-Greene, PhD, MPH, ABPP**

Board Certified Clinical Neuropsychologist Director Rehab & Neuropsychology Associate Vice-Chair

Department of Rehabilitation Medicine Miller School of Medicine, University of Miami Miami, FL

**Pamela S. Klonoff, PhD, ABPP**

Clinical Director and Clinical Neuropsychologist Center for Transitional NeuroRehabilitation Barrow

Neurological Institute St. Joseph's Hospital and Medical Center Phoenix, AZ

**Renée R. Lajiness-O'Neill, PhD**

Clinical Neuropsychologist

Clinical Associate Professor Department of Psychiatry, Neuropsychology Section University of Michigan

Health Systems Ann Arbor, MI **L. Lynn LeSueur, PhD**

Clinical Neuropsychologist

Private Practice

Salem, MA **Alfred Mansour, PhD**

Doctoral Fellow

Department of Psychology

Eastern Michigan University

Ypsilanti, MI **Anya Mazur-Mosiewicz, PhD**

Neuropsychology Resident

The Children's Hospital

University of Calgary  
Calgary, Alberta, Canada **Christopher M. McCormick, MA** Graduate Student

Department of Educational Psychology Ball State University  
Muncie, IN

**Michael McCue, PhD**

Associate Professor

Department of Rehabilitation Science and Technology University of Pittsburgh—School of Health and  
Rehabilitation Sciences Pittsburgh, PA **Katherine McKenzie Meredith, PsyD**

Clinical Neuropsychologist

Baylor Institute for Rehabilitation Dallas, TX

**Therese Meyer-Cox, PhD, ABPP**

Master Psychologist

Health Psychology Department Memorial Medical Center  
Springfield, IL

**Chad A. Noggle, PhD, ABN**

Board Certified Clinical Neuropsychologist Assistant Professor of Psychiatry Chief, Division of Behavioral  
& Psychosocial Oncology Department of Psychiatry

Southern Illinois University—School of Medicine Springfield, IL

**Amy Olszewski, MS**

Doctoral Student

Department of Psychology

Eastern Michigan University

Ypsilanti, MI;

Clinical Assistant Instructor Department of Psychiatry

State University of New York (SUNY)—Upstate Medical University Syracuse, NY

**Justin H. Ory, MA**

Graduate Student

Department of Psychology

Louisiana State University

Baton Rouge, LA **Christine V. Paradee, PhD**

Senior Psychologist

Health Psychology Department Memorial Medical Center

Springfield, IL

**Eric E. Pierson, PhD**

Associate Professor of Psychology Director, Psychoeducational Diagnostic and Intervention Clinic

Department of Educational Psychology Ball State University

Muncie, IN

**Daniel Proto, PhD**

Clinical Neuropsychology Postdoctoral Fellow Department of Mental Health & Department of Physical  
Medicine & Rehabilitation Michael E. DeBakey VA Medical Center Houston, TX;

Instructor

Department of Psychiatry

Baylor College of Medicine

Houston, TX

**Susan M. Rumble, PsyD**

Clinical Neuropsychologist

Center for Transitional NeuroRehabilitation Barrow Neurological Institute St. Joseph's Hospital and  
Medical Center Phoenix, AZ

**David B. Salisbury, PsyD, ABPP**

Board Certified Clinical Neuropsychologist Baylor Institute for Rehabilitation Dallas, TX

**Margaret Semrud-Clikeman, PhD**

Professor of Pediatrics

Department of Pediatrics

University of Minnesota Medical School Minneapolis, MN

**Carrie-Ann H. Strong, PhD**

Staff Psychologist

Department of Psychology

Mary Free Bed Rehabilitation Hospital Grand Rapids, MI **Sherry T. Thrasher, PsyD**

Neuropsychology Fellow

Department of Neurology

Yale University–School of Medicine New Haven, CT

**Lisa Vroman, MA**

Graduate Student

Department of Psychology

Michigan State University

East Lansing, MI **Mandi Wilkes-Musso, MS**

Doctoral Candidate

Department of Psychology

Louisiana State University

Baton Rouge, LA **F. Scott Winstanley, PhD**

Clinical Neuropsychologist

Assistant Professor of Neurology Department of Neurology

Yale University–School of Medicine New Haven, CT



## Preface

Over the past three decades, medical advances have led to a substantial increase in the number of individuals surviving life-threatening incidents, including neurological trauma. This has led to a sizeable inflation of the number of individuals with significant and prolonged cognitive impairments in memory, reasoning, attention, judgment and self-awareness that negatively impacts their functionality and, in turn, quality of life. The Centers for Disease Control and Prevention estimates that over 5 million Americans are living with disabilities resulting from acquired brain injuries. Neurocognitive impairments are particularly troubling to individuals as they can greatly affect adaptability and are not as easily recognized by the general public. As the brain injury population grew over the past few decades, it became readily apparent that the traditional medical rehabilitation model was insufficient to address these particular functional impairments. Neuropsychology has become particularly important in rectifying these shortcomings.

In modern-day cognitive rehabilitation, neuropsychology is one of many specialties charged with the responsibility of returning individuals with brain injuries to the highest level of functionality and independence possible. While some professionals in neuropsychology still perceive their role as solely focused on assessment and diagnosis, cognitive rehabilitation has become an established practice within the field of neuropsychology. Neuropsychologists are regularly included as part of multidisciplinary rehabilitation teams alongside rehabilitation psychologists, neurologists, cognitive psychologists, speech pathologists, occupational therapists, special education professionals, and physical medicine practitioners. Considering the trends of the last two decades, one may reasonably expect an even greater increase in the reliance on neuropsychology as a core practice in cognitive rehabilitation efforts moving forward. Consequently, professional neuropsychologists as well as those in training must remain up-to-date with the changing landscape that is the practice and science of cognitive



rehabilitation. Research continues to shape our models of intervention and rehabilitative techniques. While variability can be seen across the literature regarding the utility and efficacy of cognitive rehabilitative efforts based on their design, measurement, and outcome, the vast majority of studies, including meta-analyses, support such rehabilitative efforts.

Recognizing the advances in the science and practice of cognitive rehabilitation, *Neuropsychological Rehabilitation* was conceived, with the goal of being a text that discusses these advances from a neuropsychological perspective. This text covers the most advanced practices and techniques in the rehabilitation of neuropsychological deficits, covering both specific neuropsychological domains and approaches in neurorehabilitation. It adheres to the philosophy that it is not enough to identify a deficit or diagnose a disease unless doing so helps to direct rehabilitation efforts to improve function. Intended to advance clinical skills of professionals and trainees alike, the book goes beyond surface diagnostic practice to foster rehabilitative efforts in response to residual deficits and disease.

This text discusses the foundations of neuropsychology in rehabilitation and along with, in depth, domain-specific rehabilitation practices, with a focus on functioning. Supplemental applications and practices that go beyond function-specific methodology, including neuroimaging and pharmacological agents, are also covered. Similarly, chapters are dedicated to the discussion of the role of system/environmental manipulation and transitioning strategies in rehabilitation. Finally, presentations/groupings most commonly seen in rehabilitation practice for which there is no prototypical form are covered from the standpoint of an integrated, neuropsychological approach.



## Acknowledgments

We want to acknowledge the work put forth by the assistant editors for this book including Dr. Javan Horwitz, who served as Lead Assistant Editor on this book, Dr. Amy R. Steiner, Dr. Michelle Pagoria, and Dr. John Joshua Hall.

A book such as this is only made possible through the contribution of various authors. We want to acknowledge their willingness to volunteer their time and knowledge to this work. As always, we want to acknowledge the support of our colleagues and associated institutions; SIU School of Medicine, Ball State University, and Baylor Institute for Rehabilitation, without whom this project would not be possible. Finally, we would like to express our sincerest gratitude to our publisher and those with whom we have worked very closely to complete this book, especially Nancy S. Hale and Joseph Stubenrauch.

**SERIES EDITORS**

**Chad A. Noggle, PhD, ABN**

**Raymond S. Dean, PhD, ABPP, ABN, ABPdN**

*The Neuropsychology of Psychopathology*

*The Neuropsychology of Cancer and Oncology*

*Neuropsychological Rehabilitation*

*The Neuropsychology of Cortical Dementias*

*The Neuropsychology of Pervasive Developmental Disorders*

*The Neuropsychology of Psychopharmacology*

# Historical Principles and Foundations of Neuropsychological Rehabilitation

*Karen K. Brewer-Mixon and C. Munro Cullum*

## BRIEF HISTORY OF NEUROPSYCHOLOGICAL REHABILITATION

Modern day cognitive rehabilitation (CR) spans a number of disciplines, including speech pathology, occupational therapy, special education, physical medicine, neurology, cognitive psychology, cognitive neuroscience, rehabilitation psychology, and neuropsychology. Rehabilitation has developed as a result of the contributions of all these professions and incorporates many different theories and techniques. Throughout the history of CR, neuropsychology has played a particularly important role.

The earliest rehabilitation efforts may be traced back to the crude efforts of ancient man, who practiced trepanation on persons with damaged skulls and disordered behavior. Later, ancient Greek and Roman scientists endeavored to understand the brain and restoration of function. Although some of their observations were quite sophisticated, such as Hippocrates' recognition that paralysis occurred opposite the side of a brain injury, they were often misled by the popular religious or cultural beliefs of their time. For example, Aristotle believed mental functions were localized in the heart, and Galen's "ventricular localization hypothesis" posited that "psychic gases and humours" flowed through the body and ventricles, giving rise to mental functions.

In the 16th century, Vesalius gave us the first scientific neuroanatomical documentation; Descartes' theories on mind-body dualism and the nature of the mind became dominant in the early 17th century. In 1664, Thomas Willis

published *Cerebri anatome*, the first medical text to describe and depict in drawings the anatomy of the brain and cranial nerves. Three years later, Willis published a complementary text on brain pathology and physiology. More than a century after Willis' painstaking work in neurology was published, Franz Gall put forth his theory of phrenology. Although Gall's theory was almost completely incorrect, it was perhaps a springboard for those who would later accurately describe localization of brain function.

In the mid-1800s, Paul Broca, the French physician, anatomist, and anthropologist, began his work. Among his many contributions, he described an intuitive rehabilitation program to restore lost skills in an adult patient who became unable to read aloud ([Berker, Berker, & Smith, 1986](#)). Building on Broca's work, the great German neuropsychiatrist Carl Wernicke was one of the first to conceptualize brain function as a series of regions that were dependent on interconnected neural pathways. This localization and interconnection concept became a fundamental element of clinical neuropsychology and CR.

As the 20th century dawned, prominent American neuropsychologist Shepherd Franz was using scientific methodology to study motor learning in hemiparesis and the effectiveness of therapy in clients with aphasia, making him a pioneer in neuropsychological rehabilitation ([Boake, 2003](#)). Like Broca, Franz noticed that his aphasic patients appeared to look more like they were learning a new skill rather than relearning an old habit. This observation established a precedent for using techniques that focus on learning new skills to compensate for abilities lost or diminished due to brain damage ([Witsken, D'Amato, & Hartlage, 2008](#)).

During and after the two World Wars, neuropsychology continued to grow as a discipline and to make important contributions to the development of CR. Germany and Austria led the way in developing brain injury rehabilitation centers to treat wounded soldiers. The well-known German psychologist Kurt Goldstein documented his treatment recommendations for speech, reading, and writing impairments. In doing so, he provided a template for rehabilitation efforts that drew on preserved abilities to help brain injury survivors compensate for impaired skills. In addition, his work helped develop behavioral methods for shaping desired behaviors ([Witsken et al., 2008](#)). About this same time, the great Soviet neuropsychologist Alexander Luria was working on theories of functional brain systems, based on his work with brain-injured veterans. He would go on to develop rehabilitation methods for working with patients with a variety of cognitive deficits, including aphasia, motor planning, visual perception, and

executive functioning disorders (Christensen & Castano, 1996). In 1947, the influential British neuropsychologist Oliver Zangwill made a significant contribution to the field of rehabilitation by delineating three principles/tasks of rehabilitation following brain injury (Zangwill, 1947): (a) substitution, which involves efforts to train brain-injured patients to use alternate strategies in place of those affected by impaired functions; (b) compensation, which relies on the use of alternative strategies to solve problems caused by impaired functioning; and (c) direct training (also called restoration), which involves retraining of impaired areas (Johnstone & Stonnington, 2009).

Cognitive rehabilitation continued to develop through the 20th century using a variety of strategies and techniques. The work of Yehuda Ben-Yishay during the late 1970s and 1980s increasingly recognized the importance of systematically addressing the interpersonal and social needs of clients in order for them to successfully re-enter their social environment. Muriel Lezak (1986) also contributed to the development of rehabilitation with her work on the emotional needs and responses of brain-injured patients and their families. Cicerone went on to explore the relationship between emotional and cognitive dysfunction (Cicerone & Fraser, 2000; Cicerone & Kalmar, 1997) and the understanding and treatment of frontal lobe dysfunction (Cicerone, 2002; Cicerone, Lazar, & Shapiro, 1983). Other ground-breaking work in the understanding, treatment, and management of attentional deficits was provided by Sohlberg and Mateer (1989, 2001); the influential work of Prigatano on awareness in brain-injured patients (Prigatano, 2005) and principles of rehabilitation also advanced the field (Prigatano, 2000).

In the past 50 years, not only has the field of CR made significant strides in its development, but the number of rehabilitation facilities has also grown. Neuropsychological rehabilitation centers in the United States proliferated in the 1980s and 1990s (Parente & Stapleton, 1997), which coincided with the development of advanced medical technologies that increased survival rates for persons with brain injuries. As more and more patients survive, the demand for rehabilitation has increased. During a 5-year period in the 1980s, rehabilitation beds in acute care hospitals increased 46%, and traumatic brain injury (TBI) rehabilitation was reported to be the fastest growing area in all of health care at that time (Frank, Gluck, & Buckelew, 1990).

## **NEUROPSYCHOLOGICAL REHABILITATION–TODAY**

Cognitive rehabilitation has become an established practice within the field of

neuropsychology. Research supporting the utility and efficacy of these techniques varies widely in terms of designs, measures, and outcomes; however, this contributes to a lack of consensus in the field and skepticism of some regarding the utility of CR procedures for their patients with brain injuries. A number of individual studies and meta-analyses have lent support to CR as a useful treatment (Bowen & Lincoln, 2007; Robey, 1998), although several *Cochrane Reviews* have noted “limited evidence” for the effectiveness of CR for deficits in attention, memory, and language following stroke (Greener, Enderby, & Whurr, 1999; Lincoln, Majid, & Weyman, 2000; Majid, Lincoln, & Weyman, 2000; Turner-Stokes, Disler, Nair, & Wade, 2005). Several leaders in the field (Cicerone, 2008) have noted an array of challenging methodological confounds and limitations that exist in most of the studies involving CR to date. Such limitations include heterogeneity of patients (in terms of disorder, duration/level of recovery, level of cognitive disability, age, etc.), as well as limitations and heterogeneity of measurement and intervention techniques (e.g., lack of appropriate control groups, failure to account for spontaneous recovery, and comorbid conditions). As was the case with the early psychotherapy literature, the efficacy of CR is well known to many patients, families, and practitioners, although the class I, evidence-based support for many specific procedures in this area remains in need of further systematic exploration.

Despite mounting evidence of effectiveness (see reviews by Carney et al., 1999; Cicerone, 2008), surprisingly little consensus has yet developed among neuropsychologists and other professionals regarding the best ways in which to remediate cognitive deficits. For example, even though CR programs may be touted as highly effective by patients and families and show improved patient outcomes overall (Rohling, Faust, Beverly, & Demakis, 2009), little is known about the efficacy of specific procedures; it was recently noted that “it is apparent that there is no uniformity of neuropsychological rehabilitation methods offered today, and there is uncertainty about the efficacy of most neuropsychological rehabilitation interventions” (Johnstone & Stonnington, 2009). Still, reports are emerging that compare various neuropsychological rehabilitation approaches. For example, Vanderploeg et al. (2008) reported similar global functional improvements in military personnel and veterans a year after they received either a cognitive–didactic treatment approach or a functional–experiential approach, although participants in the cognitive–didactic arm of the study reported fewer memory problems than those who received the functional–experiential treatment. Interesting influences of age and education

also appeared to differentially affect their study participants' ultimate return-to-work and independent living outcomes. Clearly, there is still a great deal of work yet to be done in developing a more sophisticated understanding as to which CR approaches work best and for which populations.

### **Current Rehabilitation Models**

One of the difficulties in reaching a consensus about how best to remediate cognitive dysfunction has been the dearth of published theoretical rehabilitation models from which methods can be derived. In one of the early attempts at developing a systematic CR program, [Reitan and Wolfson \(1988\)](#) described the Reitan Evaluation of Hemispheric Abilities and Brain Improvement Training (REHABIT). In this approach, a patient's treatment depended on the patient's specific neuropsychological deficits. The model was comprised of three levels of information processing: (a) attention, concentration, and memory; (b) lateralized processes (i.e., verbal vs. visuospatial); and (c) higher order abilities such as abstraction and logical analysis. Outcome data for the REHABIT program have been limited ([Brodsky, Brodsky, Lee, & Sever, 1986](#)).

In terms of methods for remediation of specific cognitive deficits, [Sohlberg and Mateer's \(1989, 2001\)](#) popular Attention Process Training (APT/APT-II) series is one of a very few comprehensive approaches to the retraining of a specific area of cognition that has gained attention. This approach is based on a hierarchical model of attentional abilities, which range from simplest to most complex. The components of the model, in order of increasing complexity, include: focused attention, sustained attention, selective attention, alternating attention, and divided attention. Sohlberg and Mateer's hierarchical model underlies and guides their approach to the rehabilitation of attention. For example, a patient's attentional abilities are assessed in each of the domains, then rehabilitation focuses first on the lowest levels of attention that are found to be impaired. As lower level skills improve through a variety of training exercises, more complex skills are practiced/remediated until patients reach their highest possible level of function. This approach has been shown to improve specific attentional abilities and helps to improve overall cognitive processing ([Sohlberg & Mateer, 1987](#); [Sohlberg, McLaughlin, Pavese, Heidrich, & Posner, 2000](#)).

[Robertson \(1996\)](#) proposed a specific cognitive retraining model for addressing executive dysfunction called the Goal Management Training method. This program trains patients with executive dysfunction to master five "stages"



or steps, which are aimed at first reducing impulsivity and then helping patients plan, organize, perform a task, and then check their work after the task is completed. The clinical utility of this approach has been shown in the rehabilitation of frontal lobe dysfunction in adults (Carter et al., 2000; Levine et al., 2000, 2011) and children (Metzler-Baddeley & Jones, 2010).

Clearly, progress is being made in terms of the development of models for rehabilitating attention and executive functioning. But what of other areas of cognitive function? Unfortunately, although there are many articles that discuss different methods and ideas regarding retraining visuospatial and language functioning (Rohling et al., 2009), as well as sensorimotor functions (Pichiorri et al., 2011), no one, to date, has published a comprehensive method for retraining these abilities that has been well validated and gained widespread acceptance.

Memory is another area in which there is not yet a widely accepted or comprehensive rehabilitation model, but there has been substantial clinical and experimental work in this area, which has guided the development of various individual methods. Methods such as errorless learning, spaced retrieval, and vanishing cues and compensatory strategies such as mnemonics and visualization/imagery techniques are used by clinicians in rehabilitation with some frequency. Among the more recent class I evidence available, errorless learning has been shown to be particularly effective (Cohen, Ylvisaker, Hamilton, Kemp, & Claiman, 2010; Lloyd, Riley, & Powell, 2009; Wilson, Baddeley, Evans, & Shiel, 1994) and was recently noted as having sufficient scientific evidence backing its relative advantages over errorful (e.g., trial-and-error) learning and error reduction strategies (Piras, Borella, Incoccia, & Carlesimo, 2011). Cicerone et al. (2011) note, however, that errorless learning in severely injured persons is often limited in terms of transfer to novel tasks or reduction in overall functional memory. Piras et al. (2011) also indicated that spaced retrieval and the method of vanishing cues were “potentially effective” rehabilitation methods, based on currently available scientific evidence. Compensatory strategies were found to have evidence of “probable effectiveness” for persons with moderate or severe memory impairment after TBI or stroke (Cicerone et al., 2011).

### **Criteria for Modern Rehabilitation Approaches**

In practice, CR approaches now tend to focus on one or more conceptual criteria that generally map onto the three principles of rehabilitation first posed by Zangwill (1947): substitution, compensation, and direct training. For example, a

rehabilitation program may seek to improve cognitive function primarily through repetitive, systematic, hierarchical *restorative* cognitive stimulation (e.g., Sohlberg & Mateer's APT series). Alternatively, a program may choose to teach *compensatory* strategies that target actual task performance or train patients to *substitute* a new method for their former, impaired approach. In 2005, Cicerone and colleagues published a landmark article in which they reviewed 47 CR studies, concluding that retraining is effective for some cognitive functions (e.g., language, attention), whereas compensation appears to be most effective for others (e.g., memory).

Another conceptual dimension rehabilitation programs must consider concerns the content of the treatment tasks themselves (Rizzo & Buckwalter, 1997). Some programs choose to focus on the training of *component* cognitive processes such as attention or memory, whereas others emphasize *functional skills* training such as practicing a standard set of steps in a work routine. Cognitive rehabilitation approaches can also be contrasted in terms of whether they are more *person centered* or *environment centered*. Environment-centered approaches are most effective when a patient is not capable of learning new functional skills and changes in his/her environment are needed to maximize function/independence. In reality, most CR programs are flexible and match the rehabilitation approach(es) to the needs of the patients they serve (Parente & Herrmann, 1996; Sohlberg & Mateer, 1989).

### **The Unique Contribution of Neuropsychology to CR**

It has been previously noted that CR is a multidisciplinary enterprise. The professional diversity that comprises the field of CR is a significant strength in many ways, though having so many different perspectives may be a disadvantage when it comes to the development of uniform models and methods for treatment. Nevertheless, there is one dimension in which neuropsychologists are particularly well positioned to contribute to the development of the field of CR. Most practicing neuropsychologists in North America are trained first as clinical psychologists, with additional predoctoral and postdoctoral training in neuropsychological techniques. Because of their training, they have expertise in emotional functioning, personality, and the impact of these on behavior. As a result, among all the professionals working in the CR field, neuropsychologists are perhaps the best equipped to understand and address the emotional responses of the patients with whom they work.

Emotional difficulties are very common among brain-impaired persons,

perhaps because an individual who has suffered a brain injury has suffered a significant personal loss and may have experienced some emotional trauma associated with the injury. It should not be surprising, then, when a brain-injured person exhibits depression, anxiety, posttraumatic stress disorder (PTSD), or other emotional reactions. In 1996, Kopelman and Crawford found 40% of 200 consecutive individuals referred to a memory clinic were suffering from clinical depression. Similarly, Bowen, Neumann, Conne, Tennant, and Chamberlain (1998) reported that 38% of 99 hospitalized TBI survivors had clinically significant mood disorders. Neurobiological changes, declines in functional capacity (physical and/or cognitive), psychosocial changes, and psychological responses to these changes can all play important roles in the development of emotional dysfunction after injury. Professionals who work in the CR field quickly become familiar with the negative impact emotional dysregulation, mood disorders, anxiety disorders, grief, and other emotional reactions can have on recovery. In some cases, these issues become more prominent than the cognitive dysfunction for which the patient is being treated in rehabilitation, and progress can slow or even grind to a halt as a result of such factors.

[Wilson \(2008\)](#) alluded to the unique contribution neuropsychologists have to make in the process of rehabilitation when she described neuropsychological rehabilitation as a broader field than CR because it encompasses “the amelioration of emotional, psychosocial, and behavioral deficits caused by an insult to the brain” in addition to cognitive remediation. Indeed, holistic CR models—models that incorporate treatment of psychosocial and emotional difficulties experienced by brain injury survivors with cognitive remediation and compensation training—are increasingly being studied and recognized as effective treatment approaches ([Ben-Yishay & Daniels-Zide, 2000](#); [Cicerone et al., 2008](#); [Rattok et al., 1992](#)). [Prigatano \(1999\)](#) went so far as to suggest that rehabilitation is likely to fail if clinicians do *not* deal with emotional issues with their brain-injured patients; he has become a well-known proponent of psychotherapeutic treatment for brain injury survivors.

Despite growing recognition that psychotherapy services are not simply a nice “add-on” to rehabilitation, but are an integral part of treating the brain injury survivor, there is very little literature regarding the development (or adaptation) of specific models of psychotherapy for use in conjunction with CR programs. A review of the available literature via PubMed, PsychInfo, and MEDLINE revealed only three studies that have reported results from the integration of psychotherapy with CR for cognitively impaired persons. One

involved mindfulness meditation therapy (McMillan, Robertson, Brock, & Chorlton, 2002), although it was noted that their brief intervention was not particularly helpful in alleviating mood or cognitive symptoms in TBI. In 2005, Mateer, Sira, and O'Connell discussed ways in which specific cognitive-behavioral therapy interventions were utilized with patients undergoing CR. They specifically targeted symptoms such as *catastrophic thinking* (e.g., a distortion in which a person imagines the worst possible outcome of an event or situation) or becoming hypersensitive to one's error rate in functioning due to failure to distinguish between "normal," ordinary errors in functioning and more serious errors that are likely caused by brain injury. In doing so, they presented a case report of one such successful integration of cognitive behavioral therapy (CBT) and CR with a mild TBI survivor. Tiersky *et al.* (2005) also demonstrated the usefulness of providing both CBT and CR on emotional functioning as well as improved attentional capacity at the conclusion of an 11-week program with TBI patients in a single-blind randomized, wait-list controlled trial. Utilizing a psychodynamic approach, Ben-Yishay and Daniels-Zide (2000) presented a thoughtful treatise on the importance of not only recognizing disruption in a patient's sense of self ("ego identity") after injury, but also the enormous difference it makes in functional outcomes when he/she is helped to rebuild a fractured ego identity during the rehabilitation process. Overall, there remains much work to do in the area of psychotherapy/CR integration.

### **Use of Technology in CR**

Neuropsychologists working at rehabilitation centers today increasingly have access to sophisticated technology that assists them in their understanding of the extent and nature of their patients' brain damage and in their treatment of their patients. Indeed, the process of rehabilitation itself is becoming more and more infused with the use of technology each year. For example, 20 years ago, it was common for neuropsychologists to train memory-impaired patients to use "memory notebooks" (typically small three-ring binders) as a means of remembering appointments and other important information. When handheld electronic devices such as personal organizers and cell phones became ubiquitous at the turn of the 21st century, neuropsychologists quickly adapted, incorporating these cognitive prosthetics into the rehabilitation process. Today, many patients are encouraged to use Smart phones, iPads, personal computers, and many other types of computerized devices in the rehabilitation process.

The value of computerized skills training programs is well established in

CR for a variety of populations. Over 20 years ago, Larose, Gagnon, Ferland, and Pépin (1989) provided 12-hour computerized attention training to 60 subjects with varying degrees of cognitive deficit. Results revealed improvements in attentional functioning for all subjects, regardless of their initial level of attentional capacity. Westerberg *et al.* (2007) demonstrated the effectiveness of computerized training of attention and working memory with stroke patients. Flavia, Stampatori, Zanotti, Parrinello, and Capra (2010) showed that a 3-month course of intensive computer-assisted CR was effective in improving the attention, information processing speed, and executive functioning abilities of mildly impaired patients with multiple sclerosis. Cognitive remediation using computers has also been shown to be effective in improving the cognitive functioning of patients with schizophrenia (Eack *et al.*, 2009).

Computerized rehabilitation has also been utilized successfully in pediatric populations, though there are far less data available in this area. For example, success has been reported using eight-to ten-session computerized rehabilitation programs to improve the attention and maze learning abilities of children with HIV-related cognitive impairment (Boivin *et al.*, 2010) and in children with cerebral malaria (Bangirana *et al.*, 2009). Similarly, Kesler, Lacayo, and Jo (2011) demonstrated improvements in processing speed, cognitive flexibility, and declarative memory in pediatric cancer survivors who participated in an 8-week computerized program.

As computer technology becomes increasingly sophisticated, virtual reality (VR) programs have been developed and applied to rehabilitation. Many have hailed VR as a tool with tremendous possibilities for rehabilitation. Critics feel that VR is a potentially exciting tool for rehabilitation, “but its evidence base is too limited by design and power issues to permit a definitive assessment of its value” (Crosbie, Lennon, Basford, & McDonough, 2007). Despite the limited VR data available to date, early reports suggest promise, as it has been shown to improve visual processing, visual learning, and reaction time in a preliminary sample of brain injury survivors (Grealy, Johnson, & Rushton, 1999).

In particular, VR technology appears to be promising in its ability to deliver ecologically valid rehabilitation outcomes. For example, VR rehabilitation has been shown to be effective in training students with severe learning disabilities how to shop in a virtual supermarket, with generalization of those skills to the real world (Cromby, Standen, Newman, & Tasker, 1996). Cox *et al.* (2010) described a VR program that was designed to help brain-injured

military personnel relearn driving skills. After 4 to 9 hours of driving simulation training, subjects participating in the program demonstrated significantly better driving skills, fewer risky driving behaviors, and fewer road-rage behaviors than subjects who had been treated with residential rehabilitation treatment only. [Yip and Man \(2009\)](#) reported success in teaching community living skills (e.g., using public transportation or shopping) and memory performance with four acquired brain injury (ABI)/stroke patients using a 10-session, VR-based community living skills training program.

Another new use of technology in neuropsychological rehabilitation is telerehabilitation—the remote administration of rehabilitation consulting, monitoring, and/or treatment via telecommunications/video transmission. To date, the telerehabilitation literature is limited, but is indeed growing and promising. In one of the earliest studies, [Lemaire, Boudrias, and Greene \(2001\)](#) conducted physical medicine consultations via videoconferencing and reported good overall satisfaction with procedures. [Forducey et al. \(2003\)](#) used telerehabilitation in a patient with severe TBI who lived in a nursing home in a remote area and reported improvements in his physical and cognitive status. We were able to identify only one study that reported on CR via videoconferencing ([Peel, Russell, & Gray, 2011](#)), although this was focused on elderly patients receiving rehabilitation in their homes. Nevertheless, this method of providing rehabilitation services will certainly continue to expand, both in terms of the use of videoconferencing-based as well as home-or Internet-based interventions (see Chapter 19 by McCue and Cullum).

## **FUTURE DIRECTIONS FOR NEUROPSYCHOLOGICAL REHABILITATION**

Those who work in the field of neuropsychological rehabilitation owe a debt of gratitude to all those scientists and clinicians who have contributed to our understanding of brain function and rehabilitation. Although much progress has been made, it is clear that there is still much that we do not know and much more to be done in this field. Some of the challenges for neuropsychological rehabilitation going forward include:

- *Continued development of conceptual models of CR.* Clinical practice without a guiding theory is simply a collection of techniques. Systematic models of CR are needed that will lead to sound, cohesive treatment programs. These programs must also be subjected to empirical study to

ensure that we are utilizing evidence-based methods rather than simply using techniques that “make sense,” that patients “like,” or which are comfortable because of their familiarity.

- *Increased emphasis on evidence-based interventions and improved CR research.* Cognitive rehabilitation programs need to be replicable and utilize state-of-the-art methodologic designs that will result in more published research. With advances in our ability to phenotype and genotype individuals using a variety of procedures, future goals should include the development of personalized CR programs based upon research that can help identify which patients may benefit most from specific CR intervention strategies.
- *Greater focus on the integration of CR and psychotherapy.* The importance of making psychotherapy services available to patients undergoing CR seems obvious; however, many patients still do not receive adequate attention to their emotional status. Identification of emotional issues is the key, and those who suffer more quietly or have more subtle emotional difficulties may not receive adequate treatment.
- *Incorporation of neuroimaging with neurocognitive techniques.* [Strangman and colleagues \(2010\)](#) recently reported that regional gray matter volumes could be used to predict memory outcomes following a specific 12-session group memory training intervention (O’Neill et al., 2010). Brain regions that were most associated with memory outcome included the hippocampus, prefrontal regions, thalamus, and areas of the cingulum.
- *Continued development of advanced technology applications.* Cognitive rehabilitation will continue to creatively utilize new technology. Virtual reality and telerehabilitation are but two of the newer, exciting areas in which CR advancement will undoubtedly continue. Recent studies have already demonstrated the feasibility and validity of telemedicine-based neuropsychological assessment ([Cullum, Weiner, Gehrman, & Hynan, 2006](#)), and CR programs are increasingly incorporating such technology into evaluation and treatment protocols (see [Chapter 19](#) by McCue and Cullum). Obviously, all new CR technologies and methods will need to be carefully evaluated for efficacy before they can enjoy widespread acceptance; however, it is with optimism and excitement that rehabilitation professionals look to the future and the development of these new ways of offering hope and relief from suffering to those

patients who experience brain insults.

## REFERENCES

- Bangirana, P., Giordani, B., John, C. C., Page, C., Opoka, R. O., & Boivin, M. J. (2009). Immediate neuropsychological and behavioral benefits of computerized cognitive rehabilitation in Ugandan pediatric cerebral malaria survivors. *Journal of Developmental and Behavioral Pediatrics: JDBP*, 30(4), 310–318.
- Ben-Yishay, Y., & Daniels-Zide, E. (2000). Examined lives: Outcomes after holistic rehabilitation. *Rehabilitation Psychology*, 45(2), 112–129.
- Berker, E. A., Berker, A. H., & Smith, A. (1986). Translation of Broca's 1865 report. Localization of speech in the third left frontal convolution. *Archives of Neurology*, 43(10), 1065–1072.
- Boake, C. (2003). Stages in the history of neuropsychological rehabilitation. In B. Wilson (Ed.), *Neuropsychological rehabilitation: Theory and practice* (pp. 11–21). Lisse, Netherlands: Swets & Zeitlinger.
- Boivin, M. J., Busman, R. A., Parikh, S. M., Bangirana, P., Page, C. F., Opoka, R. O., & Giordani, B. (2010). A pilot study of the neuropsychological benefits of computerized cognitive rehabilitation in Ugandan children with HIV. *Neuropsychology*, 24(5), 667–673.
- Bowen, A., & Lincoln, N. B. (2007). Cognitive rehabilitation for spatial neglect following stroke. *Cochrane Database of Systematic Reviews*, 2, CD003586.
- Bowen, A., Neumann, V., Conner, M., Tennant, A., & Chamberlain, M. A. (1998). Mood disorders following traumatic brain injury: Identifying the extent of the problem and the people at risk. *Brain Injury*, 12(3), 177–190.
- Brodsky, P., Brodsky, M., Lee, H., & Sever, J. (1986). Two evaluation studies of Reitan's REHABIT program for the retraining of brain dysfunctions. *Perceptual and Motor Skills*, 63(2 Pt 1), 501–502.
- Carney, N., Chesnut, R. M., Maynard, H., Mann, N. C., Patterson, P., & Helfand, M. (1999). Effect of cognitive rehabilitation on outcomes for persons with traumatic brain injury: A systematic review. *Journal of Head Trauma Rehabilitation*, 14(3), 277–307.
- Carter, G., Clare, L., Duncan, J., Hong, J., Levine, B., Roberston, I., . . . Wilson, B. (2000). Rehabilitation of executive functioning: An experimental-clinical validation of goal management training. *Journal of the International Neuropsychological Society*, 6, 299–312.
- Christensen, A. L., & Castano, C. (1996). Alexander Romanovich Luria (1902–1977): Contributions to neuropsychological rehabilitation. *Neuropsychological Rehabilitation*, 6(4), 279–303.
- Cicerone, K. D. (2002). The enigma of executive functioning: Theoretical contributions to therapeutic interventions. In P. J. Eslinger (Ed.), *Neuropsychological interventions: Clinical research and practice* (pp. 246–265). New York, NY: Guilford Press.
- Cicerone, K. D. (2008). Principles in evaluating cognitive rehabilitation research. In D. T. Stuss, G. Winocur, & I. H. Robertson (Eds.), *Cognitive neurorehabilitation* (pp. 106–118). New York, NY: Cambridge University Press.
- Cicerone, K. D., & Fraser, R. T. (2000). Counseling interactions for clients with traumatic brain injury. In R.T. Fraser & D.C. Clemmons (Eds.), *Traumatic brain injury rehabilitation: Practical vocational, neuropsychological, and psychotherapy interventions* (pp. 95–127). Boca Raton, FL: CRC Press.
- Cicerone, K. D., & Kalmar, K. (1997). Does premorbid depression influence post-concussive symptoms and neuropsychological functioning? *Brain Injury*, 11(9), 643–648.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., . . . Ashman, T. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation*, 92(4), 519–530.
- Cicerone, K. D., Lazar, R. M., & Shapiro, W. R. (1983). Effects of frontal lobe lesions on hypothesis sampling during concept formation. *Neuropsychologia*, 21(5), 513–524.



- Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., & Friel, J. C. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(12), 2239–2249.
- Cohen, M., Ylvisaker, M., Hamilton, J., Kemp, L., & Claiman, B. (2010). Errorless learning of functional life skills in an individual with three aetiologies of severe memory and executive function impairment. *Neuropsychological Rehabilitation*, 20(3), 355–376.
- Cox, D. J., Davis, M., Singh, H., Barbour, B., Nidiffer, F. D., Trudel, T., . . . Moncrief, R. (2010). Driving rehabilitation for military personnel recovering from traumatic brain injury using virtual reality driving simulation: A feasibility study. *Military Medicine*, 175(6), 411–416.
- Cromby, J., Standen, P. J., Newman, J., & Tasker, H. (1996). Successful transfer to the real world of skills practiced in a virtual environment by students with severe learning difficulties. In P. Sharkey (Ed.), *Proceedings of the European Conference on Disability, Virtual Reality and Associated Technology*, Skövde, Sweden.
- Crosbie, J. H., Lennon, S., Basford, J. R., & McDonough, S. M. (2007). Virtual reality in stroke rehabilitation: Still more virtual than real. *Disability and Rehabilitation: An International, Multidisciplinary Journal*, 29(14), 1139–1146.
- Cullum, C. M., Weiner, M. F., Gehrman, H. R., & Hynan, L. S. (2006). Feasibility of telecognitive assessment in dementia. *Assessment*, 13(4), 385–390.
- Eack, S. M., Greenwald, D. P., Hogarty, S. S., Cooley, S. J., DiBarry, A. L., Montrose, D. M., & Keshavan, M. S. (2009). Cognitive enhancement therapy for early-course schizophrenia: Effects of a two-year randomized controlled trial. *Psychiatric Services*, 60(11), 1468–1476.
- Flavia, M., Stampatori, C., Zanotti, D., Parrinello, G., & Capra, R. (2010). Efficacy and specificity of intensive cognitive rehabilitation of attention and executive functions in multiple sclerosis. *Journal of the Neurological Sciences*, 288(1–2), 101–105.
- Forducey, P. G., Ruwe, W. D., Dawson, S. J., Scheideman-Miller, C., McDonald, N. B., & Hantla, M. R. (2003). Using telerehabilitation to promote TBI recovery and transfer of knowledge. *NeuroRehabilitation*, 18(2), 103–111.
- Frank, R. G., Gluck, J. P., & Buckelew, S. P. (1990). Rehabilitation. Psychology's greatest opportunity? *American Psychologist*, 45(6), 757–761.
- Grealy, M. A., Johnson, D. A., & Rushton, S. K. (1999). Improving cognitive function after brain injury: The use of exercise and virtual reality. *Archives of Physical Medicine and Rehabilitation*, 80(6), 661–667.
- Greener, J., Enderby, P., & Whurr, R. (1999). Speech and language therapy for aphasia following stroke. *Cochrane Database of Systematic Reviews*, 4, 1–62.
- Johnstone, B., & Stonnington, H. H. (Eds.). (2009). *Rehabilitation of neuropsychological disorders: A practical guide for rehabilitation professionals*. Philadelphia, PA: Psychology Press.
- Kopelman, M., & Crawford, S. (1996). Not all memory clinics are dementia clinics. *Neuropsychological Rehabilitation*, 6, 187–202.
- Kesler, S. R., Lacayo, N. J., & Jo, B. (2011). A pilot study of an online cognitive rehabilitation program for executive function skills in children with cancer-related brain injury. *Brain Injury*, 25(1), 101–112.
- Larose, S., Gagnon, S., Ferland, C., & Pépin, M. (1989). Psychology of computers: XIV. Cognitive rehabilitation through computer games. *Perceptual and Motor Skills*, 69(3 Pt 1), 851–858.
- Lemaire, E. D., Boudrias, Y., & Greene, G. (2001). Low-bandwidth, Internet-based videoconferencing for physical rehabilitation consultations. *Journal of Telemedicine and Telecare*, 7(2), 82–89.
- Levine, B., Robertson, I. H., Clare, L., Carter, G., Hong, J., Wilson, B. A., . . . Stuss, D. T. (2000). Rehabilitation of executive functioning: an experimental-clinical validation of goal management training. *Journal of the International Neuropsychological Society: JINS*, 6(3), 299–312.
- Levine, B., Schweizer, T. A., O'Connor, C., Turner, G., Gillingham, S., Stuss, D. T., . . . Robertson, I. H. (2011). Rehabilitation of executive functioning in patients with frontal lobe brain damage with goal management training. *Frontiers in Human Neuroscience*, 5, 9.

- Lezak, M. (1986). Psychological implications of traumatic brain damage for the patient's family. *Rehabilitation Psychology, 31*(4), 241–250.
- Lincoln, N. B., Majid, M. J., & Weyman, N. (2000). Cognitive rehabilitation for attention deficits following stroke. *Cochrane Database of Systematic Reviews, 4*, CD002842.
- Lloyd, J., Riley, G. A., & Powell, T. E. (2009). Errorless learning of novel routes through a virtual town in people with acquired brain injury. *Neuropsychological Rehabilitation, 19*(1), 98–109.
- Majid, M. J., Lincoln, N. B., & Weyman, N. (2000). Cognitive rehabilitation for memory deficits following stroke. *Cochrane Database of Systematic Reviews, 3*, CD002293.
- Mateer, C. A., Sira, C. S., & O'Connell, M. E. (2005). Putting Humpty Dumpty together again: The importance of integrating cognitive and emotional interventions. *Journal of Head Trauma Rehabilitation, 20*(1), 62–75.
- McMillan, T. M., Robertson, I. H., Brock, D., & Chorlton, L. (2002). Brief mindfulness training for attentional problems after traumatic brain injury: A randomized control treatment trial. *Neuropsychological Rehabilitation, 12*(2), 117–125.
- Metzler-Baddeley, C., & Jones, R. W. (2010). Brief communication: Cognitive rehabilitation of executive functioning in a case of craniopharyngioma. *Applied Neuropsychology, 17*(4), 299–304.
- Parente, R., & Herrmann, D. (1996). *Retraining cognition: Techniques and applications*. Gaithersburg, MD: Aspen.
- Parente, R., & Stapleton, M. (1997). History and systems of cognitive rehabilitation. *Neurorehabilitation, 8*, 3–12.
- Peel, N. M., Russell, T. G., & Gray, L. C. (2011). Feasibility of using an in-home video conferencing system in geriatric rehabilitation. *Journal of Rehabilitation Medicine, 43*(4), 364–366.
- Pichiorri, F., De Vico Fallani, F., Cincotti, F., Babiloni, F., Molinari, M., Kleih, S. C., . . . Mattia, D. (2011). Sensorimotor rhythm-based brain-computer interface training: The impact on motor cortical responsiveness. *Journal of Neural Engineering, 8*(2), 025020.
- Piras, F., Borella, E., Incoccia, C., & Carlesimo, G. A. (2011). Evidence-based practice recommendations for memory rehabilitation. *European Journal of Physical and Rehabilitation Medicine, 47*(1), 149–175.
- Prigatano, G. P. (1999). *Principles of neuropsychological rehabilitation*. New York, NY: Oxford University Press.
- Prigatano, G. P. (2000). A brief overview of four principles of neuropsychological rehabilitation. In A. Christensen & B. P. Uzzell (Eds.), *International handbook of neuropsychological rehabilitation* (pp. 115–125). Dordrecht, Netherlands: Kluwer Academic Publishers.
- Prigatano, G. P. (2005). Disturbances of self-awareness and rehabilitation of patients with traumatic brain injury: A 20-year perspective. *Journal of Head Trauma Rehabilitation, 20*(1), 19–29.
- Rattok, J., Ross, B., Ben-Yishay, Y., Ezrachi, O., Silver, S., Lakin, P., . . . Diller, L. (1992). Outcome of different treatment mixes in a multidimensional neuropsychological rehabilitation program. *Neuropsychology, 6*(4), 395–415.
- Reitan, R. M., & Wolfson, D. (1988). *Traumatic brain injury, volume II: Recovery and rehabilitation*. Tucson: Neuropsychology Press.
- Rizzo, A. A., & Buckwalter, J. G. (1997). Virtual reality and cognitive assessment and rehabilitation: The state of the art. *Studies in Health Technology and Informatics, 44*, 123–145.
- Robertson, I. H. (1996). *Goal management training: A clinical manual*. Cambridge, MA: PsyConsultants.
- Robey, R. R. (1998). A meta-analysis of clinical outcomes in the treatment of aphasia. *Journal of Speech, Language, and Hearing Research, 41*(1), 172–187.
- Rohling, M. L., Faust, M. E., Beverly, B., & Demakis, G. (2009). Effectiveness of cognitive rehabilitation following acquired brain injury: A meta-analytic re-examination of Cicerone *et al.*'s (2000, 2005) systematic reviews. *Neuropsychology, 23*(1), 20–39.
- Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attention-training program. *Journal of Clinical and Experimental Neuropsychology, 9*(2), 117–130.
- Sohlberg, M. M., & Mateer, C. A. (1989). *Introduction to cognitive rehabilitation: Theory and practice*.

- New York, NY: Guilford Press.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative approach*. New York, NY: Guilford Press.
- Sohlberg, M. M., McLaughlin, K. A., Pavese, A., Heidrich, A., & Posner, M. I. (2000). Evaluation of attention process training and brain injury education in persons with acquired brain injury. *Journal of Clinical and Experimental Neuropsychology*, 22(5), 656–676.
- Strangman, G. E., O’Neil-Pirozzi, T. M., Supelana, C., Goldstein, R., Katz, D. I., & Glenn, M. B. (2010). Regional brain morphometry predicts memory rehabilitation outcome after traumatic brain injury. *Frontiers in Human Neuroscience*, 4, 182.
- Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., & Deluca, J. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 86(8), 1565–1574.
- Turner-Stokes, L., Disler, P. B., Nair, A., & Wade, D. T. (2005). Multidisciplinary rehabilitation for acquired brain injury in adults of working age. *Cochrane Database of Systematic Reviews*, 3, CD004170.
- Vanderploeg, R. D., Schwab, K., Walker, W. C., Fraser, J. A., Sigford, B. J., Date, E. S., . . . Defense and Veterans Brain Injury Center Study Group. (2008). Rehabilitation of traumatic brain injury in active duty military personnel and veterans: Defense and Veterans Brain Injury Center randomized controlled trial of two rehabilitation approaches. *Archives of Physical Medicine and Rehabilitation*, 89(12), 2227–2238.
- Westerberg, H., Jacobaeus, H., Hirvikoski, T., Clevberger, P., Ostensson, M. L., Bartfai, A., & Klingberg, T. (2007). Computerized working memory training after stroke—a pilot study. *Brain Injury*, 21(1), 21–29.
- Wilson, B. A. (2008). Neuropsychological rehabilitation. *Annual Review of Clinical Psychology*, 4, 141–162.
- Wilson, B. A., Baddeley, A., Evans, J., & Shiel, A. (1994). Errorless learning in the rehabilitation of memory impaired people. *Neuropsychological Rehabilitation: An International Journal*, 4(3), 307–326.
- Witsken, D. E., D’Amato, R. C., & Hartlage, L. C. (2008). Understanding the past, present, and future of clinical neuropsychology. In R.C. D’Amato & L. C. Hartlage (Eds.), *Essentials of neuropsychological assessment, second edition: Treatment planning for rehabilitation* (pp. 9–10). New York, NY: Springer Publishing Company.
- Yip, B. C., & Man, D. W. (2009). Virtual reality (VR)-based community living skills training for people with acquired brain injury: A pilot study. *Brain Injury*, 23(13–14), 1017–1026.
- Zangwill, O. L. (1947). Psychological aspects of rehabilitation in cases of brain injury. *British Journal of Psychology*, 37(2), 60–69.

# Neurological Recovery and Plasticity

*Javan Horwitz and Natalie Horwitz*

With the advent of technologies in neuroimaging and evoked potentials, as well as advances in the field of neurobiology, neuroscientists have improved their appreciation of neurocognitive recovery. Not only does recovery lead to understanding cerebral reorganization following a brain injury, but such an understanding allows clinicians to better determine prognosis and assist in developing treatment plans specifically oriented toward rehabilitation.

Despite the growing knowledge about recovery, this domain has been plagued with logistical and methodological difficulties, which has led to incompetence or contradictory information (Heilman & Valenstein, 2003). Much of our knowledge about recovery comes from stroke studies; however, the larger the study, the more variability exists, resulting in some difficulties with deriving definitive conclusions. Recovery from trauma is even more heterogeneous than recovery from vascular injuries, as mild head injuries tend to recover with much greater variability than more severe injuries (Heilman & Valenstein, 2003). Again these discrepancies may be related to difficulties with conducting methodologically sound studies, including matching controls to experimental subjects, following injured patients after diagnosis longitudinally, involving rehabilitation specialists early in the process of diagnosis, and having adequate specificity on testing measures. This incomplete understanding of recovery of neurocognitive functions has led to the development of several theories, which will be reviewed briefly to aid in the understanding of how neural plasticity is involved in the process of recovery.

## PRINCIPLES OF NEUROLOGICAL RECOVERY AND PLASTICITY

Neuroimaging studies have revealed a preponderance of evidence that ipsilateral adjacent structures to an injury are more involved in the recovery process compared to homologous contralateral structures—with the exception of recovery of the auditory and visual comprehension of language (Heilman & Valenstein, 2003; Thiel et al., 2001). Unfortunately, some of these imaging experiments may be confounded by technical and methodological complexities. Outcomes are difficult to decipher because activation may actually be nonspecific or due to peripheral functions and not the result of cognitive compensation (Heiss, Kessler, Karbe, Fink, & Pawlik, 1993; Iglesias et al., 1996).

Regardless of these limitations, the idea of compensation is important to neuropsychologists and rehabilitation specialists because it disconfirms the theories of rigid localization of neurocognitive functioning (Heilman & Valenstein, 2003). In other words, if the recovery of a functional ability is possible following destruction of a purported functionally localized region, then it becomes clear that that particular function cannot be restricted to one neuroanatomical region—especially in the case of functional compensation, which will be discussed later. Thus, the brain is malleable and capable of dynamic reorganization—not being rigidly fixed in function as was purported by the field of neuroscience decades ago.

In 1914, Von Monakow developed the concept of diaschisis, derived from aphasia models, which revealed that damage to the nervous system results in a withdrawal or removal of connected tissues, thereby resulting in a functional impairment (Heilman & Valenstein, 2003). This connection is eventually renewed during recovery. These findings have been supported by biochemical and physiological research (Heilman & Valenstein, 2003).

Recovery from injury tends to happen in a two-stage process: namely, acute recovery followed by long-term recovery. During the acute stages of recovery, the focus of action is on several structural and chemical processes, including tissue water and electrolyte dysfunction, structural failure, neurochemical imbalances, reestablishment of vascular function, and cellular reaction to the injury.

The focus of this chapter will be on the long-term, or “second stage,” recovery that is particularly sensitive to current neuropsychological cognitive rehabilitation strategies. Although this process is not completely understood, it is believed that much of recovery from a neurocognitive and structural perspective comes as a result of compensation from functionally or structurally related areas

—with a lesser involvement of actual axonal regrowth or collateral sprouting (i.e., plasticity).

## STRUCTURAL COMPENSATION

Structural compensation involves brain compensation that uses the development of new strategies to address the task with the residual and primary functional structures—or dynamic reorganization, which accounts for a small portion of recovery following injury (Luria, 1970).

Compensation was first proposed by Flourens in 1824, who utilized animal models to oppose localization theories. Following ablation, the animals did not move much and neglected to eat, but later recovered, resulting in the disconfirmation of the idea that different areas of the cortex had exclusive specialized functions (with the exception of the brain stem and cerebellum; Kolb & Whishaw, 2003). The paradox of neuropsychology today is that behavior recovers after the area of the brain thought to be exclusively central to that function has been damaged. This leads to the current neuroscience paradigm that the cortex works in a dynamic and integrated manner with the ability of different zones to compensate for primary structures leading the specific neurocognitive task.

Plasticity, or regeneration of tissue, has been recently demonstrated to occur not only in the peripheral nervous system but also in the central nervous system. Neural plasticity after lesions has been extensively demonstrated in animal models (Merzenich et al., 1983). Lashley (1938) determined that recovery depended on the extent of injury and the remaining intact cortical tissue, as well as the fact that certain areas, for example, the visual striate cortex, were vital for compensation to occur. Regenerative sprouting or axonal regrowth into vacant terminal spaces is noted in the ascending catecholaminergic fibers, whereas collateral sprouting or regrowth into neighboring vacant terminal spaces tends to be important in terms of recovery (Liu & Chambers, 1958; Moore, 1974; Schneider, 1973). Cortical reorganization occurs with experience or training through long-term potentiation of neurons through the nerve growth factor, glutamate cascade, and gamma-aminobutyric acid (GABA) inhibition, noradrenaline, and nitric oxide systems (Bear & Malenka, 1994; Hallett, 1999; Pascual-Leone, Grafman, & Hallett, 1994). Astrocytes are also actively involved in recovery, as enriched environments and training following injury result in membrane excitability changes, growth of new connections, and increase in dendritic spines (Johansson, 2000).

Physiological studies of structural brain plasticity elucidate two primary processes: long-term potentiation and kindling (Cooke & Bliss, 2006; Serrano, Yao, & Sacktor, 2005). Long-term potentiation, a process that occurs frequently in learning and memory as a result of stimulation to the associated areas of the cerebral neocortex and hippocampus, involves changes in synaptic signal transmission and efficiency (Cooke & Bliss, 2006). However, kindling involves synaptic organization and biochemical cascades on a molecular level, resulting in formation of growth factors (Cooke & Bliss, 2006). At extreme levels, kindling has been known to result in behavioral activation of epileptic seizures.

Interestingly, functional reorganization, which will be discussed below, is often important in behavioral recovery but may also inhibit structural restitutive functions in damaged circuits. A study conducted by Levere and Levere explored this process further with visual cortex lesions in rats (Sohlberg & Mateer, 2001). The rats with damaged cortexes had a tendency to ignore visual cues presented to them and only responded to nonvisual cues. Once the nonvisual cues were made less apparent, the rats were able to utilize visual cues, but with less efficiency. It was concluded that compensatory mechanisms may inhibit the activation of structural restorative functioning processes.

Overall, structural compensation—or utilization of residual structures—is an important factor in recovery, but serves as a lesser component in the global functional outcome of the rehabilitation process.

## FUNCTIONAL COMPENSATION

Functional compensation is different from structural compensation and serves a major role in rehabilitation following injury. Functional compensation involves rerouting connections structurally to alternate functional zones, whereas with structural compensation, the brain compensates by developing new strategies to address the task with the residual and primary functional structures (Luria, 1970).

Compensation strategies appear to be part of an anticipatory system called vicarious functioning that allows an organism to substitute one structure—whose functions were not associated with the damaged area previously—to function for a damaged one (Heilman & Valenstein, 2003). In 1873, Jackson advocated a hierarchically organized nervous system where function was redundantly represented at several levels and that damage to higher levels resulted in disinhibition to lower levels, thereby leading to compensation.

The spinal cord innervations of the diaphragm that take over when

brainstem structures are damaged, the existence of several cortical sensory systems, and two visual systems with rostrocaudal connection in the brainstem and occipital cortex are examples of this redundant representation and potential for functional compensation ([Woolsey & van der Loos, 1970](#)). In human models, the right hemisphere was noted to assume some language functions following hemispherectomies and colossal sections, including comprehension (nouns better than verbs) and automatic nonpropositional speech ([Gazzaniga, 1970](#); [Smith, 1966](#)). This restitution of speech due to activity in the opposite hemisphere is known as Henschen's axiom ([Heilman & Valenstein, 2003](#)). Interestingly, injections of sodium amytal into the right carotid produced aphasia in patients who had recovered from aphasia secondary to left-hemisphere stroke ([Kinsbourne, 1971](#)).

In adults, functional recovery capacity tends to be finite based on the capacity of the compensating area to substitute for the function. However, this ability is highly variable between individuals and influenced by a number of factors ([Geschwind, 1969](#)).

## **FACTORS AFFECTING REHABILITATION**

### **Developmental Age of Injury and Critical Periods**

Early research conducted in understanding the benefit of plasticity following a brain injury was predicated on the belief that age was indirectly correlated with plasticity, vulnerability, and recovery ([Spencer-Smith & Anderson, 2011](#)). In other words, the younger the individual was at the time of the brain injury, the more likely it would be that a complete functional recovery would occur.

An early theory exploring this process is known as the Kennard Principle (1938), which purports that recovery from brain injuries obtained better prognostic outcome when sustained at an earlier age of the organism. This theory was developed after observing primates with unilateral lesions in the motor and premotor cortex during infancy. Following injury, the contralateral hemispheric region compensated by mediating motor performance both ipsilaterally and contralaterally. Additionally, prognosis of recovery from aphasia before age 10 to 12 is excellent because development of the left hemisphere tends to inhibit language abilities in the right ([Basser, 1962](#); [Hecaen, 1976](#); [Milner, 1974](#)). Thus, as the brain matures, it becomes more definitively specialized, resulting in reduced ability to compensate with alternate less



malleable zones. Thus, this research demonstrated that early age and diminished brain cell specialization were more associated with structural and functional compensation.

In contrast with this view of age of insult predicting better functional outcomes was Donald Hebb's (1949) research which postulated that brain injuries at an early age could have greater deleterious effects on behavior than injuries that occur at a later stage in life (Spencer-Smith & Anderson, 2011). Specifically, he noted that intellectual performance would be most affected by delays in certain cortical regions, especially the frontal lobes, which could result in stagnation of subsequent developmentally attained intellectual and neurocognitive functions. Therefore, the earlier the injury, the more delayed the developmental process would be, resulting in more pronounced functional impairments if they occur earlier rather than later in the developmental chronology (Sohlberg & Mateer, 2001).

Plasticity in the immature nervous system tends to depend on the adaptability of Golgi type II cells, which remain adaptive, as opposed to other cells responsible for the major transmissions of information that become highly specialized early in development secondary to genotype and phenotype expression (Hirsch & Jacobson, 1974). This flexibility is terminated in the teens by hormonal changes resulting in reduced plasticity of the brain and increased specialization/organization of particular neurological areas. Overall, the influence of age and plasticity remains controversial with limited evidence to suggest that younger adult patients recover better than older patients do—despite the clinical impression of such (Eisenson, 1949; Kertesz & McCabe, 1977).

In conclusion, developmental age when the injury occurs is a more complicated factor than just chronology alone. Instead of focusing on the sole factor of chronological age in its role in recovery, the crucial timing of brain injuries in the developmental spectrum—especially as it relates to critical developmental periods—is of utmost importance in its overall relation to structural and functional compensation. This supposition is confirmed by current research in traumatic brain injury, which demonstrates the brain working in a dynamic manner and that there are critical periods in development when injury is associated with a worse outcome and other critical periods tied with more optimal outcomes (Sohlberg & Mateer, 2001). Some of these critical periods of neural development include neurogenesis, apoptosis, postnatal proliferation and pruning, and developmental refinement of synaptic connections. In 1979, Huttenlocher examined synaptic density in different brain regions across the life

span. He reported peak densities in the frontal cortex at around 2 years of age, with high levels remaining until around age 8 ([Spencer-Smith & Anderson, 2011](#)). In 1990 he noted that adult levels of synaptic density are reached at around 16 years.

### **Sex Differences**

Although male brains are about 200 g larger than females, there is no evidence to suggest that size alone is advantageous to recovery. There exist sex differences in cortical density of neurons with males demonstrating less overproduction than females during gestation, making them more susceptible to injury, but later they have higher neuronal density leading to some protection from injury ([de Courten-Myers, 1999](#)). Logically, it would follow that considering that language is more bilaterally distributed in females than in males, recovery would be better in females with damage to language centers; however, statistically significant sex differences in aphasia recovery have not been definitively determined experimentally or on dynamic neuroimaging in human models ([Frost et al., 1999](#); [Kertesz, 1988](#); [Pedersen et al., 1995](#)).

Animal models found promising results for prevention and recovery in rats with bilateral medial frontal contusions treated with progesterone postinjury ([Roof, Duvdevani, Braswell, & Stein, 1992](#)). Progesterone not only reduced edema postinjury, but also resulted in an enhancement of neuritic outgrowth, the formation of new myelin sheaths, and the regulation of GABA receptors, which can influence the cascade of excitatory and excitotoxic activity that occurs after brain injury. Again, application of these findings to human models is still in the early stages, but is suggestive of beneficial results such as reduced mortality following injury and better recovery ([Gever, 2008](#)).

### **Handedness and Right-Versus Left-Sided Recovery**

Early research demonstrated that left-handed individuals tend to recover better from aphasia than right-handed individuals, but they are prone to become aphasic with lesions to either hemisphere ([Subirana, 1969](#)). Right-handed individuals who had a history of left-handedness in their families also tended to recover better than those without such a history ([Geschwind, 1974](#)). However, more recent research did not demonstrate such results in recovery as it relates to handedness ([Pedersen et al., 1995](#)). On the other hand, motor differences are noted between right and left hemiplegia as it relates to recovery, with left hemiplegia recovering slower due to the associated spatial–perceptual

deficiencies that make recovery more resistant (Heilman & Valenstein, 2003).

### **Premorbid Education Level**

Cognitive reserve is another important factor in the recovery process with more experiential involvement or higher level of education being associated with more resiliency postinjury (Kolb & Wishaw, 2003). These individuals tend to maintain more intricate neuroanatomical pathways resulting not only in more reinforced pathways, but also multiple and redundant neural pathways as a result of continued practice and allocation of functions to an automatized process (Sohlberg & Mateer, 2001). These developed redundant pathways or nodes of connectivity allow for easier functional compensation and ultimately better prognosis postinjury (Sohlberg & Mateer, 2001). Additionally, these individuals tend to be better able to identify and develop alternate compensatory mechanisms, demonstrate more motivation for treatment, and have more access to rehabilitation services.

Although higher cognitive reserve has its advantages, it may also result in barriers to treatment and recovery. This is particularly true when a patient perceives a more dramatic cognitive decline, resulting in emotional dysregulation (i.e., depression), which can diminish motivation for treatment. As such, it is essential to mobilize the patient's resources and support systems to enhance the treatment and rehabilitative process.

### **Motivation and Mood**

Motivation and mood also plays a role in recovery with evidence noting that intense motivation with a positive outlook results in better functional outcomes (Robinson & Benson, 1981). Thus, depression tends to play a negative role in recovery. At one time, the field of neuroscience purported that left-sided lesions result in more depression; however, current studies show no correlation with depressive symptoms and left-sided lesions (Herrmann, Black, Lawrence, Szekely, & Szalai, 1998). Instead, the extent of recovery from neurological impairment correlates with depressive symptoms and functional outcome.

## **NEUROPSYCHOLOGICAL AND NEUROBEHAVIORAL TENDENCIES**

In general, recovery from central nervous system injury tends to occur in relation to lesion location (i.e., viability of surrounding tissue and pathways) and size and follows a definitive course, with significant recovery noted in the first 2 weeks

and significant neurofunctional improvements in the first 3 months until approximately 6 month postinjury ([Kohlmeyer, 1976](#); [Sohlberg & Mateer, 2001](#); [Spencer-Smith & Anderson, 2011](#)). Additionally, early recovery in the first week is a good prognostic indicator for overall recovery ([Jorgensen et al., 1999](#)). Spontaneous recovery within the first 2 months tends to be a normal part of the organism's recovery process ([Sohlberg & Mateer, 2001](#); [Spencer-Smith & Anderson, 2011](#)). Typically, further spontaneous recovery is not seen; yet with an enriched environment (i.e., cognitive or rehabilitative therapy), individuals may demonstrate improvements years after injury ([Sohlberg & Mateer, 2001](#); [Spencer-Smith & Anderson, 2011](#)).

Recovery can also be influenced by the number of previously sustained injuries, with more injuries being associated with more cognitive decline and a tendency for the brain to be less likely to reorganize and compensate. In 1914, Von Monakow determined that slow-growing lesions tend to result in fewer deficits when compared to acute lesions as a result of compensation ([Heilman & Valenstein, 2003](#)). These findings have been supported in several studies ([Sohlberg & Mateer, 2001](#); [Spencer-Smith & Anderson, 2011](#)) as well as current observations of small strokes, transient ischemic attacks (TIAs), and slow-growing tumors, which tend to demonstrate no significant neurocognitive or functional impairments. In summary, given spontaneous recovery, slow-growing lesions tend to self-compensate, resulting in little to no functional impairments, whereas more acute injuries tend to result in functional and objective impairments.

In regard to a definitive course of recovery, different neurocognitive functional impairments tend to follow different patterns. Recovery from aphasia tends to follow a four-stage process: (a) differentiation by intonation, (b) decreased automatic utterances, (c) less-rigid stereotypic utterances, and (d) volitional, slow agrammatic speech ([Heilman & Valenstein, 2003](#)). Although, recovery from posttraumatic memory impairments tends to be correlated with the duration of posttraumatic amnesia ([Russell, 1971](#)). Recovery of motor functions typically progresses with first lower extremity recovery—with proximal recovery occurring prior to distal—followed by upper extremity recovery ([Sohlberg & Mateer, 2001](#); [Spencer-Smith & Anderson, 2011](#)). Consequently, neuropsychological and neurobehavioral findings will be variable based on the etiology and type of injury, patient demographics (including developmental factors), psychiatric functioning, psychosocial factors (including medications and illicit substances), time since injury, injury severity, focal

versus diffuse deficits, and neurocognitive functional zones involved.

For example, traumatic brain injury is often associated with the frontal and temporal lobes (Levin & Kraus, 1994). Frontal lobe impairments involve disturbances of higher level regions such as executive functioning, which is responsible for planning, sequencing, judgment and insight; whereas temporal lobe impairments involve the region of the brain that is responsible for the organization of auditory sensation and perception, attention control, arousal, language comprehension, declarative and remote memory, and emotional dysregulation. Although some deficits may not be obvious upon cursory evaluation, comprehensive neuropsychological testing will allow for quantifiable difficulties associated with the injury and treatment recommendation, allowing for enhancement of the structural and functional compensation processes of recovery.

## **TREATMENT AND REHABILITATION**

As noted, with variability in clinical presentation postinjury, patients tend to experience various and different needs, which change, dependent on their process of recovery. For instance, during early recovery from a traumatic brain injury, it is crucial that a restorative sleep hygiene program be implemented, both behaviorally and pharmacologically, to compensate for the disrupted sleep cycle and to allow for brain rehabilitation and healing to occur to capacity. However, complexity prevails with treatment planning and recovery—as some pharmacologic interventions may interfere with structural and functional compensation.

Another important consideration to optimizing traumatic brain injury recovery is to reduce cognitive overload. Patients who are early in their recovery may have decreased ability to sustain arousal, resulting in exacerbated fatigue and physical and cognitive overstimulation. Providing breaks and reducing the duration of therapies allows for more effective outcomes and reduces the likelihood of fatigue. Thus, patients will likely respond better to interventions or cognitive rehabilitative procedures when they are more alert, allowing for better participation and integration of new coping and compensatory skills.

With optimized levels of alertness, it will be important to understand areas of strengths and weaknesses and to utilize alternate pathways or modalities that will maximize functional compensation.

## **SUMMARY**

The concept of plasticity has been extensively discussed and also misunderstood. Both structural compensation and functional compensation contribute to the idea of plasticity and neural recovery. Developmental age, biological sex, handedness, and many other factors all mediate this recovery process. Given the advances in the human genome project, the field of neuroscience, and molecular and cellular biology, future treatments will likely explore both pharmacological interventions to alter biochemistry postinjury and gene and stem cell therapy to implant or graft genes or unspecialized cells to stimulate growth (Heilman & Valenstein, 2003; Sohlberg & Mateer, 2001; Spencer-Smith & Anderson, 2011). Thus, interventions during acute recovery will likely be prominent and add to better outcomes with long-term or second stage recovery, furthering our ability to implement and achieve positive outcomes with current neurocognitive rehabilitative therapies.

## REFERENCES

- Basser, L. S. (1962). Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. *Brain*, 85, 427–460.
- Bear, M. F., & Malenka, R. C. (1994). Synaptic plasticity: LTP and LTD. *Current Opinion in Neurobiology*, 4(3), 389–399.
- Cooke, S. F., & Bliss, T. V. (2006). Plasticity in the human central nervous system. *Brain*, 129(Pt 7), 1659–1673.
- de Courten-Myers, G. M. (1999). The human cerebral cortex: Gender differences in structure and function. *Journal of Neuropathology and Experimental Neurology*, 58(3), 217–226.
- Eisenson, J. (1949). Prognostic factors related to language rehabilitation in aphasic patients. *Journal of Speech Disorders*, 14(3), 262–264.
- Frost, J. A., Binder, J. R., Springer, J. A., Hammeke, T. A., Bellgowan, P. S., Rao, S. M., & Cox, R. W. (1999). Language processing is strongly left lateralized in both sexes. Evidence from functional MRI. *Brain*, 122, 199–208.
- Gazzaniga, M. (1970). *The bisected brain*. New York, NY: Appleton.
- Geschwind, N. (1969). Problems in anatomical understanding of aphasias. In A. Benton (Ed.), *Contributions to clinical neuropsychology* (pp. 107–128). Chicago, IL: Aldine.
- Geschwind, N. (1974). Late changes in the nervous system: An overview. In D. Stein, J. Rosen, & N. Butters (Eds.), *Plasticity and recovery of function in the central nervous system* (pp. 467–508). New York, NY: Academic Press.
- Gever, J. (2008). *Progesterone improves head injury recovery*. Retrieved from [www.medpagetoday.com/Neurology/HeadTrauma/9278](http://www.medpagetoday.com/Neurology/HeadTrauma/9278)
- Hallett, M. (1999). Plasticity in the human motor system. *Neuroscientist*, 5, 324–332.
- Heilman, K. M., & Valenstein, E. (2003). *Clinical neuropsychology* (4th ed.). New York, NY: Oxford University Press.
- Heiss, W. D., Kessler, J., Karbe, H., Fink, G. R., & Pawlik, G. (1993). Cerebral glucose metabolism as a predictor of recovery from aphasia in ischemic stroke. *Archives of Neurology*, 50(9), 958–964.
- Herrmann, N., Black, S. E., Lawrence, J., Szekely, C., & Szalai, J. P. (1998). The Sunnybrook Stroke Study: A prospective study of depressive symptoms and functional outcome. *Stroke*, 29(3), 618–624.
- Hirsch, H. V. B., & Jacobson, M. (1974). The perfect of brain. In M. S. Gazzaniga & C. B. Bakemore

- (Eds.), *Fundamentals of psychobiology* (pp. 289–304). New York, NY: Academic Press.
- Hecaen, H. (1976). Acquired aphasia in children and the ontogenesis of hemispheric functional specialization. *Brain and Language*, 3(1), 114–134.
- Iglesias, S., Marchal, G., Rioux, P., Beaudouin, V., Hauttemment, A. J., de la Sayette, V., . . . & Baron, J. C. (1996). Do changes in oxygen metabolism in the unaffected cerebral hemisphere underlie early neurological recovery after stroke? A positron emission tomography study. *Stroke*, 27(7), 1192–1199.
- Jackson, J. (1873). On the anatomical and physiological localization of movements in the brain. *Lancet*, 1, 84–85, 162–164, and 232–234.
- Johansson, B. B. (2000). Brain plasticity and stroke rehabilitation. The Willis lecture. *Stroke*, 31(1), 223–230.
- Jørgensen, H. S., Reith, J., Nakayama, H., Kammersgaard, L. P., Raaschou, H. O., & Olsen, T. S. (1999). What determines good recovery in patients with the most severe strokes? The Copenhagen Stroke Study. *Stroke*, 30(10), 2008–2012.
- Kertesz, A. (1988). What do we learn from aphasia? In S. G. Waxman (Ed.), *Advances in neurology: Functional recovery in neurological disease* (pp. 277–292). New York, NY: Raven Press.
- Kertesz, A., & McCabe, P. (1977). Recovery patterns and prognosis in aphasia. *Brain*, 100, 1–18.
- Kinsbourne, M. (1971). The minor cerebral hemisphere as a source of aphasic speech. *Archives of Neurology*, 25(4), 302–306.
- Kohlmeyer, K. (1976). Aphasia due to focal disorders of cerebral circulation: Some aspects of localization and spontaneous recovery. In Y. Lebrun & R. Hoops (Eds.), *Neurolinguistics: Recovery in aphasics* (pp. 79–95). Amsterdam, the Netherlands: Swets & Zeitlinger.
- Kolb, B., & Whishaw, I. (2003). *Fundamentals of human neuropsychology* (5th ed.). New York, NY: Worth Publishers.
- Lashley, K. (1938). Factors limiting recovery after central nervous lesions. *Journal of Nervous and Mental Diseases*, 88, 733–755.
- Levin, H., & Kraus, M. F. (1994). The frontal lobes and traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 6(4), 443–454.
- Liu, C., & Chambers, W. (1958). Intraspinous sprouting of dorsal route axons. *Archives of Neurology*, 79, 46–61.
- Luria, A. R. (1970). *Traumatic aphasia*. The Hague, Netherlands: Mouton.
- Merzenich, M. M., Kaas, J. H., Wall, J., Nelson, R. J., Sur, M., & Felleman, D. (1983). Topographic reorganization of somatosensory cortical areas 3b and 1 in adult monkeys following restricted deafferentation. *Neuroscience*, 8(1), 33–55.
- Milner, B. (1974). Hemispheric specialization: Score and limits. In F. O. Schmitt & F. G. Worden (Eds.), *The neurosciences: Third study program* (pp. 75–89). Cambridge, MA: MIT Press.
- Moore, R. (1974). Central regeneration and recovery of function: The problem of collateral reinnervation. In D. Stein, J. Rosen, & N. Butters (Eds.), *Plasticity and recovery of function in the central nervous system* (pp. 111–128). New York, NY: Academic Press.
- Pascual-Leone, A., Grafman, J., & Hallett, M. (1994). Modulation of cortical motor output maps during development of implicit and explicit knowledge. *Science*, 263, 1287–1289.
- Pedersen, P. M., Jørgensen, H. S., Nakayama, H., Raaschou, H. O., & Olsen, T. S. (1995). Aphasia in acute stroke: incidence, determinants, and recovery. *Annals of Neurology*, 38(4), 659–666.
- Robinson, R., & Benson, D. (1981). Depression in aphasic patients: Frequency, severity, and clinical pathological correlations. *Brain Language*, 14, 610–614.
- Roof, R. L., Duvdevani, R., & Stein, D. G. (1992). Progesterone treatment attenuates brain edema following contusion injury in male and female rats. *Restorative Neurology and Neuroscience*, 4(6), 425–427.
- Russell, W. R. (1971). *The traumatic amnesias*. London, UK: Oxford University Press.
- Schneider, G. E. (1973). Early lesions of superior colliculus: Factors affecting the formation of abnormal retinal projections. *Brain, Behavior and Evolution*, 8(1), 73–109.
- Serrano, P., Yao, Y., & Sacktor, T. C. (2005). Persistent phosphorylation by protein kinase Mzeta maintains

- late-phase long-term potentiation. *Journal of Neuroscience*, 25(8), 1979–1984.
- Smith, A. (1966). Speech and other functions after left (dominant) hemispherectomy. *Journal of Neurology, Neurosurgery, and Psychiatry*, 29(5), 467–471.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach* (pp. 59–88). New York, NY: Guilford Press.
- Spencer-Smith, M. M., & Anderson, V. A. (2011). Plasticity in a pediatric population. In A. Davis (Ed.), *Handbook of pediatric neuropsychology* (pp. 177–190). New York, NY: Springer Publishing Company.
- Subirana, A. (1969). Handedness and cerebral dominance. In P. Vinken & G. Bruyn (Eds.), *Handbook of clinical neurology* (pp. 248–273). Amsterdam, North Holland: Elsevier.
- Thiel, A., Herholz, K., Koyuncu, A., Ghaemi, M., Kracht, L. W., Habedank, B., & Heiss, W. D. (2001). Plasticity of language networks in patients with brain tumors: A positron emission tomography activation study. *Annals of Neurology*, 50(5), 620–629.
- Woolsey, T. A., & Van der Loos, H. (1970). The structural organization of layer IV in the somatosensory region (SI) of mouse cerebral cortex. The description of a cortical field composed of discrete cytoarchitectonic units. *Brain Research*, 17(2), 205–242.



# Utilizing a Developmental Perspective: The Influence of Age and Maturation on Approach

*Justin J. Boseck, Christopher M. McCormick, and Chad  
A. Noggle*

## EPIDEMIOLOGY—NEUROLOGICAL INJURIES ACROSS THE LIFE SPAN

### Statistics on the Most Common Neurological Injuries

An acquired brain injury (ABI) is a diagnostic category used to describe the presence of damage to the brain from an event that causes neurological complications (U.S. Department of Health and Human Services, 2009). There are two specific types of ABI: traumatic brain injury (TBI) and nontraumatic brain injury (non-TBI). In 1986, the National Head Injury Foundation (NHIF), now known as the Brain Injury Association of America (BIAA), adopted a definition of TBI; TBI is an insult to the brain, not of a degenerative or congenital nature but caused by an external physical force that may produce a diminished or altered state of consciousness, which results in an impairment of cognitive abilities and/or physical functioning. It can also result in the disturbance of behavioral or emotional functioning. These impairments may be either temporary or permanent and cause partial or total functional disability or psychosocial maladjustment. Incidence rates indicate that there are 1.4 million TBI occurrences per year; of these, 4% result in death, 17% result in

hospitalization, and 79% result in treatment and release from an emergency department ([Lash, 2009](#)).

Non-TBIs are caused by an event other than an external force, such as a cerebrovascular accident (CVA; stroke), hypoxia (lack of oxygen to the brain), brain tumor (a form of neurological infection or toxic-metabolic injury) ([Boake, Francisco, Ivanhoe, & Kothari, 2000](#); [Rosenburg, Simantov, & Patel, 2007](#)). In 1997, BIAA adopted a revised definition of ABI to indicate a brain injury that has occurred after birth and is not hereditary, congenital, or degenerative. The injury commonly results in a change in neuronal activity that affects the physical integrity, the metabolic activity, or the functional ability of the cell. The term does not refer to brain injuries induced by birth trauma ([Lash, 2009](#)). Ten million Americans are affected by brain injury when TBI (5.3 million) and non-TBI (4.7 million) are included in prevalence rates. This makes brain injury the second most prevalent injury and disability in the United States ([National Institutes of Health, 1998](#)), behind depression.

## Traumatic Brain Injury

The most prevalent form of acquired brain injuries are TBIs. TBI is estimated to become the leading cause of mortality and morbidity by the year 2020 ([Zitnay, 2005](#)). According to the Centers for Disease Control and Prevention ([CDC; 2011](#)), each year 1.7 million Americans are diagnosed with TBI. Of the 1.7 million cases of TBI seen by medical professionals, approximately 52,000 die, 275,000 are hospitalized, and 1.365 million (nearly 80%) are treated and released from an emergency department ([Faul, Xu, Wald, & Coronado, 2010](#)).

In children, TBIs are the leading cause of postneonatal death ([Kraus, 1995](#)). It is estimated that males are 1.4 times more likely to be diagnosed with a TBI when compared to females. Although TBIs cause close to one third of injury-related deaths in the United States ([Faul et al., 2010](#)); approximately 75% of TBIs that occur each year in the United States result in concussion or other forms of mild TBI (mTBI) ([Centers for Disease Control and Prevention, 2003](#)).

Falls are currently the leading cause of TBI in the United States, occurring in 35.2% of cases of TBI. Among children, falls cause 50% of TBIs between birth to 14 years of age, and 61% of TBI cases in adults over 65 years are caused by a fall-related incident ([Faul et al., 2010](#)). Regardless of the age group, motor vehicle accidents (MVAs) are the second highest known cause of TBI, occurring in 17.3% of TBI diagnoses; MVAs consist of the greatest incidence of TBI-related deaths (31.8%). The third most prevalent known cause of TBI is a

physical strike to the head secondary to hitting a moving or still object (16.5%). Assaults from physical violence or altercations are found in approximately 10% of TBI cases, occurring in 2.9% of children (0–14 years old) and 1% of adults (older than 65 years) (Faul et al., 2010).

## Stroke

CVAs are also common causes of ABI. A CVA occurs when there is acute focal disruption of blood flow or transportation of oxygen vital in normal brain functioning. Approximately 795,000 people are diagnosed with a stroke annually and stroke is one of the leading causes of longterm disability in adults. Stroke is the third highest cause of death in the United States killing about 137,000 people each year (National Stroke Association, 2010).

There are two main types of stroke: ischemic and hemorrhagic. *Ischemic* stroke (also known as arterial ischemic stroke or AIS) occurs when there is an interruption of blood flow from fatty tissue buildup or a formation of a blood clot (National Stroke Association, 2010). Prevalence of ischemic stroke in adults is 180 to 270/100,000 per year (Everts et al., 2008) and accounts for 87% of all cases of stroke (National Stroke Association, 2010). Ischemic stroke is typically rare in childhood; however, ischemic stroke has been found to occur with an annual incidence of 2 to 3 per 100,000 (DeVeber et al., 2000). The greatest chance of an ischemic stroke occurring is perinatally, especially during the 20th to 29th week of gestation through 1 to 4 weeks after birth, and is found to be present in every 1/2,800 to 1/5,000 live-births (Chabrier et al., 2011).

Typically associated with the term *ischemia*, *hypoxia* (or cerebral hypoxia) refers to the reduction of oxygen getting to the brain due to a blockage or disturbance, causing permanent brain damage. Cerebral hypoxia can occur from common events such as carbon monoxide poisoning, partial drowning, suffocation, and resuscitation following myocardial infarction or cardiac arrest, or a pulmonary embolus (Wilson, Harpur, Watson, & Morrow, 2003). *Hemorrhagic* stroke (HS) occurs when blood vessels rupture and blood is dispersed on surrounding brain tissue. Although HS is less frequently seen than ischemic stroke (13% of adult strokes are hemorrhagic in nature), HS accounts for 30% of stroke-related deaths annually (National Stroke Association, 2010) leading to the assumption that HS may largely be considered more severe than ischemic stroke.

## Brain Tumors

The Central Brain Tumor Registry of the United States (CBTRUS) projected the overall prevalence rate of individuals with a brain tumor to be 221.8 per 100,000 (Porter, McCarthy, Freels, Kim, & Davis, 2010). In children and adolescents, brain tumors are seen in approximately 4.28 cases per 100,000 (CBTRUS, 2005). Although it is uncommon for brain tumors to occur in utero, a 2002 study reviewed literature pertaining to 250 cases of perinatal brain tumors (neonatal and fetus) showing a 28% survival rate (Isaacs, 2002). Cognitive, behavioral, and loss of normal brain functioning can all be dramatically affected by the presence of brain tumor in children. These neuropsychological symptoms vary by the location, size, and rate of growth of the brain tumor (Lezak et al., 2004).

Prevalence and severity of ABI varies considerably across the life span. Further insight into developmental differences of brain functioning between children and adults will be looked at thoroughly in this chapter as the impact of ABI on the brain is quite different across various periods of life, from birth to childhood through late adulthood. The influence of brain maturation, rehabilitation techniques, and neurological recovery will also be discussed as these play a large part in the outcome of ABI.

## Childhood Injuries

It has been estimated that approximately 500,000 children experience a head injury every year (Langlois, Rutland-Brown, & Thomas, 2004; Semrud-Clikeman & Bledsoe, 2011). For overall rates of TBI (including emergency department visits, hospitalizations, and deaths) three of the top four incidence rates were in the age groups of children between the ages of 0 to 4 (1121 per 100,000), 15 to 19 (814 per 100,000), and 5 to 9 (659 per 100,000) (Lash, 2009). Early research in brain injury led many researchers to assume that brain injury early in life lead to much better outcomes than those brain injuries sustained later in life. For instance, the following passage was common thought on brain injury fewer than 10 years ago:

Brain damage following TBI is less common and less severe in infants and children than adults, but it depends upon the extent of vascular injury and edema. (Birmaher & Williams, 1994)

More progressive literature on this topic indicates that childhood brain injury is just as deleterious on longterm functioning as the same injury occurring later in life. Sometimes it takes much longer for the effects of trauma to be seen in children because their brains are still developing. According to Lash (2009):

Preschoolers with injuries to their frontal lobes often look fine within a few weeks or months after an injury. However, as they get older and their brains mature, that part of the brain previously damaged may not work as well as it should. When a child's brain is injured, it can have longterm devastating effects on the child and family. Too often, children who sustain a brain injury early in life may look "well" at that moment in time, but more serious cognitive and behavioral problems may emerge as the child grows.

Brain injury later in life brings about a common constellation of symptoms. Common symptoms of brain injury later in life include problems in information processing, attention, and memory (Felmingham, Baguley, & Green, 2004; & Semrud-Clikeman & Bledsoe, 2011).

*Focal Versus Diffuse.* More than 90% of pediatric brain injuries are nonpenetrating and closed and are clinically manifested by alterations of consciousness (Birmaher & Williams, 1994). A closed head injury is caused when the child's head strikes a hard surface or if the child is shaken such as in child abuse (coup-contra-coup; Semrud-Clikeman & Bledsoe, 2011). Closed head injuries may involve either diffuse (widespread) or focal damage. In *focal* damage, the injury is due to the impact of the head on an object and is generally limited to a discrete region of the brain, whereas white or gray matter in the brain is sheared due to force during a *diffuse* injury (Semrud-Clikeman & Bledsoe, 2011). Diffuse axonal injury results in individual nerve cells stretching and breaking throughout the brain (Lash, 2009) and results in extensive injury to the entire brain.

*Early Insult versus Later in Childhood.* There are five distinct brain maturation milestones that have been identified through extensive developmental study and specialized measures such as neuropsychological testing, magnetic resonance imaging (MRI), and special uses of electroencephalography (EEG). Peak maturation of the brain according to percentage of maturation increments generally include the years 2 to 3, when there is 6% maturation growth; age 7 to 8, when there is 3% maturation growth; age 12, when there is 1.8% maturation growth; age 15, when there is 3% maturation growth; and age 19, when there is approximately 2% maturation growth (Lash, 2009). The greatest percentage of brain maturation occurs in the early years, birth through age 5, and especially during the 2-to 3-year age range. Thus, injury to a child's brain before age 5 may be the most devastating time to sustain an injury. This leads neuroscientists to believe that injury to the brain early in life may be more severe, especially when based upon these five peaks in brain maturation. This is contrary to previously held beliefs that suggested earlier age of insult is related to better outcomes

because of the plasticity of the brain, providing a greater likelihood of neurological reorganization. Brain injury during these critical periods may be more significant and lead to more severe outcomes.

As this chapter discusses the role of development in TBI, it is quite pertinent to discuss the work of some of the founding psychologists who have helped to shape this field of research, including noted neuropsychologist and developmental psychologist Alexander Luria. His work and influence from Lev Vygotsky laid the groundwork for many of the concepts that still stand today regarding development (i.e., language development, writing, and behavior) and neuropsychological principles. In fact, Luria's work in patients with brain lesions in Russia during World War II, according to some, formed the field of neuropsychology. Other prominent psychologists who will be discussed either directly or indirectly in this chapter were prominent contributors to the fusion of the topics of brain injury and development, including John Hughlings Jackson, Karl Lashley, Jean Piaget, and Erik Erikson, among others.

## DEVELOPMENTAL DIFFERENCES

### Neurological Differences Between Children and Adults

#### Neurons, Synapses, and Pruning

The nervous system is composed of two basic kinds of cells, neurons and glia. There are approximately 80 billion neurons and 100 billion glia in the human nervous system (Kolb & Cioe, 2004). *Neurons* are the basic nerve cells in the central nervous system (CNS) that respond to excitatory and inhibitory responses such as attention, memory, thinking, and reasoning. The development of the human brain across the life span is complex and varies on several factors that impact neurologic maturation. This holds true for structural differences in the brain from childhood into adulthood. At birth, an infant's brain weighs approximately 25% of its total body weight, an infant's brain weighs 75% of its mature adult weight by age 2, and approximately 90% of adult weight by age 5 (Sigelman & Rider, 2009).

Communication between neurons is completed through *synapses*, which are microscopic junctions for axons and dendrites to connect for transmission of electrical and chemical messages crucial to brain functioning. The amount of synapses in the brain is astounding, in the trillions, creating vast amounts of vital connections for neurological communications (Sielger, Deloache, & Eisenberg,

2006). Interestingly, the increased development of synaptic connections in the developing brain during childhood eventually slows down through a process called synaptic *pruning*, with more than half of synaptic connections being lost by puberty. This form of cell death eliminates the abundance of “unused” neurons and synapses in the brain during childhood (Chechik, Meilijson, & Ruppin, 1999). The prevalence of neurons and synapses tends to decrease with normal aging, with elderly adults losing up to 30% of neurons that were present as young adults (Sigelman & Rider, 2009). As can be seen by the vast amount of change that occurs in the brain throughout the life span, the impact of brain injury may be quite disparate depending on when the injury occurs during the life span.

## Intellectual Differences

### Concrete and Abstract Thought Processing

Maturation of the brain during childhood and adulthood helps to define differences in intellectual ability in regard to both concrete and abstract thought processes. Piaget is one theorist among many who developed a theory of cognitive development across the life span (for a thorough review of Piaget’s theory of cognitive development refer to *Genetic Epistemology*, Piaget, 1970). Piaget’s theory of cognitive development follows the belief that children develop through four distinct stages of maturation in regard to thinking: sensorimotor stage, preoperational stage, concrete operational stage, and the formal operational stage. The final two stages involve development of concrete and abstract thinking processes. Although Piaget’s theories were not based in neuroscience, these developmental stages have since been found to correlate well with typical brain development throughout adolescence and as such his theory still currently proves relevant in the field of development and neuroscience.

Luria’s theory did not view any complex higher cortical functions as products of a particular tissue or organ, but as the coordination of several different brain areas. Luria combined elements of localization, equipotentiality theory, and the work of Hughlings Jackson to create a conceptualization of a normally functioning brain as an integration of three units. The first unit regulates activation, muscle tone, and vigilance and consists of the reticular formation, limbic system, and mesial basal frontal lobes. Injuries to the first unit can result in lethargy and apathy, which will impair higher cortical systems, even

though the areas related to higher cortical functions may remain intact. The second unit is responsible for registration, analysis, and the storage of sensory information and is comprised of the temporal, parietal, and occipital lobes. The third unit regulates complex mental activity, such as planning, abstract thought, and organization. This unit is dependent upon the integrity of the frontal lobes. All activities depend upon the cooperation of all the three units. Luria's theory hypothesizes that when a functional system ceases to operate correctly as a result of an injury, it is not obvious which cerebral structure in the brain is impaired. For example, a brain-injured patient may not be attending (unit 1), may not be able to analyze relevant stimuli (unit 2), or may not be actively trying to use the information; all three of these possibilities will have similar functional presentations (Gouvier et al., 1997). No single unit or area of the brain is solely responsible for the execution of any activity. To assess which system is impaired, Luria proposed testing a series of hypotheses that call upon each unit to sequentially demonstrate its integrity, which is a pioneering thought on the commonly held belief today that the whole brain works in concert to create human behavior. Luria's idea of harmonious processing remains very relevant today, decades after he first published his ideas.

From a developmental standpoint, Luria (1966, 1973) introduced the systemic–dynamic approach in analysis of brain organization of higher mental functions, which was an extension of Vygotsky's developmental theory in many ways, which posited that biological traits of the host are nurtured by the environment and experiences, including cultural influences that result in a development of the mind (Vygotsky, 1978). Luria (1973) proposed that higher mental functions were social in origin and complex and hierarchical in their structure. That is, Luria's concept of extracortical organization of complex mental functions suggests that external factors serve to establish functional connections between various brain systems. As individuals grow and have more experiences within their environment, further "organization" (i.e., cognitive growth) occurs. Luria purported that engagement toward external artifacts served as intrinsic factors in systemic organization. This was best exemplified in Luria's approach to rehabilitation. For example, when rehabilitating motor functioning, in lieu of simply focusing on free movement, he added objects into the mix whereby individuals would attempt to move toward them (e.g., reaching, grasping) or move them (e.g., using a tool). This added purpose, was viewed as offering wider spread reorganization.

As suggested, Luria's theory was an extension of Vygotsky's Social



Development Theory. Vygotsky, in *The Problem of Age* (1934), outlined many of the flaws behind dividing development into periods (for a thorough examination see, [Vygotsky & Blunden, 1998](#)). The primary emphasis of Vygotsky's theory was that cognitive development is a by-product of social interaction and that social learning led to development itself. Through observation, Vygotsky purported that a function first manifests vicariously as children observe the function between people (i.e., interpsychological) who have a greater understanding and application of the higher level ability, which Vygotsky termed "the more knowledgeable other" (MKO), that is later internalized by the child (i.e., intrapsychological; [Vygotsky, 1978](#)). Vygotsky referred to zones of proximal development that represented the distance between what an individual could do under supervision and what the individual could do by himself. Through play and interaction with their environment and culture, individuals develop higher level skills in thinking and communication.

## Stability of Cognition

Cognitive stability throughout the life span and into late adulthood is particularly interesting, especially due to the early development of many concepts in childhood, adolescence, and early adulthood, as previously recognized by the reviews of Piaget, Luria, and Vygotsky. These theorists outlined changes that occur throughout childhood but were unable to quantitatively support their hypotheses. More recent literature has been able to reliably quantify decline in cognitive ability throughout the life span.

One study conducted by [Schaie and colleagues \(1993\)](#) found noticeable declines in cognitive ability from previously established intelligence measured in young adulthood, showing declines in all measured abilities by age 74 ([The Seattle Longitudinal Study; Schaie, 1993](#)). [Gow and colleagues' \(2011\)](#) evaluations of the Lothian Birth Cohorts of 1921 and 1936 have also offered great insights into how cognitive ability from childhood carries over into adulthood. Investigation of cognitive functioning across a 76-year time span found that previous scores on the Moray House Test No. 12 (MHT) at age 11 were stable for the Lothian Birth Cohorts of 1936 when retaken at age 70 and for the Lothian Birth Cohorts of 1924 at ages 79 and 87. Information previously established in this chapter indicated a growth in the ability to gain higher cognitive abilities peaking at around the mid-20s, when the prefrontal cortex becomes fully developed. This new information indicating that higher cognitive abilities begin to decline in the mid-70s indicates that there is a definite normal

curve to cognitive ability throughout the life span.

## Functional Differences

### Research Regarding the Physiological Differences in White/Gray Matter

Research regarding structural and functional differences between a child's and adult's brain has been increasing due to the development of neuroimaging techniques such as positron emission tomography (PET) and MRI (Frith & Frith, 2008). Evaluating the two major components of the CNS, including *white matter* (bundles of myelinated axons) and *gray matter* (neuronal cell bodies) has helped researchers discover how brain structure changes across the life span occur with brain maturation (Haines, Ralia, & Terrell, 2002).

Further research into differences of both gray and white matter in the human brain across the life span has been helpful in discerning structural and functional differences in the brain. Gray matter volume is found to peak during childhood and typically decreases in adulthood, whereas the overall volume of white matter has been shown to increase into young adulthood (Groeschel, Vollmer, King, & Connelly, 2010). When looking at a sample of normal, healthy adults aged 20 to 86, Ge and colleagues (2002) discovered that gray matter volume appeared to begin declining at a constant and linear rate at around age 20. An increasing quadratic pattern of change of white matter volume in the participants was present as well, showing a decline after age 40. These statistics further support the hypothesis that brain maturation peaks in the 20s and begins a constant decline thereafter.

### Development of Lobes

Not only is the brain undergoing significant changes in architecture during childhood and adolescence, it also may compensate for these changes by distributing information processing in a manner that is different from what is observed in adults (Wahlstrom & Luciana, 2011). Therefore, the adult brain and child brain are unable to be compared in parallel. For instance, a decade-long MRI study of normal brain development, from ages 4 to 21, by researchers at the National Institute of Mental Health and University of California Los Angeles (UCLA) has shown that the prefrontal cortex does not fully develop until young adulthood, as gray matter wanes in a back-to-front wave as the brain matures and neural connections are pruned (Thompson, 2011). From this evidence alone, we can infer that it is impossible to reliably compare the neuroanatomical

functioning of children and adults. By extension, it cannot be inferred that injury to the developing brain will result in the same impairments as would be observed under similar circumstances in adults (Wahlstrom & Luciana, 2011). Therefore, when analyzing social, emotional, and behavioral outcomes based on brain injury between children and adults, the neuroanatomical functioning of these outcome constructs must be understood to occur in isolation when factoring in age.

### **Social, Emotional, and Behavioral Differences**

Studies have reported that during the normal aging process individuals may experience memory impairment, slower learning of new material, gait and balance problems, decreased sensory acuity, diminished executive functions, and reductions in appetite and libido (Goldstein & Shelly, 1975; Lash, 2009). It was thought by Goldstein and Shelly (1975) that the differences in these abilities in those with brain injury versus their same-aged peers would diminish; however, individuals with brain injury reported significantly more impairment than their peers in all areas of functioning, including the social, emotional, and behavioral realms. Several longterm studies have also reported that, after brain injury, persons reported persistent problems with behavior disorders, personality changes, learning and memory deficits, psychosocial readjustment issues, social isolation, chronic unemployment, and major psychiatric disorders (especially depression; Hibbard, Uysal, Sliwinski, & Gordon, 1998; Lash, 2009; O'Neil et al., 1998; Trudel & Purdum, 1998).

Differences in social, emotional, and behavioral aspects associated with the continuous development and maturation across the life span of the human brain is important to understand when dealing with concepts that may or may not have developed in a person with an ABI. According to Erikson's psychosocial theory, self-identity development occurs through biological, social, and psychological "crisis" that helps individuals transition through eight specific stages (for a thorough analysis see, *Identity and the Life Cycle* by Erikson, 1959). According to Erikson, children differ socially from adults in the sense that they have not yet developed the biological means or have experienced the social influence to successfully resolve particular life crises.

## **INFLUENCE OF AGE AND MATURATION ON INJURY**

### **Plasticity and Neurological Recovery**

In order to study the effect that development has on brain injury the history of brain injury research must first be discussed. There is a history of brain injury research dating back to the late 1800s. John Hughlings Jackson, as told by [Prigatano \(1999\)](#) in a historical review, speculated on the field of neurological rehabilitation as early as 1888 in a statement he made; “Why do patients recover from hemiplegia when the loss of nerve tissue is permanent?” The field of neurological recovery from injury has vexed brain scientists for centuries. More recently, Alexander [Luria \(1964\)](#) posited, “Tissue destruction produces irreversible loss of function” and reversible disturbances of function can be explained through proper treatment (rehabilitation) efforts, including psychotherapy and psychopharmacology, as well as a heavy emphasis on brain reorganization. These early views and more recent research on brain injury have merged to yield the conclusion that the brain is capable of rewiring and altering function to improve its ability to adapt and meet the demands of one’s environment over time.

*Neuroplasticity* forms the basis of the field of neurorehabilitation as we know it. If it were not for neuroplasticity, there would be very little improvement in the functioning of individuals with brain lesions. The brain’s ability to rewire and alter brain tissue for the purpose of adapting to changes externally or internally is called *plasticity* ([Bhatnagar, 2008](#)). Different forms of plasticity exist. *Structural plasticity* includes the change of physical structures by the brain due to environmental stimuli or injury, whereas *functional plasticity* is the brain’s ability to alter function from one area to another due to damage. *Activity-dependent plasticity* refers to changes in the brain’s ability to adapt to the environment based on activities that are performed that result in changed gene expression secondary to organized cellular mechanisms ([Flavell & Greenberg, 2008](#)). *Experience-dependent plasticity* refers to plasticity in areas of the brain that are highly used on a daily basis such as a typist having especially large differences in dendritic cells in the trunk and finger neurons of the brain ([Lezak, 2004](#)).

## Age and Neuropsychological Sequelae

### Individual Neurocognitive Domains

[Anderson et al. \(2009\)](#) looked further into the impact of early brain insult (EBI) on outcomes in cognitive function and its relation to time of brain injury. They found that children with brain injury before the age of 2 had significantly lower

scores in all domains on the Wechsler Abbreviated Scale of Intelligence ([WASI; Wechsler, 1999](#)) when compared to children injured after age 7. This supports the notion that brain insult earlier in life may have a greater effect on cognitive outcome compared to children with brain injuries later in life. Similar findings were noted by [Max and colleagues \(2010\)](#) in regard to their findings that cognitive outcomes in children experiencing a stroke prenatally or up until age 1 year is associated with poorer performance on a variety of neurocognitive measures (Test of Word Language, Third Edition [TOWL-3], Beery-Buktenica Developmental Test of Visual Motor Integration [VMI], Rey-Osterrieth Complex Figure [REY-O]) spanning multiple domains such as language, visuospatial functioning, and memory, respectively, when compared to children with later-onset stroke (mean age at stroke = 7.8).

## Academics

For a child with a brain injury the transition back into the academic environment can be very difficult. Students may need to reintegrate into school on a part-time basis or they may need in-home instruction for a period of time ([Lash, 2009](#)). There are many areas of functioning that may be impacted after a brain injury that may be exacerbated in the academic environment, including difficulty with memory, attention and concentration, higher level problem-solving (executive functioning, EF), language skills, sensorimotor abilities, and behavior and emotional abilities. The largest provider of services to children with brain injury are the schools. In October 1990 a category of “TBI” for students requiring special education services was authorized under the Individuals with Disabilities Education Act (IDEA; Public Law 101–476). Through IDEA, children with TBI may qualify for special services such as a 504 plan, which requires schools receiving federal funding to provide reasonable accommodations to allow an individual with a disability to participate. A child with a TBI may also qualify for special education services based on need through the use of an individualized education plan (IEP). An IEP is a tool that describes what help the student will be given. The IEP is a legally binding document between the school and the caregivers of the student that identifies the skills, strategies, and behaviors that the student needs to learn and function at school.

[DePompei and colleagues \(1999\)](#) identified many important areas of functioning that should be addressed in an IEP for a child with a TBI based on functional deficits:

## Cognitive Impairment

How can those working with this student:

1. Help the student pay attention and concentrate?
2. Get started in activities and work?
3. Become organized and plan ahead?
4. Reason and problem-solve?
5. Learn new information?
6. Recall previously learned information?
7. Communicate clearly and effectively in speech and writing?
8. Make good and safe decisions?
9. Be flexible and adjust to change?

## Behavioral/Adaptive Skills Deficits

How can staff working with this student:

1. Help the student with self-esteem and self-control?
2. Increase awareness of how feelings affect others?
3. Increase knowledge of expectations in social situations?
4. Increase awareness of appropriate hygienic practices?
5. Increase ability to control sexual comments, gestures, and actions?
6. Improve ability to handle frustration and control anger?

## Physical/Sensory Issues

How can staff working with the student help the student:

1. Compensate for changes in vision and hearing?
2. Detect changes in sound, height, distance, and touch?
3. Adjust to changes in body coordination?
4. Slow down or speed up movements?
5. Improve balance and steadiness?
6. Recognize and handle fatigue?
7. Improve eye and hand coordination?

## **Impact on Domains of Life**

A person with a brain injury must reacclimate to every area of their environment after the injury. Daily tasks and skills that once seemed ordinary and routinized may soon become very difficult and require extreme concentration and perseverance. Many people with brain injury develop comorbid psychiatric illnesses such as depression, anxiety, posttraumatic stress disorder (PTSD), situational stress, and substance abuse, which may be secondary to these new challenges in their daily lives. These illnesses often aggravate the person's brain injury and lead to further life complications.

### **Employment**

Adults share many of the same struggles when entering back into their daily lives as children do when they begin reentry into school. One of the main areas of friction that a person with a brain injury must navigate after injury is the work environment. If a person with a brain injury is prepared to become employed as is deemed capable, they may require assistance based on their pattern of strengths and weaknesses. There are many areas of effective programming that are essential to consider when helping a person reenter the work environment. Areas of deficit that need to be considered when planning for reentry into employment include memory impairments, executive functioning deficits, language deficits, aggression, verbal outbursts, social skills deficits, and difficulty with the routine maintenance of daily activities. Assisted employment programs can be a great service for individuals with brain injury to help identify and accommodate to each of these areas of deficit. For instance, memory impairments may be analyzed through short-term situational supported employment and remedied through the use of a written diary of events in the form of a daily planner. Executive functioning deficits may be remedied through task analysis procedures. Language deficits could be assisted through the use of word cards to facilitate effective communication. Finally, social skills deficits may be aided through modeling and role-playing in a therapeutic environment.

## **REHABILITATION FROM A DEVELOPMENTAL PERSPECTIVE**

### **Approaches to Rehabilitation**

There are many different types of rehabilitation programs for people who acquire a brain injury. The type of program that each person would benefit from

varies depending on their cognitive capability and current level of acceptance with the injury. The type of program will also vary with the person's age, maturation level, and the setting that they hope to return to after rehabilitation (school vs. work).

There are many systems of care that can be utilized by persons with brain injuries depending on the phase of their recovery and the extent of their injury. Initial hospital-based services include acute hospital care and acute rehabilitation. Posthospital services include skilled nursing facilities, postacute rehabilitation facilities, outpatient services, and supported living environments. The goals of postacute rehabilitation facilities are to help rehabilitate impairments in memory, cognition, executive functioning, initiation, speech and language, sensorimotor abilities, behavioral and emotional changes, and substance abuse.

Lash (2009) identified eight steps for effective rehabilitation:

1. Identify important skills that an individual currently can do independently.
2. Help the individual develop a realistic longterm plan, including where and how to live.
3. Determine what the individual needs to accomplish to achieve the plan.
4. Identify longterm goals.
5. Break longterm goals into specific short-term objectives.
6. Design a plan for helping the individual meet the objectives.
7. Evaluate the progress on the basis of measurable outcome criteria.
8. Review outcome criteria and revise as necessary.

Different types of neurorehabilitation systems have been established. The goal of *restorative neurorehabilitation* is to achieve functional improvements by reinforcing, strengthening, or reestablishing previously learned patterns of behavior. The goal of *compensatory neurorehabilitation* is to enable functional improvement by “establishing new patterns of cognitive activity or compensatory mechanisms for impaired neurological systems” (Harley et al., 1992). Finally, the goal of *environmental modification neurorehabilitation* is to focus on altering the external environment rather than the individual (Mateer & Raskin, 1999) to establish functional improvement. Given the plethora of types of neurorehabilitation programs and specific areas of focus in each, the age of the person with the brain injury will largely drive the type and goal of each



specialized neurorehabilitation program.

## Engagement and Compliance

A person's engagement and compliance with the rehabilitation program and facility is a major factor in their ability to benefit from the program. Initially after a brain injury, a person may have posttraumatic amnesia, confusion, an unawareness of their deficits (anosognosia), and their primary focus may be on reestablishing the activities of daily living. People with brain injury may also refuse treatment, have life upheaval that reduces their compliance with treatment, and financial setbacks that get in the way of effective treatment.

Klonoff (2010) described a period of disintegration that can occur at any stage throughout rehabilitation, which can lead to setbacks, including:

- I can't get the therapies I need/want because of insurance problems.
- I can't get the therapies I need/want because I adamantly reject these services.
- I refuse to become aware and acceptant.
- I negate my reality.
- I am filled with rage, despair, hopelessness, and helplessness.
- I am suspicious and argumentative.
- I become socially isolated and alienated from my friends and family.
- I am dependent on others in the home community.
- I experience work failures and financial ruin.
- There is fragmentation in my family life.

Disintegration can occur when a person is going through the beginning stages of rehabilitation or after a person has been discharged from rehabilitation. Any of these setbacks can lead to personal failures in a person's life, leading to catastrophic consequences. This is why a person with a brain injury, no matter how severe, must be monitored at all stages throughout their recovery even after they have left formal rehabilitation.

## Reentry and Conclusion

Permanent reentry into the community after a brain injury is always a main priority and longterm goal for people who suffer a brain injury as well as and for their care providers. The main issue regarding successful reentry into society is

insufficient personal investment and ownership over treatment, which, if mishandled, will have a negative impact on progress. There are many issues that must be confronted for successful reentry to occur, including, self-doubt, anxiety, inadequate coping responses, unwillingness or inability to develop a social network, unrealistic goals, inability to adapt to changing demands, and an unwillingness to seek help when necessary. The previously mentioned methods of neurorehabilitation and the specific methods that can be used throughout these systems can help to address each of these issues.

## SUMMARY

ABIs arise from both traumatic and nontraumatic forces. Not only are variations seen in the etiology of such injuries, but variations are also seen in their functional picture. The literature commonly discusses the role of injury type, site, and severity in the manifestation of neuropsychological sequelae in ABIs. The age of onset is one area that is not discussed as frequently, but is critical; not only in terms of the potential residuals of the neurological insult itself, but also in the rehabilitative efforts pursued after the fact. Therefore, neuropsychologists, rehabilitation psychologists, and other professionals must maintain a developmental perspective when designing and carrying out comprehensive rehabilitation plans for individuals with ABIs.

## REFERENCES

- Anderson, V., Spencer-Smith, M., Leventer, R., Coleman, L., Anderson, P., Williams, J., . . . & Jacobs, R. (2009). Childhood brain insult: Can age at insult help us predict outcome? *Brain*, *132*(Pt 1), 45–56.
- Bhatnagar, S. C. (2008). *Neuroscience for the study of communicative disorders* (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Birmaher, B., & Williams, D. T. (1994). Children and adolescents. *Neuropsychiatry of traumatic brain injury* (pp. 393–412). Edited by J. M. Silver, S. C. Yudofsky, & R. E. Hales. Washington, DC: American Psychiatric Press.
- Boake, C., Francisco, G. E., Ivanhoe, C. V., & Kothari, S. (2000). Brain injury rehabilitation. In R. Braddom (Ed.), *Physical medicine and rehabilitation*. Philadelphia, PA: WB Saunders.
- Brain Injury Association of America. (2009). *Facts about traumatic brain injury*. Retrieved from [www.biausa.org/aboutbi.htm](http://www.biausa.org/aboutbi.htm)
- Center for Brain Tumor Registry of the United States. (CBTRUS) (2005). *Statistical report: Primary brain tumors in the United States, 1998–2002*.
- Centers for Disease Control and Prevention. (2003). *Report to Congress on mild traumatic brain injury in the United States: Steps to prevent a serious public health problem*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Chabrier, S., Husson, B., Dinomais, M., Landrieu, P., & Nguyen The Tich, S. (2011). New insights (and new interrogations) in perinatal arterial ischemic stroke. *Thrombosis Research*, *127*(1), 13–22.
- Chechik, G., Meilijson, I., & Ruppin, E. (1999). Neuronal regulation: A mechanism for synaptic pruning during brain maturation. *Neural Computation*, *11*(8), 2061–2080.

- DePompei, R., Blosser, J., Savage, R., & Lash, M. (1999). *Back to school after a moderate to severe brain injury*. Wake Forest, NC: Lash and Associates Publishing/Training Inc.
- deVeber, G., Roach, E. S., Riela, A. R., & Wiznitzer, M. (2000). Stroke in children: Recognition, treatment, and future directions. *Seminars in Pediatric Neurology*, 7(4), 309–317.
- Erikson, E. (1959). *Identity and the life cycle*. New York, NY: International Universities Press.
- Everts, R., Pavlovic, J., Kaufmann, F., Uhlenberg, B., Seidel, U., Nedeltchev, K., Perrig, W., & Steinlin, M. (2008). Cognitive functioning, behavior, and quality of life after stroke in childhood. *Child Neuropsychology*, 14(4), 323–338.
- Faul, M., Xu, L., Wald, M. M., & Coronado V. G. (2010). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations and deaths 2002–2006*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Felmingham, K. L., Baguley, I. J., & Green, A. J. (2004). Effects of diffuse axonal injury on speed of information processing following severe traumatic brain injury. *Neuropsychology*, 19, 105–118.
- Flavell, S. W., & Greenberg, M. E. (2008). Signaling mechanisms linking neuronal activity to gene expression and plasticity of the nervous system. *Annual Review of Neuroscience*, 31, 563–590.
- Frith, C., & Frith, U. (2008). What can we learn from structural and functional brain imaging? In M. Rutter et al., (Eds.), *Rutter's child and adolescent psychiatry* (5th ed., pp. 134–144). Cambridge, MA: Wiley-Blackwell.
- Ge, Y., Grossman, R. I., Babb, J. S., Rabin, M. L., Mannon, L. J., & Kolson, D. L. (2002). Age-related total gray matter and white matter changes in normal adult brain. Part I: volumetric MR imaging analysis. *American Journal of Neuroradiology*, 23(8), 1327–1333.
- Giroud, M., Lemesle, M., Gouyon, J. B., Nivelon, J. L., Milan, C., & Dumas, R. (1995). Cerebrovascular disease in children under 16 years of age in the city of Dijon, France: A study of incidence and clinical features from 1985 to 1993. *Journal of Clinical Epidemiology*, 48(11), 1343–1348.
- Goldstein, G., & Shelly, C. H. (1975). Similarities and differences between psychological deficits in aging and brain damage. *Journal of Gerontology*, 30(4), 803–806.
- Gouvier, W. D., Ryan, L., O'Jile, J. R., Parks-Levy, J., Webster, J. S., & Blanton, P. D. (1997). Cognitive retraining with brain-damaged patients. *Neuropsychology Handbook*, 2, 3–46.
- Gow, A. J., Johnson, W., Pattie, A., Brett, C. E., Roberts, B., Starr, J. M., & Deary, I. J. (2011). Stability and change in intelligence from age 11 to ages 70, 79, and 87: The Lothian Birth Cohorts of 1921 and 1936. *Psychology and Aging*, 26(1), 232–240.
- Groeschel, S. S., Vollmer, B. B., King, M. D., & Connelly, A. A. (2010). Developmental changes in cerebral grey and white matter volume from infancy to adulthood. *International Journal of Developmental Neuroscience*, 28(6), 481–489.
- Haines, D. E., Ralia, F. A., & Terrell, A. C. (2002). Orientation to structure and imaging of the central nervous system. In D. E. Haines (Ed.), *Fundamental neuroscience*. Philadelphia, PA: Churchill Livingstone.
- Harley, J. P., Allen, C., Braciszewski, T. L., Cicerone, K. D., Dahlberg, C., Evans, S., . . . Smigelski, J. S. (1992). Guidelines for cognitive rehabilitation. *Neuro Rehabilitation*, 2, 62–67.
- Hibbard, M. R., Uysal, S., Sliwinski, M., & Gordon, W. A. (1998). Undiagnosed health issues in individuals with traumatic brain injury living in the community. *Journal of Head Trauma Rehabilitation*, 13(4), 47–57.
- Isaacs, H. (2002). Perinatal brain tumors: a review of 250 cases. *Pediatric Neurology*, 27(4), 249–261.
- Kennard, M. A. (1936). Age and other factors in motor recovery from precentral lesions in monkeys. *American Journal of Physiology*, 115, 138–146.
- Klonoff, P. S. (2010). *Psychotherapy after brain injury: Principles and techniques*. New York, NY: Guilford Press.
- Kolb, B., & Cioe, J. (2004). Neuronal organization and change after neuronal injury. In J. Ponsford (Ed.), *Cognitive and behavioral rehabilitation: From neurobiology to practice*. New York, NY: Guilford Press.
- Kraus, J. F. (1995). Epidemiological features of brain injury in children: Occurrence, children at risk, causes

- and manner of injury, severity and outcomes. In S. H. Broman & M. E. Michel (Eds.), *Traumatic head injury in children*. New York: Oxford University Press.
- Kraus, J. F., & Chu, L. D. (2005). Epidemiology. In J. Silver, T. McCallister, & S. Yudofsky (Eds.), *Textbook of traumatic brain injury*. Washington, DC: American Psychiatric Publishing.
- Langlois, J. A., Rutland-Brown, W., & Thomas, K. E. (2004). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Lash, M. (2009). *The essential brain injury guide* (4th ed.). Vienna, VA: Brain Injury Association of America.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment*. New York, NY: Oxford University Press.
- Luria, A. R. (1964). Restoration of function after brain injury. *California Medicine*, 101(1), 70–71.
- Luria, A. R. (1966). Higher cortical functions in man. Basic Books.
- Luria, A. R. (1973). *The working brain*. Basic Books.
- Mateer, C. A., & Raskin, S. (1999) Cognitive rehabilitation. In M. Rosenthal, E. Griffith, J. S. Kreutzer, & B. Pentland (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 254–270). Philadelphia, PA: FA Davis.
- Max, J. E., Bruce, M., Keatley, E., & Delis, D. (2010). Pediatric stroke: plasticity, vulnerability, and age of lesion onset. *Journal of Neuropsychiatry and Clinical Neurosciences*, 22(1), 30–39.
- National Institutes of Health. (1998). *National Consensus Conference*. Centers for Disease Control and Prevention National Incidence Data for 1995–1996 and Preliminary Data from the Colorado Traumatic Brain Injury Registry from 1996–1997.
- National Stroke Association. (2010). *Stroke 101*. Retrieved from [http://www.stroke.org/site/docServer/STROKE\\_101\\_Fact\\_Sheet.pdf?docID=4541](http://www.stroke.org/site/docServer/STROKE_101_Fact_Sheet.pdf?docID=4541).
- Piaget, J. (1970). *Genetic epistemology*. New York, NY: Columbia University Press.
- Porter, K. R., McCarthy, B. J., Freels, S., Kim, Y., & Davis, F. G. (2010). Prevalence estimates for primary brain tumors in the United States by age, gender, behavior, and histology. *Neuro-oncology*, 12(6), 520–527.
- Prigatano, G. P. (1999). *Principles of neuropsychological rehabilitation*. New York, NY: Oxford University Press.
- Rosenburg, C. H., Simantov, J., & Patel, M. (2007). Psychiatry and acquired brain injury. In J. Elbaum & D. Benson (Eds.), *Acquired brain injury: An integrative neurorehabilitation approach*. New York, NY: Springer Science+Business Media.
- Schaie, K. (1993). The Seattle longitudinal studies of adult intelligence. *Current Directions in Psychological Science*, 2(6), 171–175.
- Semrud-Clikeman, M., & Bledsoe, J. C. (2011). Traumatic brain injury in children and adolescents. In A. Davis (Ed.). *Handbook of pediatric neuropsychology*. New York, NY: Springer Publishing.
- Sielger, R, Deloache, J, & Eisenberg, N. (2006). *How children develop*. New York, NY: Worth Publishers.
- Sigelman, C. K., & Rider, E. A. (2009). *Life-span human development* (6th ed.). Belmont, CA: Wadsworth/Cengage Learning.
- Thompson, P. (2011). *Time-lapse imaging tracks brain maturation from ages 5 to 20*. Taken from The adolescent brain—Why teenagers think and act differently; Retrieved from [http://www.edinformatics.com/news/teenage\\_brains.htm](http://www.edinformatics.com/news/teenage_brains.htm)
- Trudel, T. M., & Purdum, C. M. (1998). Aging with brain injury: Longterm issues. *Rehabilitation Professional*, 6, 37–41.
- U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Neurological Disorders and Strokes. (2009). *Traumatic brain injury page*. Retrieved from <http://www.ninds.nih.gov/disorders/tbi/tbi.htm>
- Vygotsky, L. (1978). *Mind in society: The development of higher psychological processes*. Harvard University Press.

- Vygotsky, L. S., & Blunden, A. (1998). *The collected works of L. S. Vygotsky*, 5, 187–205.
- Wahlstrom, D., & Luciana, M. (2011). Functional neuroanatomy of the cerebral cortex. In A. Davis (Ed.), *Handbook of pediatric neuropsychology*. New York, NY: Springer Publishing.
- Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, TX: Pearson Assessments.
- Wilson, F. C., Harpur, J., Watson, T., & Morrow, J. I. (2003). Adult survivors of severe cerebral hypoxia—case series survey and comparative analysis. *Neuro Rehabilitation*, 18(4), 291–298.
- Zitnay, G. A. (2005). Lessons from national and international TBI societies and funds like NBIRTT. *Acta Neurochirurgica Supplement*, 93, 131–133.

# Rehabilitation of Attention and Executive Function Deficits

*Kaaren Bekken and L. Lynn LeSueur*

## DEFINING THE TERMS: CONCEPTUAL AND BIOLOGICAL BACKGROUND

The flurry of research in recent years addressing attention and executive function has increased our overall understanding of cognitive function, though competing models of these operations and their supporting neurology reveal an as yet incomplete picture (Banich, 2009). A debate has emerged regarding the relationship of attention and executive function in the hierarchy of cognitive processes—are executive functions part of a larger attention skill (Posner & Petersen, 1990), or does attention serve higher order executive functioning (Barkley, 1997)?

On the one hand, Posner and his colleagues list alerting, orienting, and executive control as the three major components of attention as they are associated with neuroanatomical networks. Frontal and parietal regions of the right hemisphere tend to support the alerting network; superior and inferior areas of the parietal lobes support the orienting network (Posner & Petersen, 1990; Posner & Raichle, 1994). Executive control networks are more supported by frontal regions, including the lateral prefrontal cortex and anterior cingulate, mediating behaviors such as target and error detection, conflict resolution, inhibition of automatic reactions, and goal-directed actions<sup>1</sup> (Bush, Luu, & Posner, 2000).

However elegant this conceptualization, it does not carve the cognitive

system at clean joints ([Banich, 2009](#)); for every result laying territorial claim to a region for some particular cognitive function, another exposes the dependence of that function on a finely tuned coordination of several additional regions along cortical and subcortical networks for optimal performance. For instance, regulation of arousal and relayed communication between cortical and subcortical structures are supported by the thalamus, which plays key roles in several affective and cognitive functions, such as mood and language. Movement and emotional regulations are supported by the basal ganglia, particularly the caudate and putamen, which lead to impulsivity and loss of control, like a compromised frontal lobe, when damaged. Even the lowly cerebellum contributes beyond its crucial role in movement and coordination, as these timing and sequencing integrations extend to planning cognitive and affective behaviors that suffer with cerebellar damage ([O'Reilly, 2006](#)).

If neuroanatomical structures are not complicated enough, there are, of course, the neurochemical complexities. Consider just one, dopamine; it operates through three pathways serving attention. The first of these, the energy pathway, heightens energy along the basal ganglia, connecting the substantia nigra and motor cortex. Second, desire pathway runs through the orbital prefrontal cortex connecting the premotor cortex to the caudate lobe. Third, pleasure pathway operates via the basal ganglia to connect the frontal lobes with the olfactory bulb and ventral tegmental nucleus, where dopamine cell bodies originate, transmitting the sense of drive, energy, and pleasure necessary to motivate interest and attention. In addition to neurotransmitters, hormones impact alertness, as well as dopamine levels, particularly those hormones produced by the hypothalamic–pituitary–adrenal axis<sup>2</sup> ([Marie & Defer, 2003](#)).

On the other side of the debate, [Barkley \(1997](#); see also [Willcutt et al., 2005](#)) places attention within the larger frame of executive mastery of self-control or regulation because successful attending requires such mastery, so that deficits in attention can be reduced to deficits in executive function. But attempts to reduce any aspect of cognitive function to any neurological component is a bit like reducing a performance of Beethoven's *Ninth Symphony* to one string on one cello. Both approaches carry this strong implication of coordination, so the attention–executive function controversy may well be merely one of emphasis and semantics; one man's executive function hierarchy is another man's attention foundation. Ultimately, this debate forces us to recognize just how difficult it is to separate the two; they are so intertwined. Given the vast body of evidence that these skills are intricately tangled up in neuronal pathways and

relay stations dedicated to the business of coordinating, regulating, and controlling input and responding, we are inclined to place attention in service to executive functions.

Still, any distinction between them is shadowed by their mutual reliance and may have served to distract from what we feel is the overarching point of these functions of attending the environment and executing actions, even actions in service to further attending. The point is that these functions serve as our interface with the environment, our tools for encountering incoming stimuli, our frontline to engaging the world. In short, these particular functions serve as our “first responders” in dealing with life, and thus provide the foundation for developing all other cognitive functions (and to a great extent, their maintenance), including memory and language. This point is important because of its implications for rehabilitation, not just in these specific areas of attention and executive function deficits (EFDs), but across the spectrum of rehabilitation. Because neurological compromise so rarely leaves these functions intact, we feel all rehabilitation is therefore compelled to address them in treatment plans.

Complete coordination of the many neuroanatomical and neurochemical networks supporting attention and executive function is, of course, not fully developed at birth, these show vigorous growth during postnatal months, many not fully forming until the 20s (Ruff & Rothbart, 1996). These skills run the gamut from the obvious to the subtle, including determining the relevance of stimuli, prioritizing these for the task at hand, formulating a goal and plan of action replete with sequential steps toward success, as well as maintaining an open channel of self-regulation to take account of shifts in incoming data and goal specifics that demand changes in the game plan. This list of skills represents a rough model of attention and executive functions for the generic human, but each unique organism encounters in each moment a unique situation that imposes unique variables such as age, history, and social group on the model’s implementation. The implications of these environmental variables for survival make a broad postnatal growth potential understandably imperative. However, later and longer development—as well as the inevitable decline of aging—also leave the system more vulnerable to inefficient processing patterns such as are seen in individuals manifesting attention and executive function deficits (EDSs), deficits that rehabilitation is challenged to recover.

## **A VIEW OF THE DEFICITS**

### **Life Span Disorders**



## Disorders of Attention and Executive Function in Development

Disruptions in the development of these neurological systems may occur as the result of any number of conditions or events. First-order disruptions occur at the genetic level, and there is considerable evidence that attention deficit hyperactivity disorder (ADHD), for instance, is quite heritable, some twin studies registering as high as 75% to 91% (Levy, Hay, McStephen, Wood, & Waldman, 1997). However, these numbers may be confounded by the high comorbidity rate in ADHD patients (e.g., anxiety, depression, etc.; Freitag, Rohde, Lempp, & Romanos, 2010). Heritability estimates for EFDs are not as well-researched, though Friedman *et al.* (2008) found a highly heritable (99%) common factor, as well as additional genetic influences unique to particular aspects of executive functions (e.g., inhibiting dominant responses, updating working memory representations, shifting between task sets), prompting their claim that executive functions are among the most heritable psychological traits. Indeed, imaging studies tend to show numerous differences in the ADHD brain (Semrud-Clikeman *et al.*, 2000).

In addition to compromises of attention and executive function from genetic influences, these skills can be placed at significant risk for prenatal compromise from external sources, such as toxic agents and various conditions of pregnancy and delivery. Exposure to agents can either induce ADHD and associated EFD, or trigger an underlying predisposition that otherwise might not occur. Developmental weakness in these functions can occur from prenatal exposure to cigarettes (Ernst, Mochan, & Robinson, 2003), alcohol (D'Onofrio *et al.*, 2007), marijuana (Fried, 2002), cocaine (Noland *et al.*, 2003), medications such as lithium (Viguera *et al.*, 2001), and/or other substances. The literature on the sequelae of these insults to the developing brain in utero is too vast to review here, but the profound impact on attention and executive functions is generally undisputed.

Lead is another toxic agent with serious implications from prenatal and early developmental exposure for healthy development of attention and executive function. This common chemical, once prevalent in paint and water piping, can affect synaptogenesis (Goldstein, 1990) and interfere with many aspects of the normal course of brain development. Several metaanalyses (Lanphear, Dietrich, Auinger, & Cox, 2000) have established a strong dose–response relationship, and no lower “safe level” for lead exposure on the developing brain.

A vast literature also documents cognitive and behavioral sequelae of

prematurity and very low birth weight ([Aarnoudse-Moens, Weisglas-Kuperus, van Goudoever, & Oosterlaan, 2009](#)), tending to disclose strong correlations between very premature and/or very low weight in children with moderate to severe deficits in attention, executive functions, behavior, and academic achievement. During transition to young adulthood, these children continue to lag behind term-born peers.

The profoundly impeded development of executive function in autism is well documented ([Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002](#)), but recent studies suggest that children with Asperger's syndrome and related "autism spectrum" disorders also tend to exhibit deficits in both attention and executive functions ([Ozonoff & Griffith, 2000](#)). Nonverbal learning disability (NLD) is most often accompanied by symptoms of ADHD and/or EFD ([Rourke, 2000](#)), as are other learning disabilities; even the diagnosis of dyslexia frequently carries these symptoms ([Booth, Boyle, & Kelly, 2010](#)). Another childhood neurological disorder, Tourette's, has been linked to a dysfunction of dopamine receptors ([Rogeness, Javors, & Pliszka, 1992](#)) that predictably results in diminished aspects of executive function, most particularly, the ability to self-impose behavioral inhibitions. Though [Harris et al. \(1995\)](#) have documented some EFD in children with Tourette's, those recorded levels were relatively lower than those documented for children with ADHD.

In reviewing these examples of developmental disorders, it should be remembered that even normal children, adolescents, and older adults show decrements in executive function on occasion, sometimes frequently; with increased stress, particularly disruptions in sleep and nutrition, we all suffer in our ability to field novel and unexpected events. This point is consistent with our assertion here that attention and executive functions provide "first responder" tools in our environmental encounters. In fact, the neurological developmental process could be understood to a great extent in terms of the evolving mastery of the skills that tend to decline first with aging because of their relative fragility (the down side of flexibility).

## Disorders of Attention and Executive Function in Adults and Aging

Increasingly, adults who were never diagnosed with ADHD or EFD as children are seeking diagnostic assistance in addressing what they describe as lifelong struggles with those symptom patterns ([Biederman, Petty, Evans, Small, & Faraone, 2010](#); [Wilens et al., 2009](#)). Because all attention-executive systems suffer under stress, and because we all suffer stress from time to time, it is

reasonable to relate to ADHD symptoms. For diagnostic purposes, it is critical to clarify precisely when symptoms emerged<sup>3</sup> for distinguishing between adult ADHD and early dementia.

Whenever practitioners observe signs of regression to executive dysfunction in the elderly typically seen in early childhood—such as perseveration, impulsivity, and diminished self-care (Vaughan & Giovanello, 2010)—concerns regarding dementia are inevitably raised. In fact, aging affects executive function more than any other cognitive ability (Treitz, Heyder, & Daum, 2007). Alzheimer's dementia, with its severe, insidious, and relentless decimation of all cognitive functions, begins with subtle decline of executive skills prior to any registration of neurological evidence (Escandon, Al-Hammadi, & Galvin, 2010), eventually destroying that sense of time's extension into the future so key to the sense of self. Generally considered a disease of movement, the Parkinson's profile does not exhibit the devastating loss of self-awareness and personal memory observed in Alzheimer's, though symptoms progress to compromise executive function (Milham et al., 2002; Uekermann et al., 2004).

### Psychiatric Disorders

Unsurprisingly, executive function is compromised across a broad number of psychiatric illnesses, including anxiety, mood disorders, schizophrenia, substance abuse, and posttraumatic stress disorder (PTSD) (Hart, 2008; Theotka, 2006), as well as social dysfunction in children (Riggs, Laudan, Jahromi, Razza, & Dillworth-Bart, 2006). We routinely encounter referral sources in clinical practice (parents, teachers, and clinicians alike) asking if a child's poor academic performance might stem from his documented anxiety. This raises an important consideration, especially when these children are typically encouraged to take medications for the anxiety, as well as for attention deficits (a combination that can work at cross purposes). Though there is some documented support for the logic behind their question (e.g., compromised attention and executive functions often accompany self-reports of anxiety), the evidence of brain abnormalities in the learning disabled (LD) populations argues against it. Moreover, we have observed clinically that although anxiety medication does offer some relief, the pattern of cognitive weaknesses tends to remain. This topic begs for further research to clarify the relevant distinctions and relations of anxiety and cognitive processes underlying learning problems, particularly to avoid overmedicating.

An especially difficult childhood behavioral pattern to manage clinically is

oppositional-defiant disorder (ODD). These children, in addition to exhibiting challenging willfulness and belligerence, also show high comorbidity rates with ADHD and EFD (Ghanizadeh, 2010) and tend toward poor academic performance. The psychological complexities of the ODD diagnosis have yet to be adequately plumbed, and there may be reason to suspect that some of those behavior problems are secondary to the cognitive pattern of diminished self-control and inhibition.

Both children and adults can show the symptom pattern of obsessive-compulsive disorder (OCD), though symptoms emerging in youth tend to reflect higher familial incidence (Geller, 1998). Generally considered a dysfunction of prefrontal mechanisms for response inhibition (Savage & Rausch, 2000), other aspects of executive function are also often disrupted. Similarly, schizophrenia, a highly heritable disorder with identified genetic markers, is associated with extreme compromise of executive functions across most dimensions, especially social monitoring, reality testing, planning, and organization (Hyman, 2008). Recent studies suggest, however, that despite biological determination of executive dysfunction in this population, training can improve daily living skills (see the section “Interventions Toward Rehabilitation”).

Mood disorders and the various manifestations of anxiety and phobias have also been linked to declines in executive function and attention/concentration. Of course, the manic phase of bipolar disorder is often floridly psychotic when unmedicated, mimicking schizophrenia for compromise to frontal lobe function. Still, the depressive phase and unipolar depression both frequently exhibit declines in attention and executive function due to distractions and intrusive thoughts (Kolur, Reddy, John, Kandavel, & Jain, 2009), as does PTSD due to diminished arousal levels (Bremner, 2006; Hart et al., 2008). This pattern of compromise is also observed in individuals abusing alcohol and/or various substances, in which the substances themselves take a physical toll on the frontal cortex (Theotka, 2006), in addition to effects from underlying conditions such as depression or PTSD, for which these patients are self-medicating.

### **Acquired Medical Disorders**

Acquired brain insults, particularly when there is direct trauma to the prefrontal regions, rarely fail to disrupt attention and executive function (Fletcher et al., 1990; Thornton, 2000). Traumatic brain injury (TBI) represents perhaps the most studied neurological phenomenon due to its high incidence, especially in children (Centers for Disease Control and Prevention reports 1.4 million injuries

per year, 34% children). It is this population that places the greatest demand on rehabilitation programs. Much of our neurological knowledge base has grown as a result of the increased numbers of battle-induced head injury survivors from the past century, though peacetime emergency rooms log their incidence in large numbers, virtually all showing compromised attention and executive function (Thornton, 2000). These patients, soldiers and civilians alike, arouse acute interest for clinicians because, despite their survival rates, their loss of executive function that is so crucial for self-directed behaviors leaves them unable to live independent and productive lives. These patients are also at increased risk for early dementia (Jellinger, Paulus, Wrocklage, & Litvan, 2001).

Like TBI, the incidence and cognitive sequelae of strokes have contributed significantly to our knowledge of brain function, largely due to their more lateralized and discrete areas of damage. Even with distinct posterior events, however, most stroke victims do show some losses in executive function and attention for some time during recovery (Stricker, Tybur, Sadek, & Haaland, 2010). Susceptibility to stroke is just one part of the broader arena of cardiovascular disease, which can include loss of integrity in blood flow to the brain seen in cerebrovascular dementia (CVD), where decline in self-regulatory behaviors and sustained attention are considered classic symptoms (Menon & Kelley, 2009). In addition, individuals who have suffered heart attacks or the more prolonged decline of congestive heart failure also exhibit decline in executive functioning and attention (Almeida & Tamai, 2001).

As a rule, any loss of normal blood flow to the brain, for whatever reason, will predictably result in a generalized compromise of brain function. Similarly, a number of neurotoxic agents are capable of overwhelming the brain's finely tuned neurochemical balance, disrupting synaptic flow of neurotransmitters, leaving even the cerebellum damaged (Fan, Wang, Lo, Chang, & Chen, 2009). Symptoms tend not to differ significantly; most toxins cause headaches, confusion, and assaulted concentration skills. Poisoning from overdose of any drug, recreational or prescribed, will result in a similar pattern of symptoms, though most often toxic levels of these substances can lead to complete shutdown of overall functioning, even death. In contrast, exposure to toxic agents such as carbon monoxide (CO; Blumenthal, 2001) typically results in a stealthy pattern over a period of time, varying on concentration and nature of the toxin itself, as well as by length of exposure.

The devastating effects of lead poisoning on the adult, and especially developing brain, are well documented in both animals (Rice, 1993) and humans

(Cecil et al., 2008; Finklestein, Markowitz, & Rosen, 1998; see Disorders of Attention and Executive Function in Development). Finklestein's results exposed lead's capacity to cross the blood–brain barrier, leading to primary damage in astrocytes and secondary damage to the endothelial microvasculature; further damage from extensive poisoning was also observed in the prefrontal region, hippocampus, and cerebellum. Similarly, both radiation and chemotherapy treatments of cancer have been implicated in diminished concentration and executive function capacities, and have been found to disrupt connectivity and white matter development in the growing brain (Brouwers, Riccardi, Poplack, & Fedio, 1984).

Seizure disorders represent a number of types (e.g., complex partial to generalized) and etiologies (e.g., congenital, dysplasia, febrile, TBI), naturally inflicting humans at significantly higher rates at each end of the life span, as well as during hormonal shifts in preadolescence, pregnancy, and menopause. Neuropsychological symptoms vary according to seizure genesis, with the more common temporal lobe epilepsies typically compromising working memory skills (Meador, 2002). Anticonvulsant medications often not only significantly reduce seizure frequency, but also bring complaints of diminished concentration and focus, of being “in a fog.” The nature of these drugs makes the complaints predictable; seizure incidence is reduced by reducing neuroelectrical activity, leading to dampened cognitive function. Even with surgical intervention, medication is typically continued, if titrated, as a precaution. Children with epilepsy risk learning problems at a rate estimated at up to 50% (Aldenkamp, Alpherts, Dekker, & Overweg, 2007), and though determination of risk for executive function and attention deficits is complicated for multiple reasons, Gioia, Isquith, and Guy (2001) assert that such risk is likely commensurate with the estimates of learning problems.

Like seizure disorder, multiple sclerosis (MS) symptoms are most typically restricted to the area of affected cortical demyelination, though demyelination patterns vary and are unpredictable. Still, compromised attention and executive functioning are common, even before the disease spreads into the frontal cortex (Chiaravalloti & DeLuca, 2008). Many autoimmune diseases such as lupus will present with symptoms of diminished concentration and focus, possibly as a chronic pain response, though Muscal, Bloom, Hunter, and Myones (2010) found both cognitive deficits and brain abnormalities in children with lupus. Normal pressure hydrocephalus (NPH) presents with symptoms of confusion and diminished attention and executive function. Though surgical shunts will

successfully normalize brain pressure, many patients continue to suffer some deficits of higher cortical function (Schwarzchild et al., 1997). Of course, children with hydrocephalus at birth show a broad range of symptoms, but decreased inhibition skills are prevalent, especially in monitoring distractions, with subsequent problems sustaining attention (Baron et al., 1995).

A problem likely emerging in childhood, sleep disorders result in compromised attention and executive function for those both suffering from sleep apnea (Chervin et al., 2002) and individuals working night shifts for extended periods of time (Saricaolu et al., 2005). Both causes of sleep disruption can be equally destructive to healthy brain function, starting with drastically diminished capacities to attend, concentrate, and apply executive functions. Indeed, these losses due to disrupted sleep patterns have been linked to increases in accidents, especially on the job (Dula, Dula, Hamrick, & Wood, 2001), in addition to early dementia (Bliwise, 2004), with extreme sleep deprivation leading to breakdown of executive controls in psychosis (Coren, 1998).

Like sleep disorders, the problems of underserved populations such as malnutrition, understimulation, and isolation are often overlooked. However, we see evidence for diminished executive function and attention in all those who suffer these deprivations, especially the elderly (Van Dyk & Sano, 2007) and children (Bellisle, 2004). Indeed, there is increased risk of ADHD diagnosis in children of poverty (Brooks-Gunn & Duncan, 1997), placing them at great risk for academic losses (Biederman et al., 2004).

As with most psychiatric disorders, deficits in attention and executive functioning appear to accompany many general medical complaints, though how inherent or secondarily emergent these symptoms might be has not yet been determined. More research must address this question, but the literature thus far indicates that rehabilitation programs cannot neglect these functions, when assessing and treating the full range of psychiatric and medical conditions.

### **Assessment of Attention and Executive Function**

Whether acquired or congenital, medical or psychiatric, neurological disorders rarely spare the human capacities of higher order attention and executive functioning. For this reason, rehabilitation efforts across the spectrum should always address these skills in both assessment and treatment planning. Because recovery of executive functions and attention have such fundamental and profound impact on a patient's capacity to adjust and return to daily living,

articulating the profile of postmorbid skills—both lost and retained—is of utmost importance (Crawford, 1998). In the past, neuropsychologists have relied on a relatively random collection of tasks that challenge executive functioning, though many of these measures were never expressly designed for the purposes of testing those particulars. The toolkit then included, for instance, Wisconsin Card Sorting Test and Trailmaking B to expose difficulties with set-shifting, Mazes and Tower of London to challenge planning skills, and Matrices (Wechsler and Raven’s versions) and the Booklet Category Test (BCT) to confront problem solving capacities.

More recently, numbers of both innovative and revised measures have emerged to strengthen the neuropsychologist’s repertoire with tools boasting improvements in reliability and validity, as well as expanded normative base populations and refined administration ease. For example, all versions of the Wechsler scales (e.g., WPPSI-III/WISC-III/IV/WAIS-IV) now offer revised versions of tests of attention and executive function (e.g., Letter-Number Sequencing, Matrices). The Delis-Kaplan (D-KEF) provides tasks for assessing several aspects of executive function (e.g., verbal fluency, design fluency, abstraction, resistance to intentional interference, tracking and sequencing, set-shifting, etc.) for those aged 8 through adult. For children aged 5 to 18, the Tasks of Executive Function is a computerized “go/no-go” task that many children find enjoyable. For parent, teacher, and self-input, the Behavior Rating Inventory of Executive Function (BRIEF) checklists evaluate a number of component cognitive skills in those aged 2 through college age (e.g., initiation, shifting, inhibition, emotional control, working memory, planning, organization of materials, and monitoring). The computerized PASAT (Paced Serial Addition test; for adults), TOVA (Test of Variables of Attention; aged 4 and above), and the CPT-II (Conners’ Continuous Performance Test; aged 6 and above) all assess attention, as does the Tests of Executive Control (TEC). In addition, qualitative observation of performance approach across other neuropsychological measures is always invaluable.

This listing is hardly exhaustive and is intended to provide the reader with a brief overview for purposes of becoming familiar with the repertoire (McCloskey, Perkins, & VanDivner, 2009, offer a full list). However, new tests are being devised and introduced at a demanding pace, so familiarity with the literature on their application convenience and efficacy is always highly recommended.

## **INTERVENTIONS TOWARD REHABILITATION**



## INTERVENTIONS TOWARD REHABILITATION

### Pharmacological Interventions

The design of medications capitalizes on the distinctions between neurochemical effects on specific brain activities. By activating histamine receptors, for example, Modafinil bolsters wakefulness, as do amphetamines, which act on receptors in the striatum that project to dopamine regions. Amphetamines also promote the distinction between signal and noise crucial for prioritizing sensory inputs toward organizing behavioral outputs. Atomoxetine blocks norepinephrine (NE) reuptake in the frontal cortex, impacting executive functions, and memantine hydrochloride (HCl) strengthens memory components of the attention process.

Because deficits in attention and cognitive skill such as those observed in ADHD and EFD have traditionally been considered biological, pharmacological interventions offered the most logical treatment strategy and remain the standard choice for remedy, particularly for ADHD. This fact is not surprising in light of the 70% success rate for people with this diagnosis ([Schweitzer, Cummins, & Kant, 2001](#)), often leading to quick and dramatic improvements in behavior. In addition to the standard Ritalin, other stimulants prescribed for ADHD, and for some aspects of EFD, include Adderall, Concerta, Metadate CD, Focalin, Dexedrine, and Daytrana (in patch dosage). Side effects of diminished sleep and appetite are frequently reported with stimulants, however, reducing their overall effectiveness in treating growing children.

Due to its norepinephrine blocking in the frontal cortex, Atomoxetine (Strattera), a nonstimulant, is often prescribed for both ADHD and EFD. This medication is not without its caveats though, as it has been linked to liver damage, orthostatic hypotension, syncope, and increased risk of suicide. In addition, the FDA has warned of possible heart-related and mental health problems associated with Strattera. Some antihypertensives designed to treat high blood pressure (Catapres and Tenex) can also help control aggressive and impulsive behaviors, not well-managed by other ADHD medication. The antidepressant bupropion (Wellbutrin) or a tricyclic antidepressant may also be prescribed, especially if there are signs of comorbid depression.

More recently, medications designed for treatment of Alzheimer's dementia, specifically Namenda (memantine HCl) and Aricept (donepezil HCl) have been prescribed to patients complaining of diminished attention and executive functions. Significant increases in certain cognitive functions,

particularly for attention and working memory skills, have been documented in pain patients (Bekken & Pachas, 2007), in addition to subjective reporting of lifted “mental fog” and confusion. This listing offers only the briefest of overviews of medications available for treatment of deficits of attention and executive functions, as this topic is covered in far greater depth and breadth in [Chapter 18](#) of this volume.

### Cognitive and Behavioral Techniques

Several early treatment and rehabilitation treatments applied behavior modification principles (e.g., contingency management, token economies, behavior activation) to the various emotional and behavioral symptoms of cortical compromise, such as impulsivity, anger and aggression, and substance abuse, as well as anxiety and depression. These techniques have enjoyed some consistent success within the neurological populations suffering those accompanying symptoms, with additional improvements in attention and executive dysfunctions generally associated with these behaviors. However, few studies have addressed the effects of behavioral or cognitive behavioral therapies (CBT), specifically on cognitive functions. In fact, due most likely in large part to the steady success rates with medications, and then technological treatments, the behavioral therapies and CBT have remained limited in application to those with psychiatric symptoms, for which the combination of CBT and medications holds the standard for premier effectiveness (Butler, Chapman, Forman, & Beck, 2006). Therefore CBT and the various behavior modification programs should be considered as possible rehabilitation therapies, whenever appropriate.

Although behavioral and CBT are often successfully applied to concerns of behavioral self-controls, concerns of attention and executive function are addressed more specifically through cognitive therapies and training. For example, *Audioblox* is a multisensory cognitive enhancement program designed to develop and automatize basic learning skills as applied to a range of learning difficulties, including dyslexia and dysgraphia. A promising tool designed for brain injury patients is *Tele-cognitive Rehabilitation*, which offers face-to-face communication between therapist and brain-injured client (Tam et al., 2003). Using videoconferencing technology for monitoring and customization therapy, this technique led to significant improvement of cognitive performance for three subjects over 18 sessions.

Standard cognitive rehabilitation programs have seen successes within numerous populations (Flavia, Stampatori, Zanotti, Parrinello, & Capra, 2010),

especially the brain-injury population. A review of 4 years of attention training studies on TBI patients by [Kennedy et al. \(2008\)](#) showed that meta-cognitive strategy instructions (MSI) proved effective with adults to remediate functional daily activities, with some indication of maintained progress. A similar metaanalysis by Rohling, Faust, Beverly, and Demakis (2009) found significant benefits from cognitive remediation for attention training after TBI.

### **Computer-Assisted and Technological Techniques**

Currently, the state-of-the-art attention and executive function rehabilitation is capitalizing on the many advantages of computerized technologies in systematizing task method, delivery, and monitoring for near-immediate analysis of efficacy. Generally inspired by the notion that “practice makes perfect,” that is, efficiency will increase after repetitive practice of specific skills related to attention ([Posner & Raichle, 1994](#)), cognitive training, in general, and attention training, in particular, attempt to increase cognitive efficiency through repetitive practice of related skills. Due to the convenience they offer to therapists and patients alike, as well as the fascination computers hold for children, these programs are exploding on the market, reflecting the many competing models and theories of attention and executive function noted in the introduction to this chapter.

Consistent with the supporting perspective that adaptations will occur in the underlying neuroanatomical networks linked to these processes as a result of targeted practice ([Kerns, Eso, & Thompson, 1999](#)), evoked potential measures (ERP) and fMRI evidence suggest that attention training can affect brain function ([Mateer & Mapou, 1996](#)), with increased prefrontal and parietal brain activity ([Olesen, Westerberg, & Klingberg, 2004](#)) and changes in dopamine D1 receptor binding ([McNab et al., 2009](#)). However, the neurological basis for the shifting effect from practicing attention is not yet fully understood.

Neuropsychonline Cognitive Rehabilitation Therapy (NCRT) addresses attention and executive functioning, as well as memory and problem solving in brain injury, stroke, and other acquired neurological injuries, as well as attention deficit and learning disabilities. An outpatient, home-based, computer-assisted rehabilitation program, NCRT combines face-to-face contact for evaluation, review of exercises, and assignment of new exercises. The home-based requirement holds at least two to three 1-hour periods daily, the whole program taking 9 to 12 months to complete. Using this system, investigators have reported memory improvements at 6-to 12-month follow-up testings.

Cogmed is a computerized training program developed through Sweden's Karolinska Institute that exploits the brain's neuroplasticity to support improvements in performance through consistent repetition of ability-matched auditory and visual working memory exercises. Based on the notion that working memory is the root of effective brain functioning, it is rapidly gaining expanded application worldwide. The individual user (aged 4 to adult) engages in 5 weeks of 25 intensive sessions, with extensive practice of working memory exercises, with weekly check-ins with a coach. Follow-up support is available for the first year following purchase.

To date, several Cogmed centers have conducted numbers of randomized, doubleblind studies on its effects. In addition to changes in brain activity following training (McNab et al., 2009; Olesen, Westerberg, & Klingberg, 2004), researchers have found increased cognitive performance for multilevel instructions, reading comprehension, new learning, recall of numbers, activities of daily living, filtering out of distractions and impulse control, and time management (Klingberg et al., 2002, 2005). Randomized controlled trials reported both improvements in functioning, as well as transfer of improvements on sustained attention and executive function (Penkman, 2004; Sohlberg & Mateer, 1987). The Cogmed program has also been used to treat acquired deficits in attention and executive functioning (Westerberg et al., 2007), and the *Cogmed* website itself cites studies showing significant improvements for children, adolescents, and adults following application of its training program, as well as numerous elements of transfer and sustainability, at least over the short term.

PSSCogRehab is a multimedia cognitive rehabilitation software system designed for brain-injury rehabilitation and cognitive enhancement training for schizophrenics (Bracy, 1983). Patients work daily on a home-based training program, plus a weekly face-to-face session with the therapist, to learn compensatory skills and strategies for information processing (analysis, organization, and "response implementation"). On average, patients require about 12 months to complete the program, which involves eight levels of training, including foundations, visuospatial, memory, and problem solving. Most relevant to attention and executive functions are the foundation-level exercises that train visual and auditory attention skills (focusing, tracking, targeting, shifting, sustaining, and dividing attention) while integrating them with some very basic executive functions (discrimination, initiation, inhibition, and differential responding), followed by a second level of greater attentional

demands and executive function challenges. The program also includes rehearsal and compensation strategies in various activities of daily living, including in vivo trips to the grocery store and route finding. *PSSCogRehab* has been used extensively and is most effective when used as part of a well-formulated and comprehensive treatment program. Like most such computerized attention training programs, access to background and implementation information can be found on their website.

The *Brain Train* system is another computerized attention training program that has been applied to varied patient populations, aged 5 to adult. The program features a neurofeedback system, SmartMind 2, and a selection of associated software, as well as their *Captain's Log* cognitive training exercises with associated listening skills and visual attention systems, SoundSmart and SmartDriver. *Brain Train* boasts a large supporting research base, including comparisons with other treatment approaches ([Rabiner, Murray, Skinner, & Malone, 2010](#)).

Many other programs address executive functioning, including strategies to support planning, organization, memory, study skills, time management, listening comprehension, and note taking. Programs such as *EmPOWER* ([Singer & Bashir, 1999](#)) help students improve executive and organizational aspects of the writing process by determining assignment demands, setting goals, choosing strategies to plan and organize, developing text based on those plans, and self-monitoring their writing. *Brain Frames* strategies are used for remediating executive functions (e.g., visual-spatial displays of the underlying task patterns basic to language), this broad program can be incorporated into the classroom easily and effectively.

Other computerized individual cognitive training programs include *Earobics*, a reading intervention program for preschool to grade 3, training fundamental skills such as attention and sequencing. The *Dr. Brain* series is one of a number of software games available commercially for training important brain functions in a game and/or puzzle format. Based on Gardner's theory of multiple intelligences ([Gardner, 1983](#)), this approach trains/retrains foundational brain skills, such as figure/ground, selective and sustained attention, logic, and sequencing. It is used as part of the cognitive rehabilitation battery at several hospitals in their pediatric brain-injury units.

Virtual reality programs are being developed and applied at an astonishing rate for a vast number of disabilities, including phobias, autism, retardation, dyslexia, brain injury, paraplegia, MS, dementia, and stroke ([Reiss & Weghorst,](#)

1995; Sik Lányi et al., 2005). Not limited to the teaching of more primary, linear skills typically associated with cognitive training, virtual realities expand these horizons to assist even the home-bound and the blind, and can enhance social interactive competencies, such as virtual communities with virtual economies. Because of their immersive and interfacing capacities, they offer tremendous potential for function recovery and especially improved quality of life, though further work is needed to fully integrate the array of technologies toward optimally effective systems. At this level of complexity, virtual realities may be best equipped to capture the complexities of attention and executive function, with some studies suggesting promise in this specific area. For example, Kang et al. (2008) developed an immersive *Visual Reality* (VR) program they purport to be of benefit in cognitive retraining, whereas Lo Priore, Castelnovo, and Liccione (2002) used a VR task (V-Store) expressly for cognitive rehabilitation of executive functions in everyday tasks for individuals with frontal lobe damage. Improvements in executive functioning were documented with the use of this software, although there is not as yet consistent evidence for generalizing learned skills to novel domains.

Most studies of computerized training of attention and executive function do show improvements in those skills. Chen, Thomas, Glueckauf, and Bracy (1997) reported that a computer-assisted cognitive retraining program was more effective for remediating TBI, than a traditional rehabilitation program (i.e., receiving only physical, occupational, and/or speech therapies). Similarly, Elgamal, McKinnon, Ramakrishnan, Joffe, and MacQueen (2007) found that depressed patients using *PSSCogRehab* made significant gains in attention and executive functioning while showing no change in depressive symptoms, though it could be that improvements in depressive symptoms may require more time to manifest. Substance-abuse patients showed similar improvements following computerized attention training (Fals-Stewart & Lucente, 1994). Finlayson, Alfano, and Sullivan (1987) found that computer-assisted tasks of attention and executive functions led to significant improvement in new learning and problem-solving skills, mental flexibility, and psychomotor functioning.

In addition to the application of these described computerized technologies to patient populations, such tools have been applied to children felt to be “at risk” for ADHD and/or EFD diagnoses. Given the extensive development of attentional and executive systems during the preschool years that are correlated with developments in brain structure and function, Tamm et al. (2007) hypothesized that training “at risk” children could eliminate or reduce the impact

of attention disorders. The authors reported positive effects, although conclusions were limited due to small sample size and insufficient testing of transferability or sustainability. Similarly, [Bracy et al. \(1999\)](#) used computer-assisted cognitive training for the development of basic cognitive skills (defined as attentional, executive, visuospatial, and problem solving) in undiagnosed and asymptomatic 12-to14-year-old school children. After the two groups underwent a 9-week program of either computer-assisted cognitive skills training or study hall, a significant increase in intellectual functioning was found only in the experimental group. Other studies have found that computerized cognitive training, when combined with medication for children with ADHD, is more effective than just medication alone ([Hall & Kataria, 1992](#)).

Though not computerized, EEG biofeedback is a technological technique used to treat a number of symptom profiles, including ADHD and executive function disorders, by training those areas of the brain involved in arousal and focus through self-monitoring. In the developing brain of the diagnosed ADHD child, EEG studies show excess  $\theta$  activity, with decreased  $\beta$  activity ([Lubar, 1991](#)), a low-arousal state more typically associated with sleep or daydreaming than with alert focus. The goal of EEG biofeedback is to help these children gain control over their own brain waves, and thus over their arousal level. Some EEG studies show 80% to 90% improvement in cognitive performance in children and adults with ADHD ([Robbins, 1997](#)), whereas others report reductions in ADHD medication dosage required ([Alhambra, Fowler, & Alhambra, 1995](#)).

To compare the effects of computerized cognitive training (specifically *Captain's Log*) to EEG biofeedback, Fine and Goldman (1994) applied one or the other of these techniques to 8-to 11-year-olds with ADHD. The *Captain's Log* group significantly improved focus and sustained attention, encoding and retrieval, and processing speed, whereas biofeedback reduced impulsive, hyperactive, and off-task behaviors. Tinius and [Tinius \(2001\)](#) have since tested the effects of combining these treatments in mild TBI and ADHD subjects, finding significant improvements on attention and response accuracy scales of a continuous performance task for both groups, though problem-solving errors declined only for the mild TBI group; symptom decline for both groups was also reported.

Of course, this is not an exhaustive list of available technological and computer-generated interventions for attention and EFDs, or of the research literature testing their effectiveness. Despite the successes ([Marks, Parent, & Anderson, 2009](#)), however, those gains from learned skills are most often

isolated to the medium, consistent with the well-established phenomenon of state-dependent learning. The question therefore remains as to whether such gains can translate to generalized increases in self-regulatory control in other contexts in which the patients were not trained, and the degree to which observed gains are sustained over time. In addition, such programs are subject both to plateau effects and idiosyncratic motivation/ persistence factors. Improved computer-specific learning skills hold limited benefit for any client if transfer is not occurring in other contexts, begging additional research in this particular area.

### **Lifestyle Treatment Programs (Sleep/Nutrition/Exercise/Arts)**

Consistent with findings that attention and executive functions are virtually always affected with deprivations of sleep, nutrition, exercise, sensory stimulation, and creative outlets (see A View of the Deficits), treatments that address these fundamentals of living have shown some success in various populations. However, considerably more extensive research is needed to flesh out the specifics within each area and the breadth of potential benefits.

Who among us has not suffered a stretch of sleepless nights due to overwork and/or stress and noted fully and instantly the recovery benefits of even one good night's sleep? Likewise, prescribed sleep routines show marked recovery of cognitive functions in individuals working night shifts ([Saricaoğlu et al., 2005](#)), as they do for teenagers with or without diagnosis of ADHD ([Roberts, Roberts, & Duong, 2009](#)). The various techniques prescribed for those suffering sleep disorders, particularly sleep apnea, tend to offer marked though varied relief of both fatigue and diminished attention and executive function ([Huang et al., 2007](#)). For anyone suffering neurological compromise of any kind, from seizure disorder to stroke to TBI and ADHD, the power of regular sleep habits on optimal brain function, particularly attention and executive skills, cannot be stressed enough, and should be included in any rehabilitation program.

Given fairly unequivocal evidence showing the deleterious effects of malnutrition and poor diet on brain development and function (reported here in A View of the Deficits), it is surprising that this component is so overlooked in most rehabilitation programs, and virtually ignored in addressing less formal assistance plans, such as those for ADHD and LDs. These latter populations, for example, actually show significant improvement of attention and executive functions, and overall reduction of ADHD symptoms, following introduction of essential fatty acids, amino acids, and high-grade proteins in the diet



(Richardson & Puri, 2002). Similar cognitive improvements ensue from similar dietary changes in the elderly (Nurk et al., 2007). We are remiss as clinicians if we fail to include inquiries regarding diet in our intake interviews<sup>4</sup> and ignore opportunities to include nutritional consults in our recommendations.

This advice also holds for the benefits from exercise on cognitive function<sup>5</sup> (Sibley & Etnier, 2003; Tomporowski, Davis, Miller, & Naglieri, 2008), though experimental evidence supporting specific types of exercise is scant and inconclusive (Jensen & Kenny, 2004). Yoga is now frequently recommended for a broad range of complaints, including children with ADHD and their parents (Harrison, Manocha, & Rubia, 2004), with anecdotal reports of improved sleep, school performance, and increased feelings of well-being. Similar applications of the various martial arts also offer positive results, whereas anecdotal evidence for cognitive improvements from dance and other sports also holds promise. Though the mechanism of effectiveness of exercise is not known, theories regarding reduction of cortisol levels, for instance, are under consideration.

Comparable effects on improved attention and executive function have also been reported with various therapies from the arts, including drama (Weber & Haen, 2004), visual arts (Safran, 2002), and music (Wall & Duffy, 2010). In fact, music therapy has been applied in a number of neurocognitive populations, including the elderly and schizophrenics. Thaut *et al.* (2009), for example, found that TBI patients showed improvement in executive function and overall emotional adjustment, with reduced depression, sensation seeking, and anxiety, as a result of Neurologic Music Therapy (NMT), as compared to control TBI patients.

What these various expressive methods have in common is a focusing of the mind on the present moment; however, it may unfold in the particular medium or task, a process that generally describes mindfulness meditation. Various types of meditation have been applied with some success to remediate attention and executive dysfunctions, showing capacity for increasing sustained attention, attention switching, and inhibition of elaborative processing (Bishop, Lau, Shapiro, & Carlson, 2004). Shapiro, Walsh, and Willoughby (2003) tie this enhanced capacity to increased self-regulation. The mechanism, therefore, might be an increased awareness of cognitive and physical states through a process of introspection and self-observation. Though physiological correlates, including increased interhemispheric phase coherence in frontal  $\alpha$ -EEG (Cahn & Polich, 2006), are documented regularly, the mechanism remains elusive.

## Individualized Training Programs

One important goal of any rehabilitation endeavor is to ensure transfer of the skill being taught, such that the individual knows how, when, and why to apply the new skill. In addition, the new skill, and the ability to generalize it, must be maintained over time. Transfer requires systematically teaching the new skill, and then decreasing the supports needed for the individual as he or she gains competence with the technique, while simultaneously increasing the nature of the skill application to meaningful contexts (e.g., teaching memory tools and then applying them to remembering information from a lecture for a high school student).

Ylvisaker (1998) made numerous contributions to the field of rehabilitation, but perhaps his most valuable was his focus on the importance of skill transfer, which he felt required three key elements. First, the training tasks must be similar, and organized similarly to transfer tasks used in instruction and application. Next, there must be opportunities to practice the transfer; no tool is worth having if its use is not well practiced. Finally, practice must be frequent enough to automatize the skill. When teaching an organizational system for writing, for instance, that system must translate instantly to classroom assignments.

Remediation patients might balk at first because learning an old task in a new way might seem like “too much work,” or not worth the extra effort required to unlearn bad habits and relearn old skills. It is therefore important for these patients to see the value in both working on the skills and learning how to transfer them, so that they are active participants in the process. One way to ensure that the skill is solidly in the individual’s “arsenal” is to have the individual attempt to teach the new skill to another person. This technique allows both the instructor and the student to observe how fully the new skill is understood and how easily transferred, so that any gaps can be addressed (Ylvisaker & Feeney, 1998).

In our clinical work, we have found that an Individualized Treatment Plan (ITP) specifically designed in response to an individual’s attention and executive functioning needs, as described in an extensive neuropsychological evaluation, has proven most beneficial in transfer of gains to general contexts. Moreover, when skills transfer to novel contexts practice frequency increases, which further galvanizes the skill. Patients might present with teacher reports of difficulties completing tasks independently, transferring information from one context to another, deriving the “big picture” for tasks, and/or retrieving information from

memory; all academic areas might fall below expectation in the absence of an underlying documented learning disability. For a patient with notable weakness determining relevant from irrelevant detail (demonstrated, for instance, by a lower score on the WISC-IV Picture Completion task), we may use a number of figure-ground tasks, hidden pictures, and word searches tailored to fit the patient's age and competence in reading and sustaining attention. For higher level deficits in planning, we may use mazes, tasks like Tower of London, tic-tac-toe games like Connect Four, or chess (using pieces on which allowed moves are pictured). Prediction techniques and social stories can help the patient see the "big picture," adjust plans along the way, and then generalize these skills. Similar strategies can be employed to help break down math word problems into their component parts. For those with difficulties in synthesis/analysis, we may present logic and strategy games of increasing complexity. We have used games readily available on the market, as well as tasks we generate to address individual weaknesses in attention and processing efficiency. We also individualize memory strategies to help logically organize and break down information for efficient encoding and retrieval (e.g., constructing a mental outline of main idea, supporting ideas, etc.). For those patients with stronger verbal skills, we offer verbalization and "talking through" strategies to structure problem-solving approaches, whereas children with stronger visual skills may "map out" their strategies. Computerized attention and working memory programs are also used, such as *Webspiration*, which focuses on visualizing the main idea and how details relate to it to organize written expression.

Most important, we emphasize transfer of skills to tasks that are relevant to the patients' lives. Thus, for a middle or high school student, transfer may apply learned skills to reading comprehension (e.g., determining main idea, supporting details), essay writing, note taking, creating study guides, breaking down and planning math word problems, and related tasks, depending on their individual needs. We find that approaches without this transfer piece lead to short-term gains that rarely transfer to other contexts, leaving patients with a disjointed toolbox of strategies but no clear idea of how, when, or why to apply them, and thus no opportunities to practice them. Progress is assessed with standardized measures, as well as subjective measures of status and progress via parent, teacher, and self-reports. We may also recommend adjunct therapies, such as speech therapy (e.g., with a *Visualization and Verbalization* component), occupational therapy, nutrition and exercise programs, and/or other therapies, as needed and appropriate.

Although ITP may render impossible scientific discernment of any operative mechanism of improvement, it nevertheless affords the individual patient all options available that offer positive potential. Because our clinical experience with varieties of patient profiles reflects findings in the literature of exceedingly complex neurological and neuropsychological mechanisms in play for optimal attention and executive functioning, we have chosen to respect that complexity by drawing upon the full array of available treatment techniques. Inherent in this approach, however, is a distinct sensitivity to each individual patient's needs and capacities for taking on levels of complexity. Although some students surprise us with their eagerness to try novel ideas, others resist anything new or unfamiliar, and many adults balk at changing their routines and habits. We emphasize, therefore, the importance of tailoring the ITP to the individual's needs, as they present to the practitioner. After all, these individuals, with their weakened attention and executive functioning, exhibit distinct difficulties with seeing and adjusting to novel solutions for unique problems; it seems a great disservice, then, to offer them the usual cookie-cutter tools, while we teach and preach the practice of complex higher order cognitive skills.

## SUMMARY

By acknowledging the inextricable interdependency of attention and executive functioning, as well as the “frontline” importance of these tandem skills, we assert that virtually all other cognitive functions thus rely on them. However, this assertion should not be taken as a reductionist oversimplification. Quite the contrary; this chapter considers both the profound complexities of the neurological networks supporting these functions, and the vast array of disorders, diseases, and events that can and do compromise attention and executive functions. We therefore further assert that every rehabilitation program should address attention and executive functioning.

As with all things neurological, the field of rehabilitating attention and executive function is, of course, a work in progress, calling for further research in a number of areas, particularly skill transfer beyond computer training programs, and the efficacy of addressing various lifestyle habits, such as sleep, exercise, nutrition, and creative expression. Given the complex nature of these higher order skills, and the relative infancy of our knowledge base about them, we encourage tailored treatment approaches that consider individual strengths and weaknesses, as well as personal preferences and habit patterns. Real rehabilitation successes are, after all, ultimately judged on an individual, case-

by-case basis, and must, in the end, be determined by that individual, with the expert and caring assistance of his or her practitioner.

## NOTES

1. These higher order functions geared for voluntarily planned actions toward future goals make possible the notion of a “self” that regulates, monitors, sustains, adjusts, and benefits from orchestrations.
2. In contrast to the immediate and rapid effects of neurotransmitters, hormones provide a more extensive temporal background for the organism’s biological functions.
3. Symptoms must emerge prior to age 7 to meet diagnostic criterion.
4. For instance, since incorporating these questions in our intake interviews, we have found anecdotally that virtually all youth with hyperactivity symptoms show marked indication of addiction to empty (i.e., nonnutritional) carbohydrates.
5. Recall how Parkinson’s patients suffer compromise of executive functions with loss of motor control and coordination, reflecting the subtle links between fundamental subcortical functions like movement and the higher order executive functions such as planning and rapid organization of information for implementation in responding.

## REFERENCES

- Aarnoudse-Moens, C. S., Weisglas-Kuperus, N., van Goudoever, J. B., & Oosterlaan, J. (2009). Metaanalysis of neurobehavioral outcomes in very preterm and/or very low birth weight children. *Pediatrics*, *124*(2), 717–728.
- Aldenkamp, A. P., Alpherts, W. C. J., Dekker, M. J. A., & Overweg, J. (2007). Neuropsychological aspects learning disabilities in epilepsy. *Epilepsia*, *31*, 9–20.
- Alhambra, M. A., Fowler, T. P., & Alhambra, A. A. (1995). EEG biofeedback: A new treatment option for ADD/ADHD. *Journal of Neurotherapy*, *1*, 39–43.
- Almeida, O. P., & Tamai, S. (2001). Congestive heart failure and cognitive functioning amongst older adults. *Arquivos de Neuro-psiquiatria*, *59*(2-B), 324–329.
- Banich, M. T. (2009). Executive function: The search for an integrated account. *Current Directions in Psychological Science*, *18*, 89–94.
- Barkley, R. (1997). *ADHD and the nature of self-control*. New York, NY: Guilford Press.
- Baron, I. S., Fennell, E., & Voeller, K. (1995). *Pediatric neuropsychology in the medical setting*. New York: Oxford University Press.
- Bekken, K., & Pachas, W. N. (2007). *The role of memantine (Namenda) in the treatment of mental dysfunction in patients with fibromyalgia (FM)*. Presented at Annual Conference of the International Neuropsychological Society, Bilbao, Spain.
- Bellisle, F. (2004). Effects of diet on behaviour and cognition in children. *British Journal of Nutrition*, *92* (Suppl. 2), S227–S232.

- Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., . . . Faraone, S. V. (2004). Impact of executive function deficits and attention-deficit/hyperactivity disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology, 72*(5), 757–766.
- Biederman, J., Petty, C. R., Evans, M., Small, J., & Faraone, S. V. (2010). How persistent is ADHD? A controlled 10-year follow-up study of boys with ADHD. *Psychiatry Research, 177*(3), 299–304.
- Bishop, S. R., Lau, M., Shapiro, S., & Carlson, L. (2004). Mindfulness: A proposed operational definition. *Clinical Psychological Science Practice, 11*, 230–241.
- Bliwise, D. L. (2004). Sleep disorders in Alzheimer's disease and other dementias. *Clinical Cornerstone, 6* (Suppl. 1A), S16–S28.
- Blumenthal, I. (2001). Carbon monoxide poisoning. *Journal of the Royal Society of Medicine, 94*(6), 270–272.
- Booth, J. N., Boyle, J. M., & Kelly, S. W. (2010). Do tasks make a difference? Accounting for heterogeneity of performance of children with reading difficulties on tasks of executive function: Findings from a metaanalysis. *British Journal of Developmental Psychology, 28*(Pt 1), 133–176.
- Bracy, O. L. (1983). Computer based cognitive rehabilitation. *Cognitive Rehabilitation, 1*, 7–8.
- Bracy, O. L., Oakes, A. L., Cooper, R. S., Watkins, D., Watkins, M., Brown, D. E., & Jewell, C. (1999). The effects of cognitive rehabilitation therapy techniques for enhancing the cognitive/intellectual functioning of seventh and eighth grade children. *International Journal of Cognitive Technology, 4*, 19–27.
- Bremner, J. D. (2006). Traumatic stress: effects on the brain. *Dialogues in Clinical Neuroscience, 8*(4), 445–461.
- Brooks-Gunn, J., & Duncan, G. J. (1997). The effects of poverty on children. *Future of Children/Center for the Future of Children, the David and Lucile Packard Foundation, 7*(2), 55–71.
- Brouwers, P., Riccardi, R., Poplack, D., & Fedio, P. (1984). Attentional deficits in long-term survivors of childhood acute lymphoblastic leukemia (ALL). *Journal of Clinical Neuropsychology, 6*(3), 325–336.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215–222.
- Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of metaanalyses. *Clinical Psychology Review, 26*(1), 17–31.
- Cahn, B. R., & Polich, J. (2006). Meditation states and traits: EEG, ERP, and neuroimaging studies. *Psychological Bulletin, 132*(2), 180–211.
- Cecil, K. M., Brubaker, C. J., Adler, C. M., Dietrich, K. N., Altaye, M., Egelhoff, J. C., . . . Lanphear, B. P. (2008). Decreased brain volume in adults with childhood lead exposure. *Public Library of Science, Medicine, 5*, 5.
- Chen, S. H., Thomas, J. D., Glueckauf, R. L., & Bracy, O. L. (1997). The effectiveness of computer-assisted cognitive rehabilitation for persons with traumatic brain injury. *Brain Injury, 11*(3), 197–209.
- Chervin, R. D., Archbold, K. H., Dillon, J. E., Panahi, P., Pituch, K. J., Dahl, R. E., & Guilleminault, C. (2002). Inattention, hyperactivity, and symptoms of sleep-disordered breathing. *Pediatrics, 109*(3), 449–456.
- Chiaravalloti, N. D., & DeLuca, J. (2008). Cognitive impairment in multiple sclerosis. *Lancet Neurology, 7*(12), 1139–1151.
- Coren, S. (1998). Sleep deprivation psychosis and mental efficiency. *Psychiatric Times, 15*, 11.
- Crawford, J. R. (1998). Introduction to the assessment of attention and executive functioning. *Neuropsychological Rehabilitation, 8*, 209–211.
- D'Onofrio, B. M., Van Hulle, C. A., Waldman, I. D., Rodgers, J. L., Rathouz, P. J., & Lahey, B. B. (2007). Causal inferences regarding prenatal alcohol exposure and childhood externalizing problems. *Archives of General Psychiatry, 64*(11), 1296–1304.
- Dula, D. J., Dula, N. L., Hamrick, C., & Wood, G. C. (2001). The effect of working serial night shifts on the cognitive functioning of emergency physicians. *Annals of Emergency Medicine, 38*(2), 152–155.
- Elgamal, S., McKinnon, M. C., Ramakrishnan, K., Joffe, R. T., & MacQueen, G. (2007). Successful

- computer-assisted cognitive remediation therapy in patients with unipolar depression: A proof of principle study. *Psychological Medicine*, 37(9), 1229–1238.
- Ernst, M., Mochan, E., & Robinson, M. (2003). Behavioral and neural consequences of prenatal exposure to nicotine. *Journal of the American Academy of Child & Adolescent Psychiatry*, 40, 630–641.
- Escandon, A., Al-Hammadi, N., & Galvin, J. E. (2010). Effect of cognitive fluctuation on neuropsychological performance in aging and dementia. *Neurology*, 74(3), 210–217.
- Fals-Stewart, W., & Lucente, S. (1994). The effect of cognitive rehabilitation on the neuropsychological status of patients in drug abuse treatment who display neurocognitive impairment. *Rehabilitation Psychology*, 39, 75–94.
- Fan, H. C., Wang, A. C., Lo, C. P., Chang, K. P., & Chen, S. J. (2009). Damage of cerebellar white matter due to carbon monoxide poisoning: a case report. *American Journal of Emergency Medicine*, 27(6), 757.e5–757.e7.
- Fine, A. H., & Goldman, L. (1994). *Innovative techniques in the treatment of ADHD: An analysis of the impact of EEG biofeedback training and a cognitive computer generated training*. Paper presented at 102nd Annual Convention of the American Psychological Association, Los Angeles, CA.
- Finklestein, Y., Markowitz, M. E., & Rosen, J. F. (1998). Low-level lead-induced neurotoxicity in children: An update on central nervous system effects. *Brain Research Review*, 27, 168–176.
- Finlayson, M. A., Alfano, D. P., & Sullivan, J. F. (1987). A neuropsychological approach to cognitive remediation: Microcomputer applications. *Canadian Psychology*, 28, 180–190.
- Flavia, M., Stampatori, C., Zanotti, D., Parrinello, G., & Capra, R. (2010). Efficacy and specificity of intensive cognitive rehabilitation of attention and executive functions in multiple sclerosis. *Journal of Neurological Science*, 288, 101–105.
- Fletcher, J. M., Ewing-Cobbs, L., Miner, M. E., Levin, H. S., & Eisenberg, H. M. (1990). Behavioral changes after closed head injury in children. *Journal of Consulting and Clinical Psychology*, 58(1), 93–98.
- Freitag, C. M., Rohde, L. A., Lempp, T., & Romanos, M. (2010). Phenotypic and measurement influences on heritability estimates in childhood ADHD. *European Child & Adolescent Psychiatry*, 19(3), 311–323.
- Fried, P. A. (2002). Adolescents prenatally exposed to marijuana: examination of facets of complex behaviors and comparisons with the influence of in utero cigarettes. *Journal of Clinical Pharmacology*, 49, 97–102.
- Friedman, N. P., Miyake, A., Young, S. E., Defries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual differences in executive functions are almost entirely genetic in origin. *Journal of Experimental Psychology General*, 137(2), 201–225.
- Gardner, H. (1983). *Frames of mind: A theory of multiple intelligences*. New York, NY: Basic Books.
- Geller, D. A. (1998). Juvenile onset obsessive-compulsive disorder. In M.A. Jenike, L. Baer, & W. E. Minichiello (Eds.), *Obsessive-compulsive disorders: Practical management* (pp. 276–288). St. Louis, MO: Mosby.
- Ghanizadeh, A., & Haghghi, H. B. (2010). How do ADHD children perceive their cognitive, affective, and behavioral aspects of anger expression in school setting? *Child and Adolescent Psychiatry and Mental Health*, 4, 4.
- Gilotty, L., Kenworthy, L., Sirian, L., Black, D. O., & Wagner, A. E. (2002). Adaptive skills and executive function in autism spectrum disorders. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, 8(4), 241–248.
- Gioia, G. A., Isquith, P. K., & Guy, S. C. (2001). Assessment of executive function in children with neurological impairments. In R. Simeonsson & S. Rosenthal (Eds.), *Psychological and developmental assessment*. New York, NY: Guilford Press.
- Goldstein, G. W. (1990). Lead poisoning and brain cell function. *Environmental Health Perspectives*, 89, 91–94.
- Hall, C. W., & Kataria, S. (1992). Effects of two treatment techniques on delay and vigilance tasks with

- attention deficit hyperactive disorder (ADHD) children. *Journal of Psychology*, 126(1), 17–25.
- Harris, E. L., Schuerholz, L. J., Singer, H. S., Reader, M. J., Brown, J. E., Cox, C., . . . Denckla, M. B. (1995). Executive function in children with Tourette syndrome and/or attention deficit hyperactivity disorder. *Journal of the International Neuropsychological Society: JINS*, 1(6), 511–516.
- Harrison, L. J., Manocha, R., & Rubia, K. (2004). Sahaja yoga meditation as a family treatment programme for children with attention deficit-hyperactivity disorder. *Clinical Child Psychology and Psychiatry*, 9, 479–497.
- Hart, J., Kimbrell, T., Fauver, P., Cherry, B. J., Pitcock, J., Booe, L. Q., . . . Freeman, T. W. (2008). Cognitive dysfunctions associated with PTSD: Evidence from World War II prisoners of war. *Journal of Neuropsychiatry and Clinical Neurosciences*, 20(3), 309–316.
- Huang, Y. S., Guilleminault, C., Li, H. Y., Yang, C. M., Wu, Y. Y., & Chen, N. H. (2007). Attention-deficit/hyperactivity disorder with obstructive sleep apnea: A treatment outcome study. *Sleep Medicine*, 8(1), 18–30.
- Hyman, S. E., & Ivleva, E. (2008). Cognition in schizophrenia. *American Journal of Psychiatry*, 165(3), 312.
- Jellinger, K. A., Paulus, W., Wrocklage, C., & Litvan, I. (2001). Traumatic brain injury as a risk factor for Alzheimer disease: Comparison of two retrospective autopsy cohorts with evaluation of ApoE genotype. *Neurology*, 1186, 1–3.
- Jensen, P. S., & Kenny, D. T. (2004). The effects of yoga on the attention and behavior of boys with attention-deficit/hyperactivity disorder (ADHD). *Journal of Attention Disorders*, 7(4), 205–216.
- Kang, Y. J., Ku, J., Han, K., Kim, S. I., Yu, T. W., Lee, J. H., & Park, C. I. (2008). Development and clinical trial of virtual reality-based cognitive assessment in people with stroke: Preliminary study. *Cyberpsychology & Behavior: The Impact of the Internet, Multimedia and Virtual Reality on Behavior and Society*, 11(3), 329–339.
- Kennedy, M. R., Coelho, C., Turkstra, L., Ylvisaker, M., Moore Sohlberg, M., Yorkston, K., . . . Kan, P. F. (2008). Intervention for executive functions after traumatic brain injury: A systematic review, metaanalysis and clinical recommendations. *Neuropsychological Rehabilitation*, 18(3), 257–299.
- Kerns, K. A., Eso, K., & Thompson, J. (1999). Investigation of a direct intervention for improving attention in young children with ADHD. *Developmental Neuropsychology*, 16, 273–295.
- Klingberg, T., Fernell, E., Olesen, P., Johnson, M., Gustafsson, P., Dahlstrom, K., *et al.* (2005). Computerized training of working memory in children with ADHD—a randomized, controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44, 177–186.
- Klingberg, T., Forssberg, H., & Westerberg, H. (2002). Training of working memory in children with ADHD. *Journal of Clinical and Experimental Neuropsychology*, 24(6), 781–791.
- Kolur, U. S., Reddy, Y. C., John, J. P., Kandavel, T., & Jain, S. (2006). Sustained attention and executive functions in euthymic young people with bipolar disorder. *British Journal of Psychiatry: The Journal of Mental Science*, 189, 453–458.
- Lanphear, B. P., Dietrich, K., Auinger, P., & Cox, C. (2000). Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Reports (Washington, DC, 1974)*, 115(6), 521–529.
- Levy, F., Hay, D. A., McStephen, M., Wood, C., & Waldman, I. (1997). Attention-deficit hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(6), 737–744.
- LoPriore, C., Castelnuovo, G., & Liccione, D. (2002). Virtual environments in cognitive rehabilitation of executive functions. *Proceedings Fourth International Conference on Disability, Virtual Reality, Associated Technology*, Veszpré, Hungary.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, 16(3), 201–225.
- Marks, C., Parent, F., & Anderson, J. (1986). Retention of gains in outpatient cognitive rehabilitation therapy. *Cognitive Rehabilitation*, 4, 20–23.



- Mateer, C. A., & Mapou, R. (1996). Understanding evaluating, and managing attention disorders following traumatic brain injury. *Journal of Head Trauma and Rehabilitation*, *11*, 1–16.
- Marie, R. M., & Defer, G. L. (2003). Working memory and dopamine: Clinical and experimental clues. *Current Opinion in Neurology*, *16*(Suppl. 2), S29–S35.
- McCloskey, G., Perkins, L., & VanDivner, B. (2009). *Assessment and intervention for executive function difficulties*. New York, NY: Routledge.
- McNab, F., Varrone, A., Farde, L., Jucaite, A., Bystritsky, P., Forsberg, H., & Klingberg, T. (2009). Changes in cortical dopamine D1 receptor binding associated with cognitive training. *Science*, *323*(5915), 800–802.
- Meador, K. J. (2002). Cognitive outcomes and predictive factors in epilepsy. *Neurology*, *38*, 21–26.
- Menon, U., & Kelley, R. E. (2009). Subcortical ischemic cerebrovascular dementia. *International Review of Neurobiology*, *84*, 21–33.
- Milham, M. P., Erickson, K. I., Banich, M. T., Kramer, A. F., Webb, A., Wszalek, T., & Cohen, N. J. (2002). Attentional control in the aging brain: insights from an fMRI study of the Stroop task. *Brain and Cognition*, *49*(3), 277–296.
- Muscal, E., Bloom, D. R., Hunter, J. V., & Myones, B. L. (2010). Neurocognitive deficits and neuroimaging abnormalities are prevalent in children with lupus: Clinical and research experiences at a US pediatric institution. *Lupus*, *19*(3), 268–279.
- Noland, J. S., Singer, L. T., Arendt, R. E., Minnes, S., Short, E. J., & Bearer, C. F. (2003). Executive functioning in preschool-age children prenatally exposed to alcohol, cocaine, and marijuana. *Alcoholism, Clinical and Experimental Research*, *27*(4), 647–656.
- Nurk, E., Drevon, C. A., Refsum, H., Solvoll, K., Vollset, S. E., Nygård, O., . . . Smith, A. D. (2007). Cognitive performance among the elderly and dietary fish intake: The Hordaland Health Study. *American Journal of Clinical Nutrition*, *86*(5), 1470–1478.
- Olesen, P. J., Westerberg, H., & Klingberg, T. (2004). Increased prefrontal and parietal activity after training of working memory. *Nature Neuroscience*, *7*(1), 75–79.
- O'Reilly, R. C. (2006). Biologically based computational models of high-level cognition. *Science*, *314*(5796), 91–94.
- Ozonoff, S., & Griffith, E. (2000). Neuropsychological function and the external validity of Asperger syndrome. In A. Klinj, F. Volkmar, & S. Sparrow (Eds.), *Asperger syndrome*. New York, NY: Guilford Press.
- Penkman, L. (2004). Remediation of attention deficits in children: A focus on childhood cancer, traumatic brain injury and attention deficit disorder. *Pediatric Rehabilitation*, *7*(2), 111–123.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42.
- Posner, M., & Raichle, M. E. (1994). *Images of mind*. Rantoul, IL: Scientific American Books.
- Rabiner, D. L., Murray, D. W., Skinner, A. T., & Malone, P. S. (2010). A randomized trial of two promising computer-based interventions for students with attention difficulties. *Journal of Abnormal Child Psychology*, *38*(1), 131–142.
- Reiss, T., & Weghorst, S. (1995). Augmented reality in the treatment of Parkinson's disease. In *Proceedings of Medicine Meets Virtual Reality*, *95*, (pp. 298–302). San Diego, CA: IOS Press.
- Rice, D. C. (1993). Lead-induced changes in learning: Evidence for behavioral mechanisms from experimental animal studies. *Neurotoxicology*, *14*(2–3), 167–178.
- Richardson, A. J., & Puri, B. K. (2002). A randomized doubleblind, placebo-controlled study of the effects of supplementation with highly unsaturated fatty acids on ADHD-related symptoms in children with specific learning difficulties. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *26*(2), 233–239.
- Riggs, N. R., Laudan, B., Jahromi, R. P., Razza, J. E., Dillworth-Bart, U. M. (2006). Cognitive function and the promotion of social-emotional competence. *Journal of Applied Developmental Psychology*, *27*, 300–309.

- Robbins, J. (1996). *Wired for miracles*. Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.
- Roberts, R. E., Roberts, C. R., & Duong, H. T. (2009). Sleepless in adolescence: Prospective data on sleep deprivation, health and functioning. *Journal of Adolescence*, 32(5), 1045–1057.
- Rogeness, G. A., Javors, M. A., & Pliszka, S. R. (1992). Neurochemistry and child and adolescent psychiatry. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31(5), 765–781.
- Rohling, M. L., Faust, M. E., Beverly, B., & Demakis, G. (2009). Effectiveness of cognitive rehabilitation following acquired brain injury: A meta-analytic re-examination of Cicerone et al.'s (2000, 2005) systematic reviews. *Neuropsychology*, 23(1), 20–39.
- Rourke, B. P. (2000). Neuropsychological and psychosocial subtyping: A review of investigations within the University of Windsor Laboratory. *Canadian Psychology*, 41, 34–50.
- Ruff, H., & Rothbart, M. K. (1996). *Attention in early development: Themes and variations*. New York, NY: Oxford University Press.
- Safran, D. S. (2002). *Art therapy and AD/HD: Diagnostic and therapeutic approaches*. London, UK: Jessica Kingsley Publishers.
- Saricaoglu, F., Akinci, S. B., Gözaçan, A., Güner, B., Rezaki, M., & Aypar, U. (2005). The effect of day and night shift working on the attention and anxiety levels of anesthesia residents. *Türk Psikiyatri Dergisi (Turkish Journal of Psychiatry)*, 16(2), 106–112.
- Savage, C. R., & Rauch, S. L. (2000). Cognitive deficits in obsessive-compulsive disorder. *American Journal of Psychiatry*, 157(7), 1182–1183.
- Schwarzschild, M., Rordorf, G., Bekken, K., Buonanno, F., & Schmahmann, J. D. (1997). Normal-pressure hydrocephalus with misleading features of irreversible dementias: A case report. *Journal of Geriatric Psychiatry and Neurology*, 10(2), 51–54.
- Schweitzer, J. B., Cummins, T. K., & Kant, C. A. (2001). Attention-deficit/hyperactivity disorder. *Medical Clinics of North America*, 85(3), 757–777.
- Semrud-Clikeman, M., Filipek, P., Biederman, J., Steingard, R., Kennedy, D., Renshaw, P., & Bekken, B. (2000). Attention deficit disorder differences in the corpus callosum by MRI morphometric analysis. *Presented at the Society for Research in Child and Adolescent Psychopathology*, Santa Fe, NM.
- Shapiro, S. L., Walsh, R., & Willoughby, B. B. (2002). An analysis of recent meditation research and suggestions for future directions. *Journal for Meditation and Meditation Research*, 3, 69–90.
- Sibley, B. A., & Etnier, J. L. (2003). The relationship between physical activity and cognition in children: A metaanalysis. *Pediatric Exercise Science*, 15, 243–356.
- Sik Lányi, C., Mátrai, R., Molnár, G., & Lányi, Z. (2005). User-interface design for visually impaired children. *Elektrotechnik & Informationstechnik*, 122, 488–494.
- Singer, B. D., & Bashir, A. S. (1999). What are executive functions and self-regulations and what do they have to do with language-learning disorders? *Language, Speech, and Hearing Services in Schools*, 30, 265–273.
- Stricker, N. H., Tybur, J. M., Sadek, J. R., & Haaland, K. Y. (2010). Utility of the neuropsychological assessment battery in detecting cognitive impairment after unilateral stroke. *Journal of the International Neuropsychological Society*, 10, 1–9.
- Tam, S. F., Man, W. K., Hui-Chan, C. W., Lau, A., Yip, B., & Cheung, W. (2003). Evaluating the efficacy of tele-cognitive rehabilitation for functional performance in three case studies. *Occupational Therapy International*, 10(1), 20–38.
- Tamm, L., McCandliss, B., Liang, A., Wigal, T., Posner, M., & Swanson, J. (2007). Can attention itself be trained? Attention training for children at risk for ADHD. In K. McBurnett & L. Pfiffner (Eds.), *Attention deficit hyperactivity disorder: Concepts, controversies, new directions (Medical Psychiatry Series)*. London, UK: Informa Healthcare.
- Thaut, M. H., Gardiner, J. C., Holmberg, D., Horwitz, J., Kent, L., Andrews, G., . . . McIntosh, G. R. (2009). Neurologic music therapy improves executive function and emotional adjustment in traumatic brain injury rehabilitation. *Annals of the New York Academy of Sciences*, 1169, 406–416.

- Theotka, I. (2006). Cognitive impairment in alcoholism. *Annals of General Psychiatry*, 5, 56.
- Thornton, K. (2000). Exploratory analysis: Mild head injury, discriminant analysis with high frequency bands (32–64 Hz) under attentional activation conditions & does time heal? *Journal of Neurotherapy*, 3, 1–10.
- Tinius, T., & Tinius, K. A. (2001). Changes after EEG biofeedback and cognitive retraining in adults with mild traumatic brain injury and attention deficit hyperactivity disorder. *Journal of Neurotherapy*, 4, 27.
- Tomprowski, P. D., Davis, C. L., Miller, P. H., & Naglieri, J. A. (2008). Exercise and children's intelligence, cognition, and academic achievement. *Educational Psychology Review*, 20(2), 111–131.
- Treitz, F. H., Heyder, K., & Daum, I. (2007). Differential course of executive control changes during normal aging. *Neuropsychology, Development, and Cognition: Section B, Aging, Neuropsychology and Cognition*, 14(4), 370–393.
- Uekermann, J., Daum, I., Bielski, M., Muhlack, S., Peters, S., Przuntek, H., & Mueller, T. (2004). Differential executive control impairments in early Parkinson's disease. *Journal of Neural Transmission, Supplementum*, 68, 39–51.
- Van Dyk, K., & Sano, M. (2007). The impact of nutrition on cognition in the elderly. *Neurochemical Research*, 32(4-5), 893–904.
- Vaughan, L., & Giovanello, K. (2010). Executive function in daily life: Age-related influences of executive processes on instrumental activities of daily living. *Psychology and Aging*, 25(2), 343–355.
- Viguera, A., McCarthy, C., Whitfield, T., Poitras, J., Bekken, K., Sherman, J., & Cohen, L. (2001). *Neurobehavioral outcome following lithium exposure: What happens to lithium babies?* Presented at the Annual Meeting of the American Psychiatric Association, San Francisco, CA.
- Wall, M., & Duffy, A. (2010). The effects of music therapy for older people with dementia. *British Journal of Nursing (Mark Allen Publishing)*, 19(2), 108–113.
- Weber, A. M., & Haen, C. (2004). *Clinical applications of drama therapy in child and adolescent treatment*. New York, NY: Routledge.
- Westerberg, H., Jacobaeus, H., Hirvikoski, T., Clevberger, P., Ostensson, M. L., Bartfai, A., & Klingberg, T. (2007). Computerized working memory training after stroke—A pilot study. *Brain Injury*, 21(1), 21–29.
- Wilens, T. E., Biederman, J., Faraone, S. V., Martelon, M., Westerberg, D., & Spencer, T. J. (2009). Presenting ADHD symptoms, subtypes, and comorbid disorders in clinically referred adults with ADHD. *Journal of Clinical Psychiatry*, 70(11), 1557–1562.
- Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, 57(11), 1336–1346.
- Ylvisaker, M. (1998). *Traumatic brain injury rehabilitation, children and adolescents*. Newton, MA: Butterworth-Heinemann.
- Ylvisaker, M., & Feeney, T. J. (1998) *Collaborative brain injury intervention: Positive everyday routines*. San Diego, CA: Singular Publishing.

## Rehabilitation of Aphasia

*Mark T. Barisa, Chad A. Noggle, David B. Salisbury, and  
Justin J. Boseck*

17th century philosopher Joseph Priestly is credited with the quote, “The more elaborate our means of communication, the less we communicate.” This rings very true in our current age of e-mails, texts, tweets, Facebook posts, and emoticons. Brief comments, abbreviations, and acronyms have become commonplace in societal interactions. No matter the mechanism, language is the basis for human social interactions and communication. It ranges from the simple vowel sounds of a baby to the complex hypotheses of a scientist or from the basic acronym (LOL) to the writings of a great philosopher. Regardless of the complexity, language and communication rely on specific neurologic processes, linguistic and lexical understandings, sensory–motor functions, learning and memory, emotional/ limbic expressions, and continuous information processing. Communication is multi-faceted including the ability to converse, comprehend, repeat, read, and write. When the ability to express oneself through language is damaged, many of our life’s daily activities and social relationships become nearly impossible to manage. As such, assessment and rehabilitation of disorders of language and communication require a multi-faceted approach.

While related, speech and language disturbances are distinct in their presentation and pathology. Speech is the mechanical aspect of oral communication of language. It includes the sensory–motor functions that coordinate the muscles controlling the production of verbalization. Common disorders of speech include dysarthria (disturbance of articulation) and

dysphonia (disturbance of vocalization or phonation). Language, in contrast, is the cognitive aspect of symbolic communication. It includes the ability to converse, comprehend, repeat, read, and write. It depends heavily on higher level processing for understanding of information received and the formulation of expressed thoughts and ideas through appropriate sounds and symbols of communication. Aphasia is defined as acquired language impairment after neurologic damage, which affects some or all language modalities: expression, understanding of speech, reading, and writing. The term aphasia (less commonly referred to as dysphasia) does not include other communication difficulties attributed to sensory loss, confusion, dementia, or speech difficulties caused by muscular weakness or dysfunction such as dysarthria (Kelly, Brady, & Enderby, 2010). As such, the focus of this chapter will be the classic aphasia syndromes, rather than disturbances in the motor aspects of speech production.

## **NEUROANATOMY AND CLINICAL CHARACTERISTICS OF COMMON LANGUAGE DISORDERS**

Over time and with increasingly sophisticated research tools, conceptualization of the neuroanatomy of language has progressed from an emphasis on localization to disconnection syndromes, parallel processing paradigms, dual stream theories, and now more expansive network models. In a true testament to the pioneering ideas of Paul Broca and Carl Wernicke, many of their thoughts regarding language-related areas and syndromes continue to be refined. The following brief review will primarily focus on neuroanatomy frequently associated with traditional aphasia syndromes after vascular events among individuals with left hemisphere language dominance. The reader will already understand undoubtedly that early life neurological events can result in atypical language development outside the ipsilateral perisylvian areas and within the non-language-dominant hemisphere. In addition, aphasia syndromes seen after tumor, infections, traumatic brain injuries, dementias, and other neurological conditions are not specifically discussed in the following anatomical overview.

There remains strong but still challengeable support for the conceptualization of traditional aphasia syndromes and the predominance of aphasia occurring after left hemisphere strokes (Moser, Papanicolaou, Swank, & Breir, 2011; Ochfeld et al., 2010; Yang, Zhao, Chun-Xue, Hong-Yan, & Zhang, 2008). Still, non-classic presentations can be seen with higher frequency after right hemisphere middle cerebral artery (MCA) infarctions or among individuals with aphasia syndromes that are not primarily expressive in nature (Croquelois

& Bogouslavsky, 2011). Hence, categorization systems should be used to guide the initial assessment and diagnosis. The astute clinician will be watchful for inconsistencies and atypical aphasia syndromes.

## **PRIMARY APHASIAS (PERISYLVIAN)**

### **Broca's Aphasia (Nonfluent Aphasia)**

The nonfluent aphasia syndrome commonly referred to as Broca's aphasia can have varying presentations based on the anatomical areas compromised. Case studies have supported acute presentations of initial mutism or verbal apraxia with rapid initial improvement after left posterior inferior frontal cortex lesions solely impacting Broca's region (Brodmann areas 44 and 45; Lazar & Mohr, 2011). The typical Broca's syndrome often involves infarction of additional regions dependent on MCA distribution, particularly in the superior branch. This can include cortical areas adjacent to Broca's region, the left insula, subcortical white matter pathways, and the basal ganglia (Mohr et al., 1978; Ochfeld et al., 2010). This larger network of vulnerable regions for Broca's aphasia is consistent with the later examination of Paul Broca's infamous patient "Tan" which revealed a more pronounced lesion of the frontal and parietal regions (Lazar & Mohr, 2011). This also helps in explaining the frequent initial presentation of global aphasia improving into a subsequent Broca's aphasia among individuals with infarcts impacting previously identified areas. Furthermore, disconnection of language circuitry or hypoperfusion of cortical regions have been hypothesized as variables in those who present with Broca's aphasia after subcortical infarcts or lesions that appear to be well outside of Broca's area (Fridriksson, Bonilha, & Rorden, 2007).

Broca's aphasia is characterized by a disturbance in expressive language. Fluency is disturbed in all aspects of speech (repetition, reading, and speaking) and language/grammar, whereas comprehension remains intact. Descriptions of Broca's aphasia are variable in terms of deficit severity. In its mildest description, Broca's aphasia is characterized by dysarthria and poverty of speech. Basic comprehension is typically spared, but as complexity and length of directives increase, deficits can arise in more severe cases. In milder cases, an individual may demonstrate the ability to produce verbalizations; however, deficits are demonstrated in prosody. Speech in these cases tends to be slow and effortful with errors in articulation and typically consists of transitive verbs, nouns, or specific adjectives, whereas prepositions, articles, and conjunctions are

omitted. At its most severe, loss of all aspects of expressive communication are described. In severe cases, individuals will exhibit an inability to verbalize in conversation, in reading, or in repetition though ability to shout and vocalize remains intact. Individuals may on occasion demonstrate the ability to verbalize “yes” and “no” or monophasia (repetition of verbal automatisms/perseverative pattern of utterances). Insight into deficits is typically preserved which may result in frequent attempts at self-correction or ultimately emotional despair.

In addition to impaired speech production, patients with Broca’s aphasia also typically demonstrate impairments in writing. Errors in letter formation and spelling are observed in dictation, though information may still be copied. Physical aspects associated with Broca’s aphasia include paresis of the right lower face and upper extremity with occasional involvement in the lower extremity. In some cases, the tongue may deviate to the right side. Buccofacial apraxia is often present and may be demonstrated by the inability to purse the lips, smack, lick the lips, blow, or whistle, despite the individual’s ability to understand both written and auditory commands. However, imitation of buccofacial actions may be preserved. Ideomotor apraxia of the nondominant left hand sometimes accompanies this disorder (apraxia can be present in both the hands, but often times cannot be assessed in the dominant right hand because of paresis).

### **Wernicke’s Aphasia (Fluent Aphasia)**

Wernicke’s aphasia is the other well-documented and researched aphasia syndrome that is also referred to as a fluent or sensory aphasia. Although Wernicke’s area (Brodmann area 22) has been debated, it is most frequently associated with superior temporal gyrus and inferior parietal regions including the supramarginal and angular gyri. A well-circumscribed stroke solely in Brodmann area 22 would more likely present with auditory agnosia or less severe syndrome as compared to the constellation of deficits associated with Wernicke’s aphasia often seen after an inferior division MCA infarction (Festa, Lazar, & Marshall, 2008). Varying sensory and cognitive deficits are to be expected based on the large region attributed to this syndrome.

Wernicke’s aphasia is characterized by phonemic paraphasias and deficits in receptive language, whereas fluency of speech is preserved. Individuals with Wernicke’s aphasia demonstrate an inability to comprehend both written and spoken language. Speech remains fluent and effortless with normal rate, volume, and prosody; however, paraphasic errors and neologisms are notable and speech

is typically unintelligible or meaningless. Writing-to-dictation is also impaired, though individuals may be able to copy information presented. When a patient's ability to copy written material is preserved, copy is typically slow and effortful, and insight is typically impaired. Repetition may remain intact in some cases, but this is typically associated more with transcortical sensory aphasia (see below).

The distinct language deficits of a Wernicke's aphasia syndrome can be accompanied by other more subtle findings. Because of poor comprehension, the detection of visual impairment (i.e., partial or complete right homonymous hemianopsia) can be quite challenging. Motor weakness is not expected but ideomotor apraxia with varying benefit when attempting to model motor movement can be present. Many individuals with Wernicke's aphasia syndromes demonstrate poor awareness of deficits. In early stages, agitation and even paranoia can occasionally result in concern for a psychiatric disorder before a thorough medical workup.

### Global Aphasia

Global aphasia syndromes involve profound expressive and receptive language deficits as the name suggests. Global aphasias are seen in larger distribution vascular events. Typically, this is the result of left internal artery occlusion, cardioembolic MCA stem events, or large left subcortical strokes (Kirshner & Mark, 2009). Direct and indirect damages to inferior frontal, superior temporal, inferior parietal regions along with the basal ganglia, insula, or deep white matter pathways can be implicated. As previously mentioned, global aphasia can be an initial stage of a Broca's aphasia syndrome. A much less common subtype of global aphasia without hemiparesis can be present after discrete lesions in frontal and parietal-temporal regions often related to embolic or metastatic etiologies (Damasio & Damasio, 2000).

Global aphasia is characterized by disturbances in all aspects of language. In its mildest form, patients with global aphasia may be able to produce verbalizations, though these are typically characterized by habitual phrases. Individuals may be able to imitate sounds, vocalize single syllables, or shout. These patients may also demonstrate an ability to comprehend brief phrases; however, they are unable to follow commands or name to confrontation. Reading, writing, and repetition are impaired. Gestural communication, specifically gestures of greeting, indications of modesty, avoidance behaviors, or self-help actions, may be demonstrated. Prognosis for individuals with global



aphasia resulting from vascular insults is typically poor. However, in cases of trauma, edema, postconvulsive paralysis, or metabolic imbalance superimposed on remote lesions, improvement is common. Over the course of recovery, features more consistent with Broca's aphasia tend to emerge. Associated features of global aphasia typically include right homonymous hemianopsia and hemiparesis and hemianesthesia of the right upper extremity.

## **DISCONNECTION/DISSOCIATIVE APHASIA SYNDROMES (EXTRASylvian)**

With the increasing understanding of complex cortical–subcortical circuitry, the presence of aphasia syndromes secondary to subcortical damage should not be surprising. Varying mechanisms including cortical hypoperfusion, diaschisis, disconnection syndromes or even the missed presence of accompanying subtle cortical lesions have been debated. The consistency of presentations has been controversial in this subgroup of syndromes. Striatocapsular infarctions involving the head of the caudate nucleus, anterior limb on the internal capsule, and some portion of the putamen can arise from the deep penetrating lenticulostriate branches of the left MCA (Kuljic-Obradovic, 2003). Thalamic aphasia syndromes can be seen more frequently after anterolateral damage. Vascular events affecting tuberothalamic artery from posterior communicating artery, posterior choroidal artery, and the paramedian artery of the left posterior cerebral artery (PCA) have been reported (Ferro, 2001). Furthermore, vascular insults to white matter surrounding the previously mentioned areas have also been implicated as a cause of subcortical aphasia syndromes (Ferro, 2001; Kuljic-Obradovic, 2003; Naeser et al., 1982). Although overly simplified, syndromes impacting anterior subcortical regions tend to present with more aspects of motor aphasia, whereas posterior subcortical infarctions show a greater propensity of sensory aphasia deficits.

### **Transcortical Aphasias**

The hallmark feature of transcortical aphasia syndromes is preserved repetition. Transcortical motor aphasia otherwise generally mimics Broca's aphasia and can be seen as a stage in the recovery from a Broca's aphasia syndrome. Infarctions in the anterior cerebral artery region, distal portions of the superior division of the MCA, or watershed regions between the anterior and the middle cerebral arteries are the more likely causes of this syndrome. Such infarcts result in damage anterior or superior to Broca's region, in deep white matter tracts, or in

the supplementary motor area (Kirshner & Mark, 2009). In transcortical motor aphasia, deficits are primarily in aspects of automatic speech, whereas repetition remains intact. Specifically, initiation of speech is impaired, and verbal output can be marked by semantic paraphasia or perseverative tendencies. Naming abilities can be variable relative to the severity of the disorder. Comprehension is preserved. Transcortical motor aphasia may occur during recovery from Broca's aphasia.

Similarly, transcortical sensory aphasia will present with most features of Wernicke's aphasia outside of intact repetition. This uncommon syndrome is often produced after damage to watershed regions between MCA-PCA impacting posterior temporal regions, the temporal-parietal occipital lobe junction, and the thalamus (Festa et al., 2008). Individuals with transcortical sensory aphasia demonstrate impairments in receptive language, whereas repetition remains intact. Specifically, comprehension of auditory and written information is impaired; reading and writing are also impaired; and speech is fluent, though marked by paraphasic errors, circumlocution, and anomia.

A mixed transcortical syndrome can mimic global aphasia and is more frequently seen after left hemisphere or bilateral watershed infarcts, as a transitional stage after a global aphasia, after large anterior thalamic lesions, or in some later stage of dementia syndromes. There has also been suggestion that watershed infarcts often present with a transcortical mixed subtype in the very acute stage and then progress into a primary transcortical motor and sensory aphasia syndrome (Flamand-Roze et al., 2011).

### Conduction Aphasia

The neuroanatomy and even existence of conduction aphasia has been controversial. This is further fueled by suggestion of subtypes of conduction aphasia and the presence of the syndrome in the recovery stage of Wernicke's aphasia. Initially, conduction aphasia was considered a disconnection syndrome involving the arcuate fasciculus. The arcuate fasciculus, which is found in the superior longitudinal fasciculus, was conceptualized as the key white matter tract connecting temporal lobe comprehension regions (i.e., Wernicke's area) with frontal lobe expressive language regions (i.e., Broca's area). The endpoint of the arcuate fasciculus has been questioned. There is support for premotor and motor area endpoints, suggesting an indirect connection to Broca's area (Ardila, 2010). In addition to the arcuate fasciculus, areas that may play a role in conduction aphasia include the supramarginal gyrus of the inferior parietal regions, superior

insula, and left primary auditory cortices (Bernal & Ardila, 2009; Catani, Jones, & ffytche, 2005; Damasio & Damasio, 2000). In fact, some research suggests that damage to the left inferior parietal lobe and underlying white matter are highly predictive of impaired speech repetition (Fridriksson et al., 2010). Recent network models of conduction aphasia have argued for a key left posterior sylvian parietal–temporal region which may better explain the range of deficit that can accompany primary repetition problems (Flamand-Roze et al., 2011).

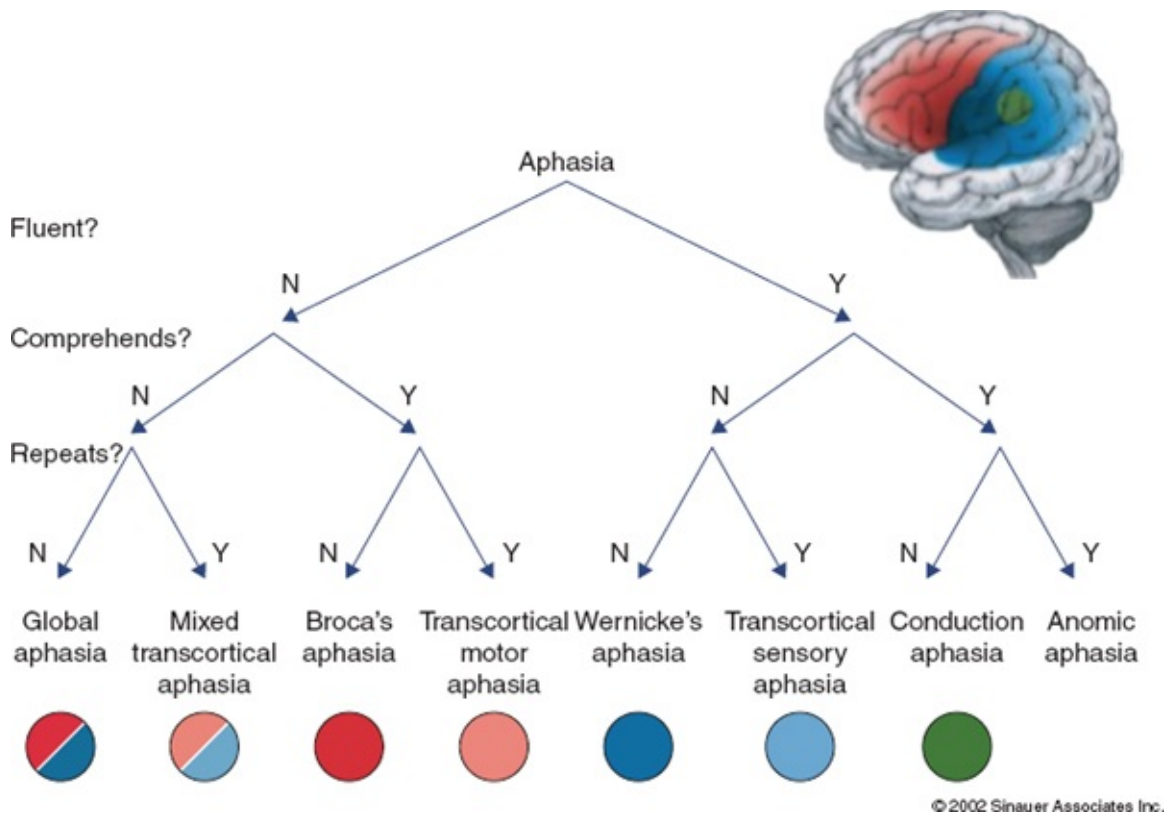
Conduction aphasia is characterized by disordered phonological processing. In conduction aphasia, repetition is impaired for both single- and non-word utterances. Grammar and comprehension are typically spared. Self-initiated speech typically remains fluent with preserved articulation and prosody, though paraphasic errors may be observed. Reading and writing may also be impaired. Patients are typically insightful with regard to deficits and often make frequent attempts at self-correction.

There are two types of conduction aphasia identified: repetition and reproduction. However, some overlap may occur between repetition and reproduction conduction aphasia and these may be better thought of as end points on a continuum. In repetition conduction aphasia, confrontation naming and automatic speech remain intact. There is typically no evidence of paraphasic errors. Phonetic discrimination, auditory short-term memory, and repetition are impaired. In reproduction conduction aphasia, impairments are observed in repetition, confrontation naming, and automatic speech with additional phonemic paraphasic errors demonstrated. Auditory short-term memory is typically spared in reproduction conduction aphasia. Figure 5.1 shows an anatomical depiction of the syndromes just discussed.

## OTHER APHASIA OR RELATED SYNDROMES

### Anomic Aphasia

Limited attention is given to anomic aphasia given the lack of neuroanatomical specificity. This is often a residual state of various aphasic disorders. It has been suggested that anomic aphasia may also be a mild form of a transcortical sensory aphasia, but this isolated deficit appears more often in the early stages of progressive dementia syndromes.



**FIGURE 5.1** Classification of language disorders. Adapted from Blumenfeld (2002). Used with permission from Sinauer Associates, Inc.

Anomic aphasia is a relatively uncommon syndrome in which individuals exhibit deficits in single-word production, specifically in production of common nouns. In this syndrome, individuals are frequently unable to name objects or people and demonstrate halting speech, wordfinding difficulties, circumlocution, and word substitution. Deficits in naming apply to the not only objects seen but also names of items that are heard or felt. Ability to correctly use objects, identify objects, or recognize the name of an object from a list is preserved. Comprehension and repetition remain intact. Insight is typically poor.

### Pure Word Deafness (Auditory Verbal Agnosia)

Pure word deafness is an uncommon syndrome characterized by an individual's inability to recognize spoken words in the absence of aphasia or defective hearing. Studies have suggested that this syndrome is because of lesions that interrupt white matter connections from left and right primary auditory receptive areas that project to Wernicke's area in the dominant hemisphere (Shoumaker, Ajax, & Schenkenberg, 1977). This is commonly caused by an occlusion of a small branch of the inferior division of the MCA. In this syndrome, auditory

comprehension, repetition, and writing-to-dictation are also impaired. Self-initiated speech typically remains without error, with the exception of occasional paraphasias. Similarly, self-initiated writing typically remains preserved. Comprehension of written information remains intact. Patients typically lack insight into comprehension deficits, often claiming experience of hearing impairment. Pure word deafness may present as Wernicke's aphasia resolves but is conceptually considered a result of injury to the auditory processing system.

### **Pure Word Blindness**

Pure word blindness or alexia without agraphia is a disconnection syndrome that involves a disruption of the communication pathways between the left and the right cerebral hemispheres. Lesions are typically noted in the left visual cortex and underlying white matter (geniculocalcarine tract). Although visual information from the left hemisphere is disrupted (because of a right hemianopsia), visual information from the right visual cortex is disconnected from left hemisphere language processing because of a disruption in the transfer through the posterior commissure and splenium of the corpus callosum. This results in the inability to read written language, despite retaining the ability to write words normally. Although most patients with pure word blindness typically experience a right homonymous hemianopsia, lesions in the deep white matter at the junction between the parietal and the occipital lobe can interrupt the projections of visual information to the language centers without an associated visual field cut.

In pure word blindness, word reading is impaired. However, patients may be able to read individual letters or numbers. A patient with this syndrome may also be able to spell aloud or identify words by having them spelled or by reading letter by letter. Individuals frequently lose the ability to name colors. Auditory comprehension, repetition, and conversational skills are typically preserved. The ability to copy written words is typically impaired, whereas self-initiated writing remains preserved. However, with regard to the latter, individuals are frequently unable to read information they have written.

### **Pure Word Mutism (Aphemia)**

Pure word mutism, also call aphemia, is characterized by complete loss of speaking ability. This is typically associated with a lesion involving the lower most part of the precentral gyrus with a sparing of Broca's area. Auditory comprehension, writing, reading, and repetition remain intact. With recovery, an

individual may demonstrate complete syntax with no loss of vocabulary or grammatical error, though dysarthria may be evident. Complete recovery, however, typically occurs within weeks to months. Associated features typically include brachial and facial paresis.

### **Nondominant Hemisphere Aphasias**

The varying literature detailing the presence of aphasia syndromes after vascular events to the right hemisphere, cerebellum, and other areas less commonly associated with language deficit will continue to challenge even the more complex network models of language. Extensive review of such syndromes is outside the scope of this chapter. Still, a few trends are worth noting in aphasia after right hemisphere stroke among individuals without prior neurological complications. These cases do not necessarily have larger lesion sizes or vastly different neuroanatomical regions as the traditional left hemisphere stroke in many cases yet atypical language patterns and anatomical variability are more frequent (Dewarrat et al., 2009; Mariën, Engelborghs, Vignolo, & De Deyn, 2001).

## **COMMON CAUSES OF LANGUAGE DYSFUNCTION**

As described previously, aphasias are acquired language impairment after brain damage, which affects some or all language modalities. Although speech and language deficits are also noted in the context of some congenital/developmental disorders, these will not be presented in this chapter. The most common acquired brain etiologies that result in aphasia include strokes, head trauma, brain tumors, and brain infections; however, any brain tissue damage, for whatever reason, that occurs in the language centers of the brain may cause aphasia.

### **Cerebrovascular Accidents and Malformations**

The most common cause of aphasia remains cerebrovascular accidents, mainly involving the left hemisphere, where the language function of the brain is usually situated (Kelly et al., 2010). A small percentage of left-hand-dominant individuals may be cross-dominant (right hemisphere dominant for language), but this is a rare occurrence. Most of the available information related to aphasia is based on research conducted on patients after a stroke. This is because of the precise boundaries of the affected area and the related functional deficits. Strokes involving the left MCA or its branches commonly result in aphasic

disturbance because of its perfusion of the perisylvian regions and connection pathways. Strokes involving the other arterial branches are less likely to result in primary aphasic disturbances but can lead to other disruptions of speech and communication.

Although the resulting aphasia after a cerebral hemorrhage is similar to that noted after infarctions, patients with a hemorrhage often present as being much more impaired initially because of the complex physiologic pathology including intraventricular blood, mass effects, increased intracranial pressure, and other factors. As such, the initial effects of the hemorrhage do not adhere to the typical margins associated with specific arterial branches. Aphasia syndromes after hemorrhage tend to be more complex and involve co-morbid deficits associated with areas outside of the primary area of injury. On a positive note, these patients tend to show more rapid recovery of deficits once the acute hemorrhage has resolved.

Cerebrovascular events in children have demonstrated a unique recovery trajectory relative to adults. A predominant theory regarding early stroke and its effect on language development is that early left hemisphere lesions trigger compensatory processes that allow the right hemisphere to assume dominant language functions. [Beharelle \*et al.\* \(2010\)](#), in a study testing this theory, found that participants with bilateral activation in left and right superior temporal-inferior parietal regions had better language function than those with either predominantly left-or right-sided unilateral activation. This supports a strong predisposition for typical neuronal language organization, despite an intervening injury, and argues against models suggesting that the right hemisphere fully accommodates language function after early injury.

### **Traumatic Brain Injury**

Cerebral contusions, depressed skull fractures, and hematomas of the intracerebral, subdural and epidural spaces after traumatic brain injury may all cause aphasia when they disrupt or compress the language centers of the left hemisphere ([Drago & Foster, 2012](#)). The types of presenting language disorders follow the localization patterns discussed earlier in this chapter. However, in comparison, trauma tends to be less localized than ischemic stroke. In addition, damage to the frontal and the temporal lobes can often interfere with the ability to use knowledge and skills, especially verbal skills, or result in neurobehavioral or neuropsychiatric syndromes that negatively impact rehabilitation interventions. Additional communication deficits after frontal lobe injury reflect

an impaired quality of speech and interactions, but not necessarily aphasic disturbance. These include a lack of logical content, cohesiveness, and clarity; insensitivity to other's needs and interests; insensitivity to the amount of information necessary for another to comprehend the information given; confabulation and inconsistencies; and impaired social comportment and pragmatics (knowledge and activities of socially appropriate communication; Lezak, Howieson, & Loring, 2004). Thus, cognitive-communication deficits after traumatic brain injury are multi-faceted and extend beyond the typical aphasia syndromes, making rehabilitation more complicated.

### **Brain Tumors**

Brain tumors can cause language impairments based on the location of the tumor itself, and the potential mass effect. However, tumors are more often associated with a gradual onset of aphasia when they involve the dominant, usually left, hemisphere. This allows for ongoing compensation as the difficulties progress. Aphasic symptoms typically present according to the location of the tumor, level of malignancy, and speed of growth of the tumor. Tumors arising near the cranial fossas are not directly associated with aphasia, but contribute to symptoms of increased intracranial pressure indirectly affecting language centers. To further complicate language disorders in patients with tumors, the effects of chemotherapy, radiation, and neurosurgical interventions may further contribute to the level and type of presenting deficits.

### **Infectious Processes**

Chronic infections, such as tuberculosis, syphilis, and herpes simplex, that have a predilection for the temporal lobe and frontal cortex may be associated with aphasia as well as a range of other symptoms such as seizures, confusion, headaches, and fever. Although severe amnesic deficits are seen in these cases, patients with extensive left-sided involvement, including the left inferior temporal lobe, often show associated language deficits (Warrington & Shallice, 1984). AIDS and more opportunistic infections can cause focal lesions in a variety of locations in the brain and the AIDS–dementia complex may be associated with language deficits depending on the extent and the location of the associated lesions.

### **Neurodegenerative Processes**



Neurodegenerative processes are commonly associated with impairments in language. The types of language deficits vary across the different dementia subtypes and several will be presented in this chapter. However, in all cases, the onset of the language dysfunction is gradual in nature, and at times can be mistaken as disturbances of memory or other cognitive processes. Only brief descriptions will be provided here as rehabilitation in neurodegenerative conditions focuses more on maintenance of daily living skills rather than rehabilitation of progressive language declines.

### Dementia of the Alzheimer Type (DAT)

The initial symptoms of DAT include memory loss and language disturbances. The language-based difficulties typically begin as an anomia and gradually evolve toward symptoms associated with transcortical sensory aphasia. There is a slow erosion of semantic knowledge that begins with complex words gradually progressing to a loss of concepts (Hodges, Salmon, & Butters, 1992; Smith, Faust, Beeman, Kennedy, & Perry, 1995). This coupled with the insidious decline of memory and other cognitive processes result in a dramatic loss of communication abilities in the later stages of the disease process.

### Multi-Infarct Dementia (MID)

The presentation of language deficits in MID is based primarily on the location of the infarcts. If one or more of the lesions is located in primary language areas, the presenting aphasia will reflect the subtype associated with that location as it would with an individual infarction. However, in MID, the typical course is characterized by multiple small infarctions in the subcortical white matter. As a result, a variety of direct and indirect cognitive-communication deficits develop including hypophonia, dysprosody, articulation problems, and limited rate, quantity, and quality of verbal expressions. A recognizable aphasic syndrome may not be present as patients will instead demonstrate cognitive deficits commonly associated with frontal and prefrontal lesions including poor generative fluency, limited elaboration of response, and reduced spontaneity of speech. These difficulties are typically accompanied by slow acquisition and impaired retrieval of novel information in the context of relative sparing in recognition memory.

### Primary Progressive Aphasia (PPA)

PPA is a neurodegenerative condition that is characterized by a relentless progression of language disturbance in the context of spared cognitive functions in other domains (Mesulam, 1982). PPA is characterized by an insidious and progressive course of impairment in word finding, naming, syntax, and word comprehension that can remain isolated for two or more years, whereas daily activities and other cognitive functions remain relatively intact. Ideomotor apraxia and acalculia will sometimes be present as well. After the first 2 years, additional cognitive domains are gradually affected, but language remains the most prominent impairment.

PPA has been divided into three subtypes that differ in their underlying neuropathology which corresponds with differences in their clinical presentation. In *semantic dementia*, sometimes referred to as *fluent PPA*, individuals present with a prominent loss in the meaning of words and objects in the presence of grammatically correct speech and preserved syntactic comprehension. Spontaneous speech is fluent despite the aforementioned loss of object and word meaning making verbal output empty (Hodges et al., 1992). Articulation, syntax, and repetition remain intact in the early stages but decline later in the disease process. *Nonfluent PPA*, in comparison, is characterized by poor and difficult articulation, disrupted fluency and prosody, paraphasias (most commonly phonemic), agrammatism, and variable anomia in the presence of preserved comprehension (Gorno-Tempini et al., 2004). Apraxia of speech and anarthria have also been associated with this variant (Broussolle et al., 1996; Gorno-Tempini et al., 2004). *Lopogenic PPA* manifests as slowed speech and frequent wordfinding pauses that are noted in the presence of relatively normal articulation. This is thought to be related to short-term phonological memory deficits (Gorno-Tempini et al., 2004). Phonemic paraphasias are common (Kertesz, Davidson, McCabe, Takagi, & Munoz, 2003), and syntax and comprehension are impaired aside from the simplest incidences. This form of PPA has also been described as an atypical variant of Alzheimer dementia because of its similarities in terms of memory and comprehension deficits.

### Temporary Impairments

Beyond the more chronic deficits in language associated with the aforementioned disorders, language impairments can present in a transient fashion as well. A number of neurologic conditions can result in a temporary aphasic disturbance, but some of the most common etiologies include transient ischemic cerebrovascular events (TIAs ), complex migraine presentations,

seizures (peri-ictal), drug or alcohol toxicity, and acute metabolic or infectious processes. These temporary language disruptions typically fully resolve in a short period of time or after appropriate medical management of the acute medical problem. Rehabilitative follow-up is rarely warranted in such cases, provided that they show the expected recovery trajectory.

## ASSESSMENT OF APHASIA AND LANGUAGE

The differential diagnosis of the various aphasia subtypes can seem complex on the surface, but a good understanding of the neuroanatomy and related signs and symptoms make this relatively straightforward. [Figure 5.1](#) and [Table 5.1](#) provide summaries of common aphasia subtypes to provide a framework for differential diagnosis. Assessment of verbal and language functioning should consider multiple skills including: speech content, fluency, repetition, comprehension, naming, reading, and writing. Clinicians may pursue this assessment through both informal and standardized fashions. Because the goal of language assessment is to identify the strengths and weaknesses across multiple areas of language functioning, a variety of measures are often required. As is the case with other domains, there is no single assessment method that all researchers or practitioners use to assess language functioning; rather, a great deal of variation exists in research and clinical practice ([Sullivan & Riccio, 2010](#)).

The assessment of language, including aphasia, begins with history taking, clinical review, and informal assessment. Within the framework of the initial interview, information such as handedness, visual and auditory acuity, and educational and occupational histories should be obtained ([Beeson & Rapcsak, 2006](#)). The interview process has an added benefit in that it engages the patient in direct conversation, providing a wealth of diagnostic information. Much can be learned through basic conversational interaction coupled with simplistic tasks such as having the patient name items on the clinician's person, asking the patient to repeat phrases, or asking the patient to follow a multi-step command. Such techniques are typically adequate to discern the presence or the absence of aphasia, provide an estimate of the severity, establish a profile, and document the relative strengths and weaknesses of the patient ([Beeson & Rapcsak, 2006](#)).

As noted by [Lezak et al. \(2004\)](#), formal testing should be undertaken once aphasia is known to be present or strongly suspected. The nature and extent of the evaluation, including the measures utilized, are then dependent on the purpose of the evaluation itself ([Spreeen & Risser, 1991](#)). Measures of language and communication range from the very specific to comprehensive batteries

designed to assess the full spectrum of cognitive-communication activities. Some are meant to identify abilities in a single area (e.g., auditory comprehension), whereas others are designed to yield specific diagnostic categorization (Boston Diagnostic Aphasia Examination, BDAE). [Table 5.2](#) includes a listing of some of the more common batteries and individual measures, though this list is not exhaustive.

**TABLE 5.1 Clinical Presentations of Common Aphasia Syndromes**

TYPE OF APHASIA	COMPREHENSION IMPAIRED	EXPRESSIVE SPEECH IMPAIRED	REPETITION IMPAIRED	POSSIBLE ASSOCIATED DEFICITS
Broca's aphasia	Primarily intact although may have impaired comp. of syntactically dependent phrases	X	X	Dysarthria and Rt. hemiparesis of arm and face more than leg primarily when Lt MCA superior division infarct is cause
Wernicke's aphasia	X	Primarily spared although speech may be empty, meaningless, and full of nonsensical paraphasic errors. May demonstrate occasional neologisms	X	Contralateral visual field cut (usually of Rt. upper quadrant); dysarthria and Rt. hemiparesis usually absent or mild. Often demonstrate anosognosia. May exhibit angry or paranoid behavior
Global aphasia	X	X	X	Eventually improve to become a Broca's aphasia**
Conduction aphasia	Normal/spared	Normal/spared	X	
Transcortical motor aphasia	Normal/spared	X (impaired fluency)	Normal/spared	Related to possible ACA-MCA watershed infarct—destroys connections other regions of the frontal lobe**
Transcortical sensory aphasia	X	Normal/spared	Normal/spared	Related to possible MCA-PCA watershed infarct—affects connections to structures in the parietal and temporal lobes**
Mixed transcortical aphasia	X	X	Normal/spared	Combined ACA-MCA and MCA-PCA watershed infarcts**

ACA, anterior cerebral artery; MCA, middle cerebral artery; PCA, posterior cerebral artery. "Adapted from [Pagoria \(2012\)](#).

**TABLE 5.2 Common Measures of Language Functioning**

APHASIA BATTERIES	REFERENCE
<b>Name of Measure</b>	
Boston Diagnostic Aphasia Examination-3rd Ed. (BDAE-3)	Goodglass, Kaplan, and Barresi (2000)
Communication Abilities in Daily Living-2nd Ed.	Holland, Frottall, and Fromm (1999)
Multilingual Aphasia Examination	Benton, Hamsher, and Sivan (1994)
Western Aphasia Battery	Kertesz (1982)
Aphasia Screening Test	Halstead and Wepman (1949)
<b>Skill-Specific Measures</b>	
<b>Tests of Naming</b>	
Boston Naming Test	Goodglass and Kaplan (2000)
Visual Naming Test	Benton et al. (1994)
<b>Tests of Vocabulary and Expression</b>	
Clinical Evaluation of Language Fundamentals-4 <sup>th</sup> Ed.	Semel, Wig, and Secord (2003)
Comprehensive Receptive & Expressive Vocabulary Test-2 <sup>nd</sup> Ed.	Wallace and Hammill (2002)
Expressive One-Word Picture Vocabulary Test	Gardner (2000a)
Expressive Vocabulary Test-Second Edition	Williams (2007)
Peabody Picture Vocabulary Test-4 <sup>th</sup> Ed.	Dunn and Dunn (2007)
Receptive One-Word Picture Vocabulary Test	Gardner (2000b)
Vocabulary subtest-Part of the Wechsler intelligence tests	Wechsler (2008)
<b>Tests of Spontaneous Speech &amp; Discourse</b>	
Cookie Theft picture narrative-Part of BDAE-3	Goodglass et al. (2000)
<b>Tests of Verbal Fluency</b>	
Controlled Oral Word Association Test (COWAT)	Benton et al. (1994)
DKEFS Verbal Fluency	Delis, Kaplan, and Kramer (2001)
<b>Tests of Verbal Repetition</b>	
Sentence Repetition	Benton et al. (1994)
<b>Tests of Verbal Comprehension</b>	
Comprehensive Receptive & Expressive Vocabulary Test-2nd Ed.	Wallace and Hammill (2002)
Test for Auditory Comprehension of Language-3rd Ed.	Carrow-Woolfolk (1999)
Token Test	Benton et al. (1994)
<b>Tests of Reading</b>	
National Adult Reading Test-2nd Ed.	Nelson and Willison (1991)
North American Adult Reading Test	Blair and Spreen (1989)
Reading subtest-Wide Range Achievement Test 3	Wilkinson (1993)
Letter-Word Identification subtest-Woodcock-Johnson III-Tests of Achievement	Woodcock, McGrew, and Mathers (2001)
Wechsler Test of Adult Reading	Wechsler (2001)
<b>Tests of Spelling</b>	
Spelling subtest-Wide Range Achievement Test-3	Wilkinson (1993)
Spelling subtest-Woodcock-Johnson III-Tests of Achievement	Woodcock et al. (2001)

## THE ASSESSMENT PROCESS

Observation remains a powerful tool of practicing neuropsychologists and greatly contributes to the diagnostic, assessment, and treatment-planning process. Within the aphasia, as previously suggested, assessment starts at the time of initial consultation, clinical review, and informal assessment which includes continual observation of the patient through ongoing interaction. This can become crucial in differentiating aphasic disorders from speech deficits that are actually breakdowns in the mechanics of phonation and articulation, which was discussed in the previous sections. Interview and basic assessment can, in and of itself, be sufficient to diagnose many of the specific aphasia syndromes described in this chapter. By simply engaging in conversation with the patient,

the clinician may begin to develop a feel for the patient's language abilities. Professionals may determine whether the patient appears to understand questions posed, exhibits fluent or nonfluent speech, or exhibits wordfinding errors, paraphasic responses, or neologisms. Observation of speech fluency during general conversation is of particular interest from a differential and, in turn, intervention standpoint. Similarly, the clinician's evaluation of the actual content of the patient's speech is relevant to the clinical picture. Speech content is sometimes overlooked in the discussion of aphasia/ language assessment in favor of the traditional areas of comprehension, naming, and repetition. Yet, observation of phonemic or semantic paraphasias, neologisms, paragrammatism, or unrelated jargon in spoken or written responses can provide further data toward characterizing potential aphasic syndromes (Festa et al., 2008). Critical questions to answer during the observation of conversational speech, as noted by Beeson and Rapcsak (2006) include:

- (a) Is there evidence of wordfinding difficulties?
- (b) Does the patient exhibit circumlocution in speech?
- (c) Is speech characterized by long pauses or hesitations?
- (d) Does the patient exhibit neologisms, semantic paraphasias, or phonemic paraphasias?
- (e) Is speech output fluent or nonfluent?
- (f) Is speech prosody normal?
- (g) How long are speech utterances?
- (h) Is articulation impeded?

Depending on the information obtained through clinical review and informal evaluation, a clinician can decide whether to pursue a comprehensive assessment, utilize a more limited screening of specific aspects of language functioning, or not pursue any further assessment. When formal evaluation is pursued, test batteries remain the most commonly used measures of aphasia. Although such batteries have the benefit of being comprehensive and precise, they are rather lengthy. Consequently, the importance of a thorough initial clinical review and informal evaluation is reiterated as they can serve to determine the necessity of a comprehensive assessment approach, which may include an aphasia battery, versus select domain screening. Such batteries commonly assess speech production, naming, auditory reception and comprehension, repetition, letter and word identification, writing, and spelling.

Examples of these comprehensive aphasia batteries include, but are not limited to, the BDAE-3<sup>rd</sup> Edition (BDAE-3) and the *Multilingual Aphasia Examination* (MAE).

Components of the BDAE-3, MAE, and similar batteries are commonly used in isolation to formally assess specific aspects of language functioning and aphasia, or in combination to assess language and aphasia comprehensively. For example, the Boston Naming Test is frequently used outside the confines of the BDAE-3 to assess patient’s capabilities in confrontational naming. Similarly, auditory comprehension can be formally assessed utilizing the Token Test, which is an individual component of the MAE. Although the patient’s capabilities in repetition can be assessed informally in an effective manner, tests such as the Sentence Repetition task of the MAE can also be used to further evaluate the patient’s capabilities within this skill. Similarly, fluency is best assessed through observation in general conversation or even in observing the patient’s narrative of a visual scene, such as the cookie theft scene of the BDAE-3. The latter has the benefit of allowing the clinician to observe speech content and discourse. Tasks such as the Controlled Oral Word Association Test (COWAT) may appear on the surface to be measures of speeded generative verbal fluency, but are more accurately discussed as part of the executive functions which is why batteries such as the Delis–Kaplan Executive Functioning System (DKEFS) include a verbal fluency subtest.

**TABLE 5.3 Proposed Battery of Tests for Aphasia Assessment**

TEST/MEASURE	SKILL ASSESSED
Boston Naming Test	Confrontational naming
MAE Token Test	Auditory comprehension
MAE sentence repetition	Repetition
FAS fluency	Fluency
Animal fluency	Fluency
Cookie theft picture	Speech content
Wechsler Test of Adult Reading	Reading
Spelling subtest-Woodcock–Johnson III-Tests of Achievement	Spelling

Written language capabilities can be effectively assessed through the combined use of reading and spelling tasks. Spelling subtests included as part of larger academic achievement batteries may serve the purpose of assessing the latter. This may include the spelling subtests of the Wide Range Achievement Test-3 or Woodcock–Johnson III-Tests of Achievement. Measures such as the Wechsler Test of Adult Reading offer a standardized assessment of patient’s capabilities in word reading, whereas reading directives both silently and aloud can aid in differential diagnosis. For example, if the patient is able to read the statement “close your eyes” silently and execute the command successfully, it suggests posterior preservation. If the patient is unable to read this same statement aloud, it could suggest involvement of systems/structures of a more anterior origin, such as disturbance of the arcuate fasciculus or Broca’s area. Because writing oftentimes mirrors oral output, the use of informal writing tasks, including writing spontaneous sentences, writing-to-dictation, and writing of longer passages, can be very useful in identifying additional written language deficits. [Table 5.3](#) includes an example of a battery for the assessment of aphasia that involves a compilation of tests/tasks.

By establishing an objective and standardized profile for patients across those domains assessed by these measures, a clinician may then consider the differentials previously discussed.

## **REHABILITATION APPROACHES FOR APHASIA**

Recovery from aphasia occurs over a period of time ranging from several months to many years ([Goodglass, 1993](#)). The brain’s ability to adapt to an injury and recover function is the basis for rehabilitation. In reality, considerable spontaneous recovery occurs in the first few months after the onset of aphasia, with ongoing improvement of a lesser degree continuing for several more months. Many factors influence the degree and speed of recovery from aphasia, such as the type and the size of the lesion and the person’s general health ([Drago & Foster, 2012](#)). In general, the greatest recovery occurs during the first 3 months, but improvement may continue over a prolonged period, especially in young patients and in patients with global aphasia. Consequently, the type of aphasia often changes during recovery such that global aphasia evolves into a Broca’s aphasia or Wernicke’s aphasia into conduction or anomic aphasia ([Drago & Foster, 2012](#)). Although spontaneous recovery of some degree of language functioning is common and may be substantial, clinicians agree that language therapy is helpful for most aphasic patients.



The ability to successfully communicate a message via spoken, written, or nonverbal modalities (or a combination of these) within day-to-day interactions is known as functional communication (Kelly et al., 2010). The primary goal of aphasia rehabilitation is to maximize functional communication skills (Schwartz & Fink, 2003). There is no universally accepted treatment that can be applied to every person with aphasia. Rather, there are different classical schools of thought as to how aphasia is rehabilitated. In addition, contemporary movements in the field have led to an emergence of new strategies in the rehabilitation of aphasia.

The Brain Injury-Interdisciplinary Special Interest Group (BI-ISIG) of the American Congress of Rehabilitation Medicine specified that rehabilitative interventions may have various approaches, including (a) reinforcing, strengthening, or reestablishing previously learned patterns of behavior; (b) establishing new patterns of cognitive activity through compensatory cognitive mechanisms for impaired neurologic systems; (c) establishing new patterns of activity through external compensatory mechanisms such as personal orthoses or environmental structuring and support; and (d) enabling persons to adapt to the cognitive disability. Even though it may not be possible to directly modify their cognitive impairment, rehabilitation interventions are designed to improve their overall level of functioning and quality of life (Cicerone et al., 2000). The current best practice recommends cognitive linguistic therapies during acute and postacute rehabilitation. Group communication treatment is also recommended as it can produce clinically meaningful improvements in language functioning, including improved functional communication beyond the effect of social contact alone. In addition, increased therapeutic intensity is encouraged for individuals with aphasia.

Although symptoms of aphasia may be resolved by appropriate speech therapy techniques, in some cases the disability remains permanent. Still, there is strong evidence that aphasia therapy does produce significant improvements in language function (Robey, 1994). Speech therapy, provided by speech-language pathologists, utilizes a number of techniques to facilitate language recovery and to help the patient compensate for lost functions.

### **Classical Schools of Aphasia Therapy**

There are three distinct schools of aphasia therapy that incorporate a number of specialized techniques. The majority of clinicians subscribe to *the traditional language-oriented school of aphasia therapy* which incorporates an array of

therapeutic and assessment techniques focused on the remediation of impairment and limitation in daily activity. Assessment incorporates traditional, standardized measures, many of which were previously discussed, for the purpose of identifying deficits to be targeted in rehabilitation. Using this approach, rehabilitation efforts are tailored to the patient. The approach taken in rehabilitation, in many ways, is dependent on how the clinician views aphasia. For example, [Shewan and Bandur \(1986\)](#) view aphasia as a loss or disruption of aspects of the language system. Consequently, their language-oriented therapy seeks to strengthen areas of impairment using training methods derived from behavioral learning theory. Research has shown improvements in language skills in patients receiving language-oriented therapy ([Doyle, Goldstein, Bourgeois, 1987](#); [Fink, Schwartz, Rochon, Myers, Socolof, 1995](#); [Poeck, Huber, Williams, 1989](#)), but this has not necessarily translated into improved quality of life.

In comparison, [Schuell, Jenkins, and Jimenez-Pabon \(1964\)](#) view aphasia as a consequence of a reduced access to, or efficiency in the language system. This is the premise of the stimulation approach to aphasia rehabilitation. This strategy uses intensive auditory or multimodality input to elicit production through a variety of means (e.g., repetition, phonemic cueing, and reading) and in a variety of contexts (linguistic and situational; [Schwartz & Fink, 2003](#)).

The *functional school of aphasia therapy* in many ways stems from the shortcomings of the traditional language-oriented therapy. Although the latter improves language skills, the traditional language-oriented therapy has been criticized for not translating into improved functional communication and social participation. The functional school of aphasia, also termed the pragmatic or social school of aphasia therapy, is focused on reducing participation restriction. Specifically, for those who adhere to this school of thought, rehabilitation is directed toward enhancing functional communication, both verbal and nonverbal ([Schwartz & Fink, 2003](#)). To accomplish this, patients are taught to utilize compensatory strategies that rely on their strengths. Promoting aphasics communication effectiveness (PACE) is rooted in the functional school of aphasia therapy ([Davis & Wilcox, 1985](#)). In this program, patients are encouraged to convey information through any available modality (e.g., speech, gestures, drawings, expressions, and mime) to convey what they mean. PACE is believed to work with any kind of aphasia.

The *cognitive neuropsychology school of aphasia therapy* focuses on remedying impaired functioning. Within this school of thought, rehabilitation is directed by theory-based assessments of the patient's language abilities

(Mitchum & Berndt, 1989; Seron & Deloche, 1989). Most closely matching neuropsychological practice, this theoretical approach aims to strengthen impaired processes through the incorporation of empirically based techniques. Therapies are focused on impaired skills instead of language on a broader level. For example, to address naming deficits, a clinician may use a combination of orthographic cueing and conversational context as demonstrated by Herbert, Best, Hickin, Howard, and Osborne (2003). This can lend to a modular approach to treatment in which multiple, focused therapies are used to treat multiple, specific impairments.

In addition to the traditional approaches to aphasia rehabilitation, new therapies like constraint-induced therapy and transcranial magnetic stimulation (TMS) have been developed. Although results are encouraging with some of these new strategies, in many instances the research behind some of these new therapies remains in its infancy.

### **Constraint-Induced Language Therapy (CILT)**

CILT is a form of rehabilitation in which patients with aphasia undergo an intense treatment program relying solely on verbal communication; no other means of communication (e.g., gesture or writing) are permitted (Carter, Connor, & Dromerick, 2010). In other words, CILT is an approach to therapy for language dysfunction based on the principles of use-dependent learning. Mechanisms are actually put into place so that no other form of communication is possible. CILT was first proposed by Pulvermuller *et al.* (2001), and is a modification of use-dependent learning applications in motor rehabilitation, referred to as constraint-induced movement therapy (Taub, Uswatte, & Pidikiti, 1999). The principles of constraint-induced approaches to therapy include: (a) constraint to the impaired modality; (b) restraint of the unimpaired modality (in this case alternative modes of communication), and (c) massed practice occurring in an enriched environment using behavioral shaping. In CILT, verbal communication is required and therapy typically lasts 2 to 3 hr/d, every day, for a 2-week period; research has shown that more intense approaches lead to maximized recovery (Bhogal, Teasell, & Speechley, 2003; Cherney, Patterson, Raymer, Frymark, & Schooling, 2008). Patients are forced to use speech while a therapist plays language games with two or three aphasic patients. The picture cards and the hands are hidden for other players to prevent visual input, and all communication, mainly questions and answers, have to be performed by spoken words and sentences. Extensive training provided 3 hr/d resulted in a significant

effect compared to standard training of 1 hr/ day (Pulvermuller et al., 2001). In their review of five different studies on CILT, Cherney et al. (2008) found that greater intensity of treatment produced greater change in language impairment both in the acute and in the chronic phases. However, it was unclear as to whether those gains translated into significantly improved functional communication in everyday settings.

In comparison to CILT, *visual action therapy* is based on gestural expression and represents a nonvocal approach to intervention using pictures, drawings, and gestures to indicate objects. Visual action therapy ultimately trains patients to produce symbolic gestures for visually absent stimuli and may be indicated for patients affected by global aphasia (Drago & Foster, 2012). Functional communication therapy is very much the same as visual action therapy as it encourages individuals to take advantage of extralinguistic communication capabilities.

### Melodic Intonation

It has long been observed that patients with nonfluent aphasia are oftentimes still able to sing (Yamadori, Osumi, Masuhara, & Okubo, 1977). Researchers have used this to suggest the right hemisphere has a compensatory function in speech recovery (Saur et al., 2006). These observations have served as the basis for melodic intonation therapy (MIT), which remains highly debated (Helm-Estabrooks, Nicholas, Morgan, 1989). Tamplin (2008) reported that both the intelligibility and the naturalness of speech can improve after vocal exercises and singing training in patients with nonfluent aphasia after stroke or trauma. Racette, Bard, and Peretz (2006) found that MIT was beneficial to natural speech when patients were singing along to vocal playback. Others have failed to support the benefit of singing in comparison to rhythmic speech or natural speech (Hebert, Racette, Gagnon, & Peretz, 2003).

MIT includes three main components: melodic intoning, rhythmic speech, and the use of common phrases. Intensive MIT (Gordon, Schon, Magne, Aste'sano, & Besson, 2010) is a specific MIT program in which words and melody are intertwined in singing (Gordon et al., 2010). The method includes three important components: melodic intonation, intense training 1½ hr/day for 5 d/week, and simultaneous tapping with the left hand to prime the sensorimotor and premotor cortices on the right side for articulation. Again, this method of intervention is ideal for nonfluent aphasics. Specifically, MIT delivered at high intensity to patients with chronic severe Broca's aphasia leads to remodeling of

the right arcuate fasciculus, demonstrating that plasticity can be induced in the contralateral homologous tract (Johansson, 2011; Schlaug, Marchina, & Norton, 2009). This coincides with findings, as previously noted, that suggest two routes of recovery, both of which can involve recruitment of homologous regions of the right hemisphere.

### **Computer-Based Rehabilitation**

One of the newest developments in recent years that has called into question traditional views of aphasia rehabilitation has been positive outcomes related to computer-based therapies (Katz, 2001; Petheram, 2004). For example, Manheim, Halper, and Cherney (2009) reported success with computer-based script-training in improving communication skills. Script-training is a computer-based approach in which patients practice communication skills utilizing extended home-based practice of personally relevant material through interaction with a computer avatar. Therapists set up scripts, facilitate successes, and assist clients in overcoming challenges during one-on-one meetings, but clients execute extended practice at home (Manheim et al., 2009). In their study, Manheim et al. (2009) found that script-training significantly reduced reported communication difficulties in 20 individuals with chronic aphasia that completed the program in 9 wk from home, while checking weekly with a speech therapist to monitor practice and progress.

*MultiCue*, developed by Doesborgh et al. (2004), is another computer-based program that has shown positive outcomes. The MultiCue program engages individuals with aphasia by allowing them to not only experience different cueing strategies for naming but also evaluate which are most useful to them. The benefit of this aspect of the program is that the treatment is thereby tailored to the individual. A similar system has been developed by Mortley, Wade, and Enderby (2004).

### **TMS and Transcranial Direct Current Stimulation (tDCS)**

TMS and tDCS each represent noninvasive brain stimulation methods that have been used and tested in stroke patients to aid in rehabilitative efforts. Both techniques work by modulating cortical excitability. High-frequency TMS increases cortical excitability to activate hypoactive networks, whereas low-frequency TMS increases inhibition of hyperactive networks. The problem with TMS is that when it is applied to the injured hemisphere it can cause seizures. In comparison, tDCS is relatively safer and is capable of modulating cortical

excitability in a polarity-specific manner using weak direct currents (Nitsche & Paulus, 2000). Like TMS, tDCS can be used to either increase cortical excitability (i.e., anodal) or decrease cortical excitability (i.e., cathodal; Nitsche & Paulus, 2000). Although most studies have focused on the utility of these techniques in the rehabilitation of hemiparesis, there is growing interest in their use in aphasia rehabilitation (Devlin & Watkins, 2007).

The basis for the use of TMS and tDCS in aphasia was first proposed based on findings from functional imaging which showed increased activity in contralesional undamaged brain areas. Studies have demonstrated that an imbalance of interhemispheric interactions stems from a combination of disinhibition of contralesional motor regions along with increased inhibition of ipsilesional motor regions (Duque et al., 2005; Shimizu et al., 2002). This imbalance is reported to interfere with neurological recovery at times. For example, imaging studies in patients showing an activation of the contralesional motor regions when the affected arm/ hand performs a motor task has been noted (Nair et al., 2007). Thus, the proposal behind TMS and tDCS is to reestablish a balance between the hemispheres by either downregulating activity in the contralesional hemisphere or increasing activity in the ipsilesional hemisphere. In terms of aphasia, research has shown that normalizing the interhemispheric excitability within a bihemispheric language network and, in turn, reactivating perilesional areas is beneficial in regaining language functions (Heiss & Thiel, 2006; Rosen et al., 2000; Saur et al., 2006). Repetitive TMS, for example, has been shown to be useful in poststroke rehabilitation of aphasia by facilitating improvements in language function via stimulating cortical plasticity (Barwood et al., 2011a, 2011b; Hamilton, Chrysikou, Coslett, 2011; Kakuda, Abo, Uruma, Kaito, Watanabe, 2010; Kakuda, Abo, Kaito, Watanabe, & Senoo, 2010; Szaflarski et al., 2011; Weiduschat et al., 2011). Research has demonstrated tDCS also improves language processing (Hesse et al., 2007; Iyer et al., 2005; Monti et al., 2008). Monti et al. (2008) have noted improvements in confrontational naming abilities in eight patients with nonfluent aphasia in response to tDCS treatment. Still, however, the use of these technologies remains experimental in practice at this time.

### **Pharmacotherapy in Aphasia Treatment**

Beyond the aforementioned therapeutic techniques, growing research has also suggested potential utility of pharmacotherapy. Studies of pharmacological intervention in rehabilitation have consistently shown that successful drug

therapy is always accompanied by behavioral practice. When it comes specifically to the rehabilitation of aphasia, results are mixed across different drug types, with benefits being primarily associated with amphetamines and acetylcholinesterase inhibitors. For example, [Walker-Batson \*et al.\* \(2001\)](#) studied the effects of D-amphetamine in aphasia rehabilitation. In this study, a greater percentage of patients receiving D-amphetamine in addition to speech and language therapy (SLT) improved on a test of communicative ability compared to those who received a placebo in addition to SLT. After 6 weeks of treatment, 83% of those that received treatment with D-amphetamine improved when compared to only 22% of those who received SLT alone. There was a nonsignificant trend for a persistent benefit at 6 months after dosing. These results are interpreted with caution; however, because the group that received the D-amphetamine actually received 21% more therapy time than did the placebo group. Similar results have been reported elsewhere (i.e., [Whiting, Chenery, Chalk, & Copland, 2007](#)), though this involved only two individuals.

The neurophysiological basis for this benefit potentially stems from increased left hemispheric activation given other study outcomes. [Sommer \*et al.\* \(2006\)](#) found that D-amphetamine administration during verb generation and semantic decision increased overall left hemispheric activation, and increased activation of both inferior frontal gyri and the left supramarginal gyrus, suggesting D-amphetamine can act to potentiate activity and plasticity of behaviorally activated networks, rather than in a nonspecific fashion.

Propranolol has also demonstrated a positive effect in the supplementation of aphasia rehabilitation ([Beverdorsdorf \*et al.\*, 2007](#)). In their study, [Beverdorsdorf \*et al.\* \(2007\)](#) gave single doses of propranolol or placebo to patient's with Broca's aphasia and then assessed language performance on the Boston Naming Test across three separate drug trials. Results revealed consistent small increases in naming performance on the Boston Naming Test which they related to a suppression of background activity.

Bromocriptine has also shown positive effects in aphasia treatment. In a single cohort study involving chronic nonfluent aphasics, [Bragoni \*et al.\* \(2001\)](#) found that bromocriptine, in combination with SLT for 60 days, improved performance on several language metrics over SLT alone. There was a trend for benefit to be sustained after a 60-day washout period. A similar study by [Seniów, Litwin, Litwin, LeŚniak, and Członkowska \(2009\)](#) found that L-dopa, delivered 30 minutes before SLT, everyday, over the course of 5 weeks, led to significant improvements on all metrics of the BDAE. However, these

improvements only reached significance in the areas of repetition and verbal fluency. It would seem that far more studies have failed to find positive outcomes with this group of agents (Ashtary, Janghorbani, Chitsaz, Reisi, & Bahrami, 2006; Gupta, Mlcoch, Scolaro, & Moritz, 1995; Sabe, Salvarezza, Garcia Cuerva, Leiguarda, & Starkstein, 1995)

Acetylcholinesterase inhibitors are most commonly thought of as agents used in Alzheimer's disease, but research has demonstrated some benefits in the treatment of aphasia. Berthier *et al.* (2006) reported significant improvements on the Western Aphasia Battery as well as other measures in a group of chronic aphasic patients after 16 weeks of donepezil. However, the results were not sustained out to 20 weeks. Berthier *et al.* (2009) also reported benefits of using memantine. These researchers found that 20 mg daily of memantine for 16 weeks without additional SLT corresponded with significant improvements on the Western Aphasia Battery, whereas the addition of CILT for 2 weeks led to additional improvements. Yet, once the patients were taken off memantine for 4 weeks, outcomes were significantly worse though still better than those in the placebo group.

## FINAL THOUGHTS

Aphasia represents a loss of language functions, in part or in total, after an acquired neurologic event. These deficits may present within the realms of expression, comprehension, repetition, reading, writing, or combinations of these skills. Variability is seen across individuals based on the nature, location, and extent of the insult. Although spontaneous recovery is typically a normal part of the recovery trajectory, rehabilitative efforts are often warranted ranging from various therapeutic programs to pharmacological interventions. Although medicinal, computer-based, and technologically advanced therapeutic options are gaining traction in the literature, individual SLT techniques remain the gold standard in rehabilitative endeavors. Interestingly, current treatment strategies employ hour-long periods of therapy which has been shown to be less efficacious than more intense approaches (i.e., 2–3-hour long treatment sessions every day). A meta-analysis of 30 speech-language therapy trials in patients with stroke found that intensive approaches (approximately 3-hour sessions) were more effective than conventional (1 hour) therapy approaches (Kelly *et al.*, 2010).

Although symptoms of aphasia may be resolved by appropriate speech therapy techniques, in some cases the disability remains permanent. Still, there is



strong evidence that aphasia therapy does produce significant improvements in language function. Moving forward, much more can be learned about ways of rehabilitating language impairments when they occur. The importance of this cannot be questioned as effective communication is essential in today's world. The impact that language impairment can have on an individual, their caregivers, and their family is extensive; therefore, proper management and treatment are necessary.

## REFERENCES

- Ardila, A. (2010). A review of conduction aphasia. *Current Neurology and Neuroscience Reports*, 10(6), 499–503.
- Ashtary, F., Janghorbani, M., Chitsaz, A., Reisi, M., & Bahrami, A. (2006). A randomized, double-blind trial of bromocriptine efficacy in nonfluent aphasia after stroke. *Neurology*, 66(6), 914–916.
- Barwood, C. H., Murdoch, B. E., Whelan, B. M., Lloyd, D., Riek, S., O'Sullivan, J. D., Coulthard, A., Wong, A. (2011a). Improved language performance subsequent to low-frequency rTMS in patients with chronic nonfluent aphasia poststroke. *Eur J Neurol*, 18(7):935–943.
- Barwood, C. H., Murdoch, B. E., Whelan, B. M., Lloyd, D., Riek, S., O'Sullivan, J., . . . Hall, G. (2011b). The effects of low frequency repetitive transcranial magnetic stimulation (rTMS) and sham condition rTMS on behavioural language in chronic nonfluent aphasia: Short term outcomes. *NeuroRehabilitation*, 28(2), 113–128.
- Beharelle, A. R., Dick, A. S., Josse, G., Solodkin, A., Huttenlocher, P. R., Levine, S. C., & Small, S. L. (2010). Left hemisphere regions are critical for language in the face of early left focal brain injury. *Brain: A Journal of Neurology*, 133(Pt 6), 1707–1716.
- Beeson, P. M., & Rapcsak, S. Z. (2006). The Aphasias. In P. J. Snyder, P. D. Nussbaum, & D. L. Robins (Eds.) *Clinical neuropsychology: A pocket handbook for assessment* (2nd Ed., pp. 436–162). Washington, DC: American Psychological Association Press.
- Benton, A. L., Hamsher, K., & Sivan, A. B. (1994). *Multilingual aphasia examination* (3rd ed.). Iowa City: AJA.
- Bernal, B., & Ardila, A. (2009). The role of the arcuate fasciculus in conduction aphasia. *Brain: A Journal of Neurology*, 132(Pt 9), 2309–2316.
- Berthier, M. L., Green, C., Higuera, C., Fernández, I., Hinojosa, J., & Martín, M. C. (2006). A randomized, placebo-controlled study of donepezil in poststroke aphasia. *Neurology*, 67(9), 1687–1689.
- Berthier, M. L., Green, C., Lara, J. P., Higuera, C., Barbancho, M. A., Dávila, G., & Pulvermüller, F. (2009). Memantine and constraint-induced aphasia therapy in chronic poststroke aphasia. *Annals of Neurology*, 65(5), 577–585.
- Beversdorf, D. Q., Sharma, U. K., Phillips, N. N., Notestine, M. A., Slivka, A. P., Friedman, N. M., . . . Hillier, A. (2007). Effect of propranolol on naming in chronic Broca's aphasia with anomia. *Neurocase*, 13(4), 256–259.
- Bhogal, S. K., Teasell, R., & Speechley, M. (2003). Intensity of aphasia therapy, impact on recovery. *Stroke*, 34(4), 987–993.
- Blair, J. R., & Spreen, O. (1989). Predicting premorbid IQ: A revision of the National Adult Reading Test. *The Clinical Neuropsychologist*, 3, 129–136.
- Blumenfeld, H. (2002). *Neuroanatomy through clinical cases* (2nd ed., p. 834). Sunderland, MA: Sinauer Associates, Inc.
- Bragoni, M., Altieri, M., Di Piero, V., Padovani, A., Mostardini, C., & Lenzi, G. L. (2000). Bromocriptine and speech therapy in nonfluent chronic aphasia after stroke. *Neurological Sciences*, 21(1), 19–22.

- Broussolle, E., Bakchine, S., Tommasi, M., Laurent, B., Bazin, B., Cinotti, L., . . . Chazot, G. (1996). Slowly progressive anarthria with late anterior opercular syndrome: A variant form of frontal cortical atrophy syndromes. *Journal of the Neurological Sciences*, *144*(1–2), 44–58.
- Carrow-Woolfolk, E. (1999). *Test for auditory comprehension of language* (3rd ed.). Austin, TX: Pro-Ed.
- Carter, A. R., Connor, L. T., & Dromerick, A. W. (2010). Rehabilitation after stroke: Current state of the science. *Current Neurology and Neuroscience Reports*, *10*(3), 158–166.
- Catani, M., Jones, D. K., & ffytche, D. H. (2005). Perisylvian language networks of the human brain. *Annals of Neurology*, *57*(1), 8–16.
- Cherney, L. R., Patterson, J. P., Raymer, A., Frymark, T., & Schooling, T. (2008). Evidence-based systematic review: Effects of intensity of treatment and constraint-induced language therapy for individuals with stroke-induced aphasia. *Journal of Speech, Language, and Hearing Research*, *51*(5), 1282–1299.
- Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., Bergquist, T. F., . . . Morse, P. A. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation*, *81*(12), 1596–1615.
- Croquelois, A., & Bogousslavsky, J. (2011). Stroke aphasia: 1,500 consecutive cases. *Cerebrovascular Diseases (Basel, Switzerland)*, *31*(4), 392–399.
- Damasio, A., & Damasio, H. (2000). Aphasia and the neural basis of language. In M. Mesulam (Ed.), *Principles of behavioral and cognitive neurology* (2nd ed., pp. 294–310). New York: Oxford University Press.
- Davis, G. A., & Wilcox, M. J. (1985). *Adult aphasia rehabilitation: Applied pragmatics*. San Diego, CA: College Hill.
- Delis, D., Kaplan, E., & Kramer, J. (2001). *Delis–Kaplan Executive Function Scale*. San Antonio: Psychological Corporation.
- Devlin, J. T., & Watkins, K. E. (2007). Stimulating language: Insights from TMS. *Brain: A Journal of Neurology*, *130*(Pt 3), 610–622.
- Dewarrat, G. M., Annoni, J. M., Fornari, E., Carota, A., Bogousslavsky, J., & Maeder, P. (2009). Acute aphasia after right hemisphere stroke. *Journal of Neurology*, *256*(9), 1461–1467.
- Doesborgh, S. J. C., van de Sandt-Koenderman, M. W. E., Dippel, D. W. J., van Harskamp, F., Koudstaal, P. J., & Visch-Brink, E. G. (2004). Cues on request: The efficacy of Multicue, a computer program for wordfinding therapy. *Aphasiology*, *18*, 213–222.
- Doyle, P. J., Goldstein, H., & Bourgeois, M. S. (1987). Experimental analysis of syntax training in Broca's aphasia: A generalization and social validation study. *The Journal of Speech and Hearing Disorders*, *52*(2), 143–155.
- Drago, V., & Foster, P. (2012) Aphasias. In C. A. Noggle, R. S. Dean, & A. M. Horton Jr. (Eds.), *The encyclopedia of neuropsychological disorders* (pp. 85–101). New York: Springer Publishing Company.
- Dunn, L. M., & Dunn, D. M. (2007). *Peabody picture vocabulary test* (4th ed.). Minneapolis, MN: Pearson.
- Duque, J., Mazzocchio, R., Dambrosia, J., Murase, N., Olivier, E., & Cohen, L. G. (2005). Kinematically specific interhemispheric inhibition operating in the process of generation of a voluntary movement. *Cerebral Cortex*, *15*(5), 588–593.
- Ferro, J. (2001). Neurobehavioral aspects of deep hemisphere stroke. In L. Bogousslavsky & L. Caplan (Eds.), *Stroke syndromes* (2nd ed., pp. 252–261). New York: Cambridge University Press.
- Festa, J., Lazar, R., & Marshall, R. (2008). Ischemic stroke and aphasic disorder. In J. Morgan & J. Ricker (Eds.), *Textbook of clinical neuropsychology* (pp. 363–383). New York: Taylor & Francis.
- Fink, R. B., Schwartz, M. F., Rochon, E., Myers, J. L., & Socolof, G. S. (1995). Syntax stimulation revisited: An analysis of generalization of treatment effects. *American Journal of Speech and Language Pathology*, *4*, 99–104.
- Flamand-Roze, C., Cauquil-Michon, C., Roze, E., Souillard-Scemama, R., Maintigneux, L., Ducreux, D., . . . Denier, C. (2011). Aphasia in border-zone infarcts has a specific initial pattern and good long-term prognosis. *European Journal of Neurology*, *18*(12), 1397–1401.

- Fridriksson, J., Bonilha, L., & Rorden, C. (2007). Severe Broca's aphasia without Broca's area damage. *Behavioural Neurology*, 18(4), 237–238.
- Fridriksson, J., Kjartansson, O., Morgan, P. S., Hjaltason, H., Magnusdottir, S., Bonilha, L., & Rorden, C. (2010). Impaired speech repetition and left parietal lobe damage. *The Journal of Neuroscience*, 30(33), 11057–11061.
- Gardner, M. F. (2000a). *Expressive one-word picture vocabulary test* (2000 ed.). Novato, CA: Academic Therapy Publications.
- Gardner, M. F. (2000b). *Receptive one-word picture vocabulary test* (2000 ed.). Novato, CA: Academic Therapy Publications.
- Goodglass, H. (1993). *Understanding aphasia*. San Diego, CA: Academic Press.
- Goodglass, H. & Kaplan, E. (2000). *Boston Naming Test*. Philadelphia, PA: Lippincott Williams & Wilkins.
- Goodglass, H., Kaplan, E., & Barresi, B. (2000) *The Boston Diagnostic Aphasia Examination (BDAE-3)* (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.
- Gordon, R. L., Schön, D., Magne, C., Astésano, C., & Besson, M. (2010). Words and melody are intertwined in perception of sung words: EEG and behavioral evidence. *PLoS One*, 5(3), e9889.
- Gorno-Tempini, M. L., Dronkers, N. F., Rankin, K. P., Ogar, J. M., Phengrasamy, L., Rosen, H. J., . . . Miller, B. L. (2004). Cognition and anatomy in three variants of primary progressive aphasia, *Ann Neurol*, 55(3), 335–346.
- Gupta, S. R., Mlcoch, A. G., Scolaro, C., & Moritz, T. (1995). Bromocriptine treatment of nonfluent aphasia. *Neurology*, 45(12), 2170–2173.
- Halstead, W. C., & Wepman, J. M. (1949). The Halstead–Wepman aphasia screening test. *The Journal of Speech Disorders*, 14(1), 9–15.
- Hamilton, R. H., Chrysikou, E. G., & Coslett, B. (2011). Mechanisms of aphasia recovery after stroke and the role of noninvasive brain stimulation. *Brain and Language*, 118(1–2), 40–50.
- He'bert, S., Racette, A., Gagnon, L., & Peretz, I. (2003). Revisiting the dissociation between singing and speaking in expressive aphasia. *Brain*, 126, 1838–1850.
- Helm-Estabrooks, N., Nicholas, M., & Morgan, A. (1989). *Melodic intonation therapy. Manual*. Austin, TX: Pro-Ed.
- Heiss, W. D., & Thiel, A. (2006). A proposed regional hierarchy in recovery of poststroke aphasia. *Brain and Language*, 98(1), 118–123.
- Herbert, R., Best, W., Hickin, J., Howard, D., & Osborne, F. (2003). Combining lexical and interactional approaches to therapy for word finding deficits in aphasia. *Aphasiology*, 17, 1163–1186.
- Hesse, S., Werner, C., Schonhardt, E. M., Bardeleben, A., Jenrich, W., & Kirker, S. G. (2007). Combined transcranial direct current stimulation and robot-assisted arm training in subacute stroke patients: A pilot study. *Restorative Neurology and Neuroscience*, 25(1), 9–15.
- Hodges, J. R., Salmon, D. P., & Butters, N. (1992). Semantic memory impairment in Alzheimer's disease: Failure of access or degraded knowledge? *Neuropsychologia*, 30(4), 301–314.
- Holland, A. L., Frattali, C. M., & Fromm, D. (1999). *Communicative abilities in daily living (CADL-2)* (2nd ed.). Negrang East, Australia: Pro-Ed.
- Iyer, M. B., Mattu, U., Grafman, J., Lomarev, M., Sato, S., & Wassermann, E. M. (2005). Safety and cognitive effect of frontal DC brain polarization in healthy individuals. *Neurology*, 64(5), 872–875.
- Johansson, B. B. (2011). Current trends in stroke rehabilitation. A review with focus on brain plasticity. *Acta Neurologica Scandinavica*, 123, 147–159.
- Kakuda, W., Abo, M., Uruma, G., Kaito, N., & Watanabe, M. (2010). Low-frequency rTMS with language therapy over a 3-month period for sensory-dominant aphasia: Case series of two poststroke Japanese patients. *Brain Injury*, 24(9), 1113–1117.
- Kakuda, W., Abo, M., Kaito, N., Watanabe, M., & Senoo, A. (2010). Functional MRI-based therapeutic rTMS strategy for aphasic stroke patients: A case series pilot study. *The International Journal of Neuroscience*, 120(1), 60–66.
- Katz, R. C. (2001). Computer applications in aphasia treatment. In: R. Chapey (Ed.), *Language intervention*

- strategies in aphasia and related communication disorders* (pp. 718–741). Philadelphia, PA: Lippincott, Williams & Wilkins.
- Kelly, H., Brady, M. C., & Enderby, P. (2010). Speech and language therapy for aphasia following stroke. *Cochrane Database of Systematic Reviews*, 5, CD000425.
- Kertesz, A. (1982). *Western Aphasia Battery*. San Antonio, TX: Psychological Corporation.
- Kertesz, A., Davidson, W., McCabe, P., Takagi, K., & Munoz, D. (2003). Primary progressive aphasia: Diagnosis, varieties, evolution. *Journal of the International Neuropsychological Society: JINS*, 9(5), 710–719.
- Kirshner, H., & Mark, V. (2009). Ischemic and intracerebral hemorrhagic stroke. In J. Festa & R. Lazar (Eds.), *Neurovascular neuropsychology* (pp. 19–48). New York: Springer.
- Kuljic-Obradovic, D. C. (2003). Subcortical aphasia: Three different language disorder syndromes? *European Journal of Neurology*, 10(4), 445–448.
- Kelly, H., Brady, M. C., & Enderby, P. (2010). Speech and language therapy for aphasia following stroke. *Cochrane Database of Systematic Reviews (Online)*, 5, CD000425.
- Lazar, R. M., & Mohr, J. P. (2011). Revisiting the contributions of Paul Broca to the study of aphasia. *Neuropsychology Review*, 21(3), 236–239.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment*. New York: Oxford University Press.
- Manheim, L. M., Halper, A. S., & Cherney, L. (2009). Patient-reported changes in communication after computer-based script training for aphasia. *Archives of Physical Medicine and Rehabilitation*, 90(4), 623–627.
- Mariën, P., Engelborghs, S., Vignolo, L. A., & De Deyn, P. P. (2001). The many faces of crossed aphasia in dextrals: Report of nine cases and review of the literature. *European Journal of Neurology*, 8(6), 643–658.
- Mesulam, M. M. (1982). Slowly progressive aphasia without generalized dementia. *Annals of Neurology*, 11(6), 592–598.
- Mitchum, C. C., & Berndt, R. S. (1989). Aphasia rehabilitation: An approach to diagnosis and treatment of disorders of language production. In M. G. Eisenbert (Ed.), *Advances in clinical rehabilitation* (pp. 183–198). New York: Springer-Verlag
- Mohr, J. P., Pessin, M. S., Finkelstein, S., Funkenstein, H. H., Duncan, G. W., & Davis, K. R. (1978). Broca aphasia: Pathologic and clinical. *Neurology*, 28(4), 311–324.
- Monti, A., Cogiamanian, F., Marceglia, S., Ferrucci, R., Mameli, F., Mrakic-Sposta, S., . . . Priori, A. (2008). Improved naming after transcranial direct current stimulation in aphasia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 79(4), 451–453.
- Mortley, J., Wade, J., & Enderby, P. (2004). Superhighway to promoting a client-therapist partnership? Using the Internet to deliver word-retrieval computer therapy, monitored remotely with minimal speech and language therapy input. *Aphasiology*, 18, 193–211.
- Moser, D. C., Papanicolaou, A. C., Swank, P., & Breier, J. I. (2011). Evidence for the solidarity of the expressive and receptive language systems: A retrospective study. *Journal of the International Neuropsychological Society: JINS*, 17(1), 62–68.
- Naeser, M. A., Alexander, M. P., Helm-Estabrooks, N., Levine, H. L., Laughlin, S. A., & Geschwind, N. (1982). Aphasia with predominantly subcortical lesion sites: Description of three capsular/putaminal aphasia syndromes. *Archives of Neurology*, 39(1), 2–14.
- Nair, D. G., Hutchinson, S., Fregni, F., Alexander, M., Pascual-Leone, A., & Schlaug, G. (2007). Imaging correlates of motor recovery from cerebral infarction and their physiological significance in well-recovered patients. *NeuroImage*, 34(1), 253–263.
- Nelson, H. E., & Willison, J. (1991). *The National Adult Reading Test (NART): Test manual* (2nd ed.). Windsor, UK: NFER Nelson.
- Nitsche, M. A., & Paulus, W. (2000). Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *The Journal of Physiology*, 527(Pt 3), 633–639.

- Ochfeld, E., Newhart, M., Molitoris, J., Leigh, R., Cloutman, L., Davis, C., . . . Hillis, A. E. (2010). Ischemia in Broca area is associated with Broca aphasia more reliably in acute than in chronic stroke. *Stroke*, *41*(2), 325–330.
- Pagoria, M. R. (2012). Language functioning in cancer. In C. A. Noggle & R. S. Dean (Eds.), *The neuropsychology of cancer and oncology* (pp. 293–302). New York: Springer.
- Petheram, B. (2004). Computers and aphasia: A means of delivery and a delivery of means. *Aphasiology*, *18*, 187–191.
- Poeck, K., Huber, W., & Willmes, K. (1989). Outcome of intensive language treatment in aphasia. *The Journal of Speech and Hearing Disorders*, *54*(3), 471–479.
- Pulvermüller, F., Neininger, B., Elbert, T., Mohr, B., Rockstroh, B., Koebbel, P., & Taub, E. (2001). Constraint-induced therapy of chronic aphasia after stroke. *Stroke*, *32*(7), 1621–1626.
- Racette, A., Bard, C., & Peretz, I. (2006). Making nonfluent aphasics speak: Sing along! *Brain*, *129*(Pt 10), 2571–2584.
- Robey, R. R. (1994). The efficacy of treatment for aphasic persons: A meta-analysis. *Brain and Language*, *47*(4), 582–608.
- Rosen, H. J., Petersen, S. E., Linenweber, M. R., Snyder, A. Z., White, D. A., Chapman, L., . . . Corbetta, M. D. (2000). Neural correlates of recovery from aphasia after damage to left inferior frontal cortex. *Neurology*, *55*(12), 1883–1894.
- Sabe, L., Salvarezza, F., García Cuerva, A., Leiguarda, R., & Starkstein, S. (1995). A randomized, double-blind, placebo-controlled study of bromocriptine in nonfluent aphasia. *Neurology*, *45*(12), 2272–2274.
- Saur, D., Lange, R., Baumgaertner, A., Schraknepper, V., Willmes, K., Rijntjes, M., & Weiller, C. (2006). Dynamics of language reorganization after stroke. *Brain*, *129*(Pt 6), 1371–1384.
- Schlaug, G., Marchina, S., & Norton, A. (2009). Evidence for plasticity in white-matter tracts of patients with chronic Broca's aphasia undergoing intense intonation-based speech therapy. *Annals of the New York Academy of Sciences*, *1169*, 385–394.
- Schuell, H., Jenkins, J. J., & Jimenez-Pabon, E. (1964). *Aphasia in adults: Diagnosis, prognosis, and treatment*. New York: Harper and Row.
- Schwartz, M. F., & Fink, R. B. (2003). Rehabilitation of aphasia. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neurology and neuropsychology* (pp. 179–193). New York: McGraw-Hill.
- Semel, E., Wiig, E. H., & Secord, W. A. (2003). *Clinical evaluation of language fundamentals* (4th ed.). San Antonio, TX: Psychological Corporation.
- Seniów, J., Litwin, M., Litwin, T., Lésniak, M., & Członkowska, A. (2009). New approach to the rehabilitation of poststroke focal cognitive syndrome: Effect of levodopa combined with speech and language therapy on functional recovery from aphasia. *Journal of the Neurological Sciences*, *283*(1–2), 214–218.
- Seron, X., & Deloche, G. (1989). Introduction. In X. Seron & G. Deloche (Eds.), *Cognitive approaches in neuropsychological rehabilitation* (pp. 383–398). Hillsdale, NJ: Erlbaum.
- Shewan, C., & Bandur, D. (1986). *Treatment of aphasia: A language-oriented approach*. Boston: College-Hill Press.
- Shimizu, T., Hosaki, A., Hino, T., Sato, M., Komori, T., Hirai, S., & Rossini, P. M. (2002). Motor cortical disinhibition in the unaffected hemisphere after unilateral cortical stroke. *Brain*, *125*(Pt 8), 1896–1907.
- Shoumaker, R. D., Ajax, E. T., & Schenkenberg, T. (1977). Pure word deafness. (Auditory verbal agnosia). *Diseases of the Nervous System*, *38*(4), 293–299.
- Smith, S., Faust, M., Beeman, M., Kennedy, L., & Perry, D. (1995). A property level analysis of lexical semantic representation in Alzheimer's disease. *Brain and Language*, *49*(3), 263–279.
- Sommer, I. E., Oranje, B., Ramsey, N. F., Klerk, F. A., Mandl, R. C., Westenberg, H. G., & Kahn, R. S. (2006). The influence of amphetamine on language activation: An fMRI study. *Psychopharmacology*, *183*(4), 387–393.
- Spreen, O., & Risser, A. (1991). Assessment of aphasia. In M. T. Sarno (Ed.), *Acquired aphasia* (2nd ed.). San Diego: Academic Press.

- Sullivan, J. R., & Riccio, C. A. (2010). Language functioning and deficits following pediatric traumatic brain injury. *Applied Neuropsychology*, 17(2), 93–98.
- Szaflarski, J. P., Ball, A., Grether, S., Al-Fwawress, F., Griffith, N. M., Neils-Strunjas, J., . . . Reichhardt, R. (2008). Constraint-induced aphasia therapy stimulates language recovery in patients with chronic aphasia after ischemic stroke. *Medical Science Monitor*, 14(5), CR243–CR250.
- Tamplin, J. (2008). A pilot study into the effect of vocal exercises and singing on dysarthric speech. *NeuroRehabilitation*, 23, 207–216.
- Taub, E., Uswatte, G., & Pidikiti, R. (1999). Constraint-Induced movement therapy: A new family of techniques with broad application to physical rehabilitation—a clinical review. *Journal of Rehabilitation Research and Development*, 36, 237–251.
- Walker-Batson, D., Curtis, S., Natarajan, R., Ford, J., Dronkers, N., Salmeron, E., Lai, J., & Unwin, D. H. (2001). A double-blind, placebo-controlled study of the use of amphetamine in the treatment of aphasia editorial comment. *Stroke*, 32, 2093–2098.
- Wallace, G., & Hammill, D. D. (2002). *Comprehensive receptive and expressive vocabulary test* (2nd ed.). Austin, TX: Pro-Ed.
- Warrington, E. K., & Shallice, T. (1984). Category-specific language impairment. *Brain*, 107, 829–854.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale-IV*. San Antonio: The Psychological Corporation.
- Wechsler, D. (2001). *Wechsler Test of Adult Reading*. San Antonio: The Psychological Corporation.
- Weiduschat, N., Thiel, A., Rubi-Fessen, I., Hartmann, A., Kessler, J., Merl, P., . . . Heiss, W. D. (2011). Effects of repetitive transcranial magnetic stimulation in aphasic stroke: A randomized controlled pilot study. *Stroke*, 42(2), 409–415.
- Whiting, E., Chenery, H. J., Chalk, J., & Copland, D. A. (2007). Dexamphetamine boosts naming treatment effects in chronic aphasia. *Journal of the International Neuropsychological Society*, 13, 972–979.
- Wilkinson, G. S. (1993). *WRAT-3: The Wide Range Achievement Test administration manual* (3rd ed.). Wilmington, DE: Wide Range.
- Williams, K. T. (2007). *Expressive Vocabulary Test* (2nd ed.). Minneapolis, MN: Pearson.
- Woodcock, R. W., McGrew, K. W., & Mather, N. (2001). *Woodcock-Johnson III Tests of Achievement*. Itasca, IL: Riverside.
- Yamadori, A., Osumi, Y., Masuhara, S., & Okubo, M. (1977). Preservation of singing in Broca's aphasia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 40, 221–224.
- Yang, Z., Zhao, X., Chun-Xue, W., Hong-Yan, C., & Zhang, Yu.-M. (2008). Neuroanatomic correlation of the poststroke aphasias studied with imaging. *Neurological Research*, 30, 356–360.

## Rehabilitation of Memory Deficits

*Renée R. Lajiness-O'Neill, Laszlo A. Erdodi, Alfred Mansour, and Amy Olszewski*

The understanding that experience-driven changes have the potential to alter the human brain throughout the life span has been called a *nascent revolution* in neuroscience (Holloway, 2003). Indeed, the adult brain is capable of substantial change as a result of practice and experience—including the damaged brain. Evidence suggests that plastic changes occur in the cerebral cortex to compensate for loss of function in damaged areas, resulting from injury and illness. However, these changes may not always be beneficial. Moreover, change or recovery of function is not likely in the absence of specific intervention. Memory impairment is one of the most common neuropsychological sequelae observed in both pediatric and adult clinical populations following brain injury and the onset of degenerative disorders. A myriad of cognitive rehabilitation strategies to treat memory impairment informed by research and practice have been developed over the past quarter century since the seminal book, *Rehabilitation of Memory*, was published by Wilson in 1987. Current methods emphasize three general paths for treatment: (a) strategies that facilitate residual explicit memory with support provided at both encoding and retrieval, (b) methods that attempt to exploit the intact implicit memory system, and (c) interventions that enhance performance of daily functions through the use of external memory aids (De Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001). Despite the method of intervention, the goal of cognitive remediation is to improve functional abilities and promote increased independence.

This chapter will address methods of memory rehabilitation based on an

integration of current theory and practice. First, a brief introduction of the prominent theories of memory, including essential theoretical constructs, will be reviewed. This will be followed by a general discussion of frequently encountered disorders that result in memory impairment, and commonly employed clinical methods of assessment of memory functioning. We did not intend to expand on all or even most acquired disorders or diseases that result in compromised memory functioning, as this list is extensive. Instead, those disorders with the highest prevalence rates within the population are addressed, with particular emphasis on brain injury and dementias. Finally, a comprehensive review of memory rehabilitation interventions will be explored, addressing adult and pediatric populations with both nondegenerative (e.g., traumatic brain injury [TBI], stroke) and degenerative (e.g., dementia) disorders.

## MEMORY THEORY AND CONSTRUCTS

Memory as an ability can be defined as the process of recording experiences and adjusting behavior based on that stored information (McGaugh, 1966). As a mental product, memory is a lasting representation of the perceived reality reflected in cognitive, affective, and behavioral changes (Moscovitch et al., 2007). From a biological perspective, memory is a relatively enduring change in the individual's neural architecture and cell physiology as a result of experience (Alberini, 2005; Dudai, 2000). Functionally, memory is the formidable capacity of the brain to recreate the awareness of a past event in the absence of an external stimulus that led to the original experience (Sheslow & Adams, 2003). Also, memory preserves the individual's identity by building and maintaining mental connections between the past and present (Reynolds & Bigler, 1994). Memory allows for both a comprehensive perception of reality at any given time, and the ability to preserve a personal chronology and mental inventory of past events. As such, it is a building block for higher order cognitive and motor skills, as well as a prerequisite for normal daily functioning. Memory helps organize the sensory input and enables experience to accumulate, thus increasing the individual's problem-solving efficiency over time.

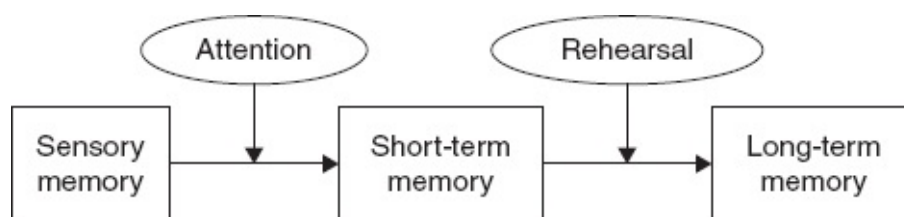
Traditionally, memory is conceptualized as a direct one-to-one correspondence between the initial learning and later recall: stimuli are captured, stored, and retrieved in the same format. This positivistic view is questioned by a century of memory research, which made it clear that the original stimulus, its immediate perception, and the retrieval of its longterm mental representation are far from being equivalent. Accumulating disconfirming evidence led to the



formulation of the process view, which emphasizes the dynamic nature of memory, recognizing that remembering is a reconstructive mechanism dependent on a cascade of molecular events (Frankland et al., 2001; Izquierdo et al., 2002; Lisman & Morris, 2001), and thus prone to several sources of distortion (Loftus, 2005; Loftus & Davis, 2006; Moscovitch et al., 2007).

### Time-Dependent and Stage-of-Processing Models of Memory

As it gradually became apparent that the old conceptualization of memory as a unitary system is unnecessarily abstract and detached from the accumulating neurobiological data (Squire, 2004), new models were developed to describe the increasing awareness of the complexity of memory formation. Although the idea of multiple memory systems had been introduced previously (Broadbent, 1958; Waugh & Norman, 1965), Atkinson and Shiffrin (1968) were the ones who formalized the theory of short-term memory (STM) in its most systematic version. As Figure 6.1 illustrates, prior to entering the STM, information is first captured in the sensory register, a transient memory system with limited capacity (can last a maximum of 5–10 seconds). Sensory input is constantly and rapidly deleted to make room for new incoming sensations. The visual–sensory system processes the raw material for iconic memories, whereas the antecedents of echoic memory are housed in the auditory–sensory system. According to their theory, only information consciously attended to enters the STM, an intermediate storage system, where it can be kept longer through rehearsal. Also referred to as memory span, the STM has a capacity limited to about seven elements. Through several repetitions, the information stored in STM is encoded into longterm memory (LTM), a relatively permanent storage system.



**FIGURE 6.1** The short-term memory model of Atkinson and Shiffrin (1968).

Although few researchers accept the STM model as it was originally presented, it has been a powerful influence on subsequent theories of memory functioning (Anderson, 2005). The three-component model of working memory (WM) introduced in 1974 by Baddeley and Hitch builds upon the initial

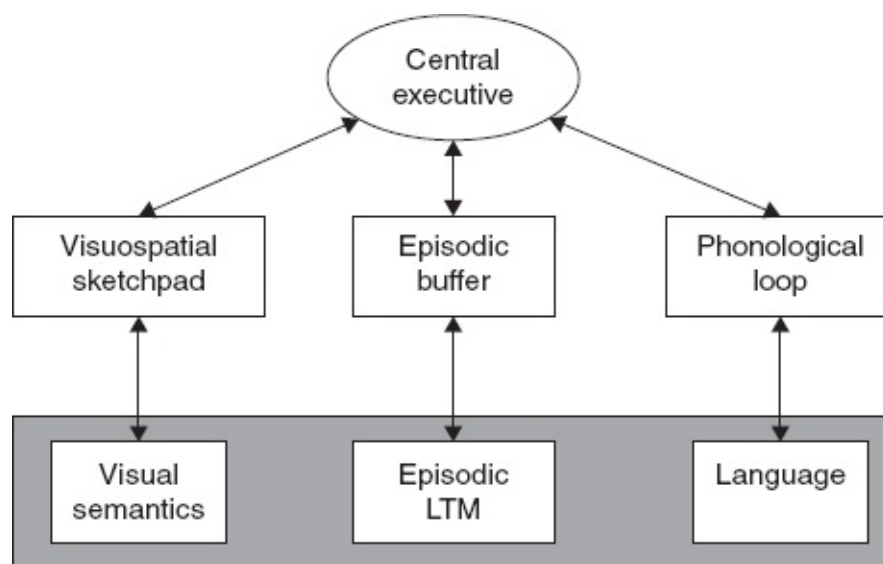
conceptualization of the STM systems, but emphasizes a different function: a temporary storage system that enables the simultaneous access and process of information (Baddeley, 1992). It preserves and accentuates the distinction between a visual and auditory input and the importance of attention as a mediating process, but does not require rehearsal as necessary for information to reach LTM. Later, a new component was introduced to improve the explanatory power of the model. As shown in Figure 6.2, the episodic buffer is conceptualized as an interface between the transient (unshaded boxes) and LTM (shaded box) systems (Baddeley, 2000). Using the high positive correlation among the previously discussed measures of memory as an argument, Engle (2002) pointed out that at a practical level, WM is difficult to distinguish from executive attention and is perhaps isomorphic to fluid intelligence.

Memory is a time-dependent construct. The evolution of memory over time can be examined at two different levels of analysis: the stages the memory traces goes through, from initial encoding to repeated retrievals, and the process through which memory as a cognitive ability develops from infancy throughout late adulthood. Each approach reveals important characteristics of memory performance that has direct clinical implications.

The process of memory formation has a clear temporal dimension: encoding takes time. This aspect of memory was first systematically studied by Müller and Pilzecker (1900). They introduced the term “consolidation” to label the phenomenon through which newly learned material, initially vulnerable to disruption, is gradually transferred into permanent storage. Some of their methodological innovations (i.e., fixing the number of learning trials and using percentage retention as a dependent measure) are standard psychometric practices today (Lechner, Squire, & Byrne, 1999). Müller and Pilzecker were the first to observe retroactive interference: they noticed that the temporal processing of successive trials of a list-learning task was reliably disrupted when a distractor (a different list to memorize) was introduced. This is the first known experiment to demonstrate that silent information processing occurred between the first exposure to a stimulus and a later recall.

The first attempt to coalesce the consolidation phenomena into a coherent theory was made by Hebb (1949). His dual-trace theory suggested that the perseveration of the initially fragile memory trace over time eventually translates into lasting synaptic changes, which he considered as the neural representation of learning. Hebb’s structural theory of learning conceptualizes memory as a time-dependent process—a notion that remains the core assumption of modern

consolidation hypotheses. In his seminal paper, [McGaugh \(1966\)](#) reviewed the cumulative evidence for time-dependent processes in memory, and argued that if the existing body of research continued to expand in the same directions, [Lashley's \(1930\)](#) prophecy that understanding the neuroplasticity underlying learning is beyond human reach could eventually be falsified. His review culminates in a few important conclusions: (a) the trace of an experience is not fixed immediately after the initial exposure—consolidation takes time, (b) consolidation is susceptible to both facilitating and impairing influences, and (c) there are several distinct memory processes.



**FIGURE 6.2** [Baddeley's \(2000\)](#) four-component model of working memory.

Recent discoveries in molecular biology support the hypothesis of time-dependent changes in cell physiology that translate into functional alterations in the neuron. [Nayak and Sikdar \(2007\)](#) described a form of “molecular memory”: after sustained depolarization, the voltage-gated sodium channels exhibit a *novel* pattern of activation that ultimately influences the excitability of the neuron. Although it is not yet clear to what extent this phenomenon depends on external input (i.e., learning), it offers a plausible mechanism through which experience is encoded and stored at a very basic level. In fact, [Gilboa, Chen, and Brenner \(2005\)](#) showed through mathematical modeling of single neuron activity that ion channel functioning depends on recent activation history. This interdependence of function and usage in molecular neurophysiology provides indirect evidence that experience modulates an organism’s nervous system at the lowest observable levels. Similarly, [Eguia, Rabinovich, and Abarbanel \(2000\)](#) proposed

a model of neural connectivity in which multiple signal transmission represents an opportunity for self-correction: neurons give priority to incoming signals that show consistency over time. This preferential processing of electrical impulses results in improving the overall signal-to-noise ratio. Such molecular processes serve as plausible physiological mechanisms underlying the phenomenon of consolidation.

Consolidation has many possible definitions, each one having a slightly different theoretical emphasis. Although most theorists agree on the basic mechanisms, many of its details are topics for ongoing speculations (Meeter & Murre, 2004). Consolidation has been described as a gradual reorganization of the stored information, with an implied shift in the locus of storage from the medial-temporal structures to the neocortex (Medina et al., 2008; Murray & Bussey, 2001; Squire & Alvarez, 1995; Squire & Bayley, 2007; Tse et al., 2007), as well as a progressive, domain-specific stabilization of memory traces (Moscovitch et al., 2006, 2007; Nadel et al., 2000; Sutherland et al., 2006). As a synthesis of nuances articulated earlier, the definition could be condensed into a single notion: consolidation is a time-dependent reorganization of the brain structures underlying the ability to store a specific type of information (McGaugh, 2000). This implies that new memories are fragile, vulnerable to forgetting, and are gradually transformed into a more permanent state that is resistant to decay (Frankland & Bontempi, 2005). Consolidation theory continues to be vague about the specific time line that new memories follow during their journey from the hippocampus to the neocortex. McGaugh (2000) introduced a three-stage model of consolidation, distinguishing among (a) short-term (seconds to hours), (b) long-term (hours to months), and (c) long-lasting (months to lifetime) memories. This classification system is sound and grounded in empirical research, yet not very specific. Nadel and Moscovitch (1997) criticized the extremely long time frame (as long as 25 years) classical consolidation theory uses, stating that it was hard to see how a cognitive strategy that takes two thirds of the lifetime of the medieval man would serve any meaningful adaptive function. Indeed, since its inception, consolidation theory has been confronted by three distinct yet overlapping competing explanations of the process of memory formation: connectionist models, multiple trace theory (MTT), and semantization. The core assumption of MTT introduced by Moscovitch and Nadel (1997) is that the hippocampal complex continues to play an instrumental role in the retrieval of episodic and autobiographical memories, even after they have been transferred to the neocortical memory systems

(Moscovitch et al., 2005). They hypothesize that memories are replicated over time in the hippocampus. Older memories have more copies than newer ones; therefore, they are easier to retrieve. Phenomenologically, this manifests itself as the Ribot gradient. Although an explanation of these models is beyond the scope of this chapter, it will suffice to say that our understanding of the processes involved in memory formation and memory loss continues to be an active area of research within cognitive neuroscience. For a review of these competing and complimentary models, see McClelland, McNaughton, and O'Reilly (1995) (Connectionist models); Moscovitch and Nadel (1997) (MTT); Frankland and Bontempi (2005); and Squire and Bayley (2005) (Semantization). Central to consolidation theory is the hypothesized underlying neural mechanism through which memory traces are gradually transferred from the hippocampus into the neocortex, to the point where they can be retrieved without hippocampal mediation (Alvarez & Squire, 1994).

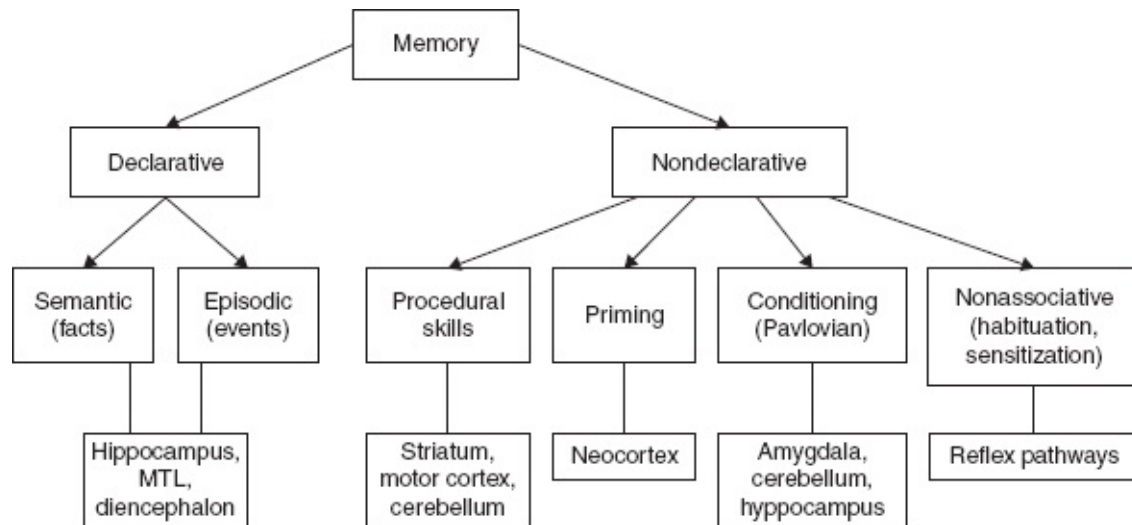
### Modality-Specific Memory, Systems, and Subtypes

Although the preceding models of memory offer intuitive conceptualizations of information storage and processing, and are consistent with clinical and experimental data, they are biased toward human cognition and are not explicit about the neural substrates underlying their central constructs. In contrast, Squire's (1987) taxonomy includes LTM systems that apply to all mammalian species, and specifies the locus of each form of memory in the brain (Figure 6.3). By differentiating among salient subtypes of memory and explicitly linking cognitive function to neuroanatomy, his model allows for more specific predictions in prospective experimental studies of memory.

In general, in this model, LTMs can be distinguished by the types of information to be remembered. Declarative (or explicit) memory can be further divided into semantic and episodic memory. Nondeclarative (or implicit) memory can be further subdivided into procedural memory, priming, classical conditioning, and nonassociative learning. LTMs can be further understood through their modal-specific nature. That is, we learn information through verbal and visual processing. Left and right hemispheric specialization for verbal and visual information, respectively, was evident in early studies following unilateral temporal lobectomies (Milner, 1965, 1968).

*Declarative memory* is knowledge to which we have conscious access and includes: (a) *semantic memory*, that is, memory for general knowledge, such as world, object, and language knowledge and (b) *episodic memory*, that is,

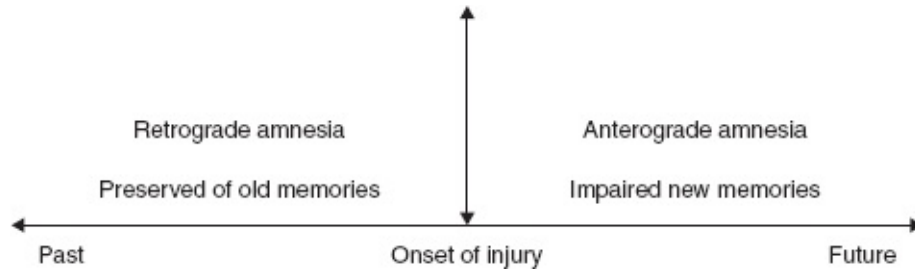
memories that we have, and information that we can access about our lives, such as what one had for breakfast. The distinction between semantic and episodic memory was first presented by [Tulving \(1972\)](#), and the latter is particularly vulnerable to disruption following the onset of dementia. *Explicit memory* is similar to episodic memory and merely means that one has conscious recollection of a specific incident or episode from the past.



**FIGURE 6.3** [Squire's \(1987\)](#) model of the mammalian memory systems.

*Nondeclarative memory*, on the other hand, does not require conscious awareness during the learning process. Likewise, the term *implicit memory* is often used synonymously with nondeclarative memory. Nondeclarative memory is observed when previous experience facilitates performance on a task for which intentional recollection of the experience is not required. Memory rehabilitation efforts described in the text that follows have attempted to capitalize on the relative preservation of implicit memory in patients with amnesia through the use of strategies such as errorless learning (EL) and the method of vanishing cues (VC) ([Glisky & Schacter, 1988](#); [Wilson, 1994](#)). *Procedural memory* is an example of nondeclarative or implicit memory that allows one to learn motor skills, such as riding a bicycle or cognitive tasks like how to read. Another form of implicit memory that is relevant for memory rehabilitation is observed through priming. Within the perceptual representation system (PRS), the form and structure of objects can be primed. *Priming* occurs when there is a change in response to a stimulus or in one's ability to recognize a stimulus due to prior exposure to that stimulus. The final two forms of

nondeclarative memory include *classical conditioning* (i.e., Pavlovian conditioning) and *nonassociative learning*. The later consists of forms of learning, such as habituation and sensitization.



**FIGURE 6.4** Illustration of anterograde and retrograde amnesia.

A final distinction between subtypes of memory that is relevant in a chapter on memory rehabilitation is that between retrospective and prospective memory. *Retrospective memory* is merely memory for past events, incidents, and experiences. Following injury or illness, a loss of memory of past events before the onset of the illness or injury is referred to as retrograde amnesia (RA). Typically, there is a temporal gradient of loss in amnesia that results in memories closest to the injury or onset more vulnerable to disruption, whereas more remote memories are relatively preserved. In contrast, *prospective memory* is the ability to recall a future event and has been defined as the realization of delayed intentions (Ellis, 1996). Prospective memory is one of the most commonly reported everyday memory problems (Baddeley, 2004). This form of memory will be discussed in further depth when discussing rehabilitation methods. To parallel RA, anterograde amnesia refers to the loss of ability to form new memories. The relationship between retrograde and anterograde amnesia is illustrated in Figure 6.4.

## ABNORMALITIES OF MEMORY

### Acquired Injury and Degenerative Disorders

Given the fragility of memory, it is not surprising that impairments in memory and amnesia are common neuropsychological sequelae observed in both the pediatric and adult clinical populations. They are the consequences of cellular or neurological damage or dysregulation and may be more transient, as in the case of mild traumatic injury (mTBI) in adults, or persistent and degenerative, as seen with Alzheimer's disease (AD) and other dementias. Despite the prevalence of

memory disorders, they are often difficult to rehabilitate due to individual and developmental differences, the variable severity and type of the underlying lesions, and their chronicity. Consistent with theoretical conceptualizations, deficits are not monolithic, and may be predominantly episodic, semantic, visuospatial, verbal, or contextual, and may involve short-, intermediate-, or longterm processes. Further complicating the clinical presentation, memory disorders rarely occur in isolation and are usually associated with losses in other neurocognitive domains as difficulties in attention, concentration, executive functioning, and in learning new information.

TBI defined as trauma caused by a blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain, continues to be a leading cause of death (Graham, McIntosh, Maxwell, & Nicoll, 2000; Jankowitz, & Adelson, 2006). Each year an estimated 1.4 million Americans experience a TBI and 80,000 to 90,000 suffer longterm disabilities (Rutland-Brown et al., 2006). In addition to attention and concentration problems and difficulties in executive functioning, individuals with TBI often exhibit persistent memory deficits. Although the TBI population has no characteristic or unique memory profile, performance seems to be mediated by factors such as injury severity and processing speed (Mottram & Donders, 2006). Clearly, overall outcome is mediated by a host of injury-related (e.g., severity, time postinjury, locus, length of unconsciousness) and demographic variables (e.g., age, premorbid abilities, family support).

Structurally, the human memory is particularly vulnerable to the effects of TBI due to the boney prominences of the anterior and middle cranial fossa that encapsulate these cortical lobes. The preferential impact on the frontal and temporal lobes likely contributes to the typical constellation of neurobehavioral disruption coupled with memory and executive impairment following moderate-to-severe injury (Bigler, 1999). In addition, the temporal and frontal lobes of pediatric brains are more susceptible to damage, regardless of the original locus of injury (Bigler, 1999; Bigler & Dodrill, 1997). Another plausible contributing mechanism to this pattern of deficits is the impact that acceleration/deceleration injury is likely to have on the anterior commissure; the critical pathway of communication between affective orbitofrontal and limbic regions of the temporal lobe, such as the amygdala. Moderate and severe TBI has consistently been reported to cause diffuse and focal damage with generalized atrophic changes, resulting in a loss in overall brain volume (Levin et al., 2008) and diffuse white matter atrophy (Gale, Johnson, Bigler, & Blatter, 1995; Levin et



al., 2008). Some of the most consistently reported morphological alterations and pathological findings noted during the chronic phase of injury on neuroimaging include general ventricular dilation, with enhancement of the temporal horns and third ventricle; atrophic changes specifically noted in the anterior temporal and frontal regions; enlargement of the interhemispheric and Sylvian fissures, as well as other cortical sulci; and thinning of the corpus callosum (Bigler, 1999).

Modality-specific memory deficits may be evident given the primary locus of disruption. That is, right-hemispheric injuries result in a greater degree of deficits in visuospatial memory, whereas left-hemispheric injuries or infarcts lead to a more substantial compromise in verbal memory. This impairment in processing verbal information, particularly in children, may in turn disrupt the normative rate of knowledge acquisition, resulting in a compounding deficit later, especially in academic achievement. Prospective memory impairment results in a significant compromise to everyday functioning in those with TBI. Deficits are linearly related to injury parameters and especially pronounced in those with severe TBI. Increases in cognitive demands result in further decline in prospective memory performance, which is consistent with the high prevalence of frontal lobe lesions. Age has also been identified as a mediating variable: older children tend to perform better, but they are also more vulnerable to the adverse effect of the increase in cognitive load (Ward, Shum, McKinlay, Baker, & Wallace, 2007).

Procedural memory, on the other hand, is less vulnerable to injury and relatively well preserved after TBI. In fact, children with TBI often show a normative learning curve, retention, and recall on such tasks, even when their performance is impaired on explicit memory tests (Ward, Shum, Wallace, & Boon, 2002). These findings may, in part, be due to the fact that subcortical structures, which are less prone to injury during a closed head injury, may mediate implicit memory that is critical for such tasks. One of the implications of these findings is the potential utility of activity-based learning in this population, where the physical aspects of the acquisition process can later serve as cues for retrieval.

Unlike the transient neurocognitive losses often seen with mild adult TBI, neurodegenerative disorders such as AD or the dementias associated with Pick's, Parkinson's, and Huntington's diseases, rarely improve without intervention. AD that accounts for two thirds of the dementias seen clinically is characterized by deficits in memory and language, abstract reasoning, executive functioning, attention, and visuospatial abilities (Salmon & Bondi, 1999). The neurocognitive

losses are associated with cortical atrophy, synaptic and dendritic spine loss, and the accumulation of amyloid plaques and neurofibrillary tangles in the medial temporal lobe structures (entorhinal cortex, hippocampus) and association cortices of the frontal, temporal, and parietal lobes (Braak & Braak, 1991).

Although the neurocognitive deficits associated with AD cut across cognitive domains, several lines of evidence suggest that the earliest pathological changes seen with individuals suffering from this disorder occur in medial temporal and parietal lobe structures that mediate and are critical in maintaining episodic memory (Cabeza et al., 2008; Gold, 2008; Salmon, 2000). As noted, episodic memory refers to the ability of an individual to consciously retrieve an experienced event or perceived item. Characteristic disruptions in episodic memory, as seen with delayed recall tasks, are the most clinically relevant and among the earliest signs and symptoms of AD. In fact, investigators have argued that deficits in episodic memory may be the best behavioral predictor of imminent development of Alzheimer's dementia and essential in differentiating this disorder from the normal age-associated changes in cognitive functioning.

As the neuropathology extends beyond the structures of the medial temporal cortex to the temporal, frontal, and parietal associative cortices, individuals with AD typically experience higher order cognitive impairments. As the disorder progresses, in addition to impairment in episodic memory, individuals with AD develop semantic memory deficits that are expressed as a loss of general knowledge and impairment in language. These neurocognitive deficits are associated with pathology in the temporal (anterior and inferolateral) and frontal lobes, and characterized by a decline in neuronal dendritic spines in these regions (Kasai et al., 2010; Starr et al., 2005). Deficits in executive functioning, problem solving, and attention follow and are likely mediated by dysregulation in the associative frontal and temporal cortical areas. Visuospatial abilities also decline with AD, but these deficits may not be as salient as the other cognitive changes seen at the earlier stages of the disorder (Butters et al., 1984).

There is a growing body of evidence that suggests that Alzheimer's dementia, and perhaps all the cortical dementias, results in a loss of effective connectivity and integration of information across multiple cortical areas (Dickerson & Sperling, 2008; Sperling et al., 2010). Anatomically, early studies demonstrated that the neurofibrillary tangles associated with AD tend to localize in specific cortical layers (Layers III and V) or in the pyramidal cells of the

hippocampus, disrupting the synaptic connectivity between these regions and other associative cortical areas (Hof & Morrison, 1999; Hyman et al., 1984). Consistent with these neuropathological findings, results from neurophysiological, neuroimaging, and behavioral studies all suggest that a functional disconnectivity of cortical areas and impairment in information integration is a central and defining feature of Alzheimer's dementia (Dickerson & Sperling, 2009; He et al., 2010).

Although AD is the leading cause of dementia, several other neurological disorders, such as Huntington's and Parkinson's diseases, dementia with Lewy bodies (DLBs), frontotemporal dementia (FTD), and vascular dementia, are known to produce neurocognitive deficits with significant memory impairment. Although there may be overlapping cognitive deficits seen across these dementias, their clinical presentation differs depending on the brain regions, neuronal circuits, and cellular elements that are affected (Vitali et al., 2008). Huntington's and Parkinson's diseases, primary disorders of the basal ganglia, in their later stages, results in dysregulation of striatocortical connections in the brain and are characterized by slowness of thought, impaired attention and executive functioning, learning difficulties, visuospatial and constructional deficits, and personality changes (McHugh & Folstein, 1975). Similarly, although there is overlap in the clinical presentation of DLBs and AD, individuals with DLB have greater visuospatial, attention, and executive functioning impairment, and those with AD and Alzheimer patients tend to have more prominent memory impairment (Stavitsky et al., 2006). FTD is characterized by the deterioration of personality and cognitive functioning associated with frontal and temporal lobe atrophy. FTD typically begins with personality and behavioral changes, followed by cognitive losses in attention, executive functioning, and language. Unlike Alzheimer's dementia, there is a relative initial sparing of memory and visuospatial abilities, and parietal lobe function with FTD (Boxer & Miller, 2005; Mackenzie & Rademakers, 2007; Rabinovici & Miller, 2010). Finally, investigators have differentiated the deficits seen with vascular dementia from Alzheimer's dementia, particularly those with vascular lesions in subcortical regions, based on greater impairment in attention and executive function seen with subcortical vascular dementia in contrast to the deficits in episodic memory that are more prominent in AD (Desmond, 2004).

In addition to the dementias, other neurological disorders can produce persistent disruption in memory processes. Epilepsy, particularly childhood onset, has long been known to produce neurocognitive deficits, because as seen

with childhood TBI, the disorder disrupts brain maturation and often interferes with the acquisition of academic skills. Memory impairments have been most extensively studied in focal epileptic lesions in the temporal lobe and are correlated with the degree of hippocampal atrophy seen with brain MRI and clinical variables, such as age of onset of epilepsy, seizure frequency, and lifetime number of seizures (Dodrill, 1992; Oyegbile et al., 2004). Hermann *et al.* (2002), for example, compared patients with temporal lobe epilepsy (TLE) to healthy controls, using MRI volumetric imaging. In addition to a reduced hippocampal volume, childhood onset (<14 years), but not adult onset, TLE is associated with significantly reduced total brain tissue that was generalized in nature and extended into extratemporal regions. In addition to these structural differences, there was significant reduction in intellectual functioning and memory with childhood onset TLE patients. Statistical analyses indicate that increasing duration of epilepsy in childhood onset patients is associated with declining performance across both intellectual and memory measures, suggestive of progressive cognitive effects. The deficits seen with epilepsy also depend on the specific locus of the seizures and its lateralization, as childhood onset frontal lobe epilepsy produces symptoms (deficits of planning, attention, and motor dexterity) that are more akin to those found in frontal lobe lesions (Lassonde et al., 2000). Similarly, left-hemispheric TLE causes more pronounced deficits in verbal memory, and, less consistently, right-hemispheric TLE affects nonverbal memory (Gleissner et al., 1998).

## ASSESSMENT OF MEMORY

### What Is Currently Assessed in Memory Rehabilitation?

Most standardized memory assessment batteries examine declarative memory; the intentional, conscious recollection of previous experiences. Tasks used to measure declarative memory often involve showing the patient a series of pictures or reading him or her list of words, in an effort to have the patient recall or recognize the previously presented stimuli at a later time (Spreeen & Strauss, 1998). These are highly common, and make up the majority of tasks in the memory batteries described below.

Although several measures are used to assess explicit memory ability, prospective memory is especially important for functional living skills, such as medication adherence among older individuals with AD, and the ability for children with TBI to remember to turn in school assignments. Prior research

examining the efficacy of memory rehabilitation has focused on an examination of explicit memory tasks, such as list-learning or story and design recall, but has generally neglected to consistently examine prospective memory or memory for a future intention, such as remembering to do a future errand or job (McCauley & Levin, 2004; Ward, Shum, McKinlay, Baker, & Wallace, 2007); a classic example of an ecologically valid memory task. Likewise, implicit memory has rarely been examined, although there are clear implications affecting the choice of intervention contingent on the preservation of implicit abilities (Ward, Shum, Wallace, & Boon, 2002). Although prospective and implicit memory may be critical aspects of memory that have consequences for intervention choices, until recently, limited standardized measures focused on either aspect of memory with the exception of some subtests of the Rivermead Behavioural Memory Test (Wilson, Cockburn, & Baddeley, 1985). This instrument is now in its third edition, the Rivermead Behavioural Memory Test-3 (RBMT-3; Wilson et al., 2008), and is appropriate for the assessment of individuals 16 to 96 years of age. More recently, The Cambridge Test of Prospective Memory (CAMPROMPT) (Wilson et al., 2005) was also developed to address this void.

### **Pediatric and Adult Memory Batteries and Measures**

Psychometric measures are typically used to diagnose memory abnormalities, and follow-up assessments are used to help determine short-and longterm progress or decline in memory abilities. Whether an individual is suffering from a degenerative disease, such as Alzheimer's or other dementia, or recovering from a TBI, accurate memory assessment is crucial in order to gauge the patient's progress and assist with treatment plans. There are several standardized adult and pediatric memory batteries that evaluate different aspects of memory and learning, and these are composed of various verbal and nonverbal/visual memory subtests. Common verbal memory subtests include story recall and list-learning tasks; whereas visual memory subtests often involve recall of pictures or designs. In addition, some assessment batteries include a WM or an attention/concentration index based on tasks such as digit span and number-letter sequencing. Scores on each subtest are converted to scaled scores that are then converted into composite scores or indices. A list of commonly used memory batteries is provided in Table 6.1.

Assessments such as the *Test of Memory and Learning–Second Edition (TOMAL-2)* (Reynolds & Voress, 2007) and *Wide Range Assessment of Memory and Learning–Second Edition (WRAML-2)* (Sheslow & Adams, 2003) are more

amenable to repeated testing across different developmental levels, as they encompass a wide age range, whereas the *Children’s Memory Scale (CMS)* (Cohen, 1997) and *Wechsler Memory Scale–Fourth Edition (WMS-IV)* (Wechsler, Holdnack, & Drozdick, 2009) are more age-specific. The *WMS-IV* also derives scores for an Older Adult battery that is a modified version of the standard Adult battery intended for use with individuals aged 65 to 90 years.

**TABLE 6.1 Commonly Used Memory Batteries**

TEST	INDEX SCORES	AGE RANGE
<i>Children’s Memory Scale (CMS)</i>	Visual immediate, visual delayed, verbal immediate, verbal delayed, general memory, learning, attention/concentration, delayed recognition	5 to 16 years
<i>Rivermead Behavioural Memory Test— Third Edition (RBMT-3)</i>	General Memory Index based on 14 subtests assessing various aspects of declarative and prospective memory, as well as novel learning	16 to 96 years
<i>Test of Memory and Learning— Second Edition (TOMAL-2)</i>	Verbal memory, nonverbal memory, composite memory, verbal delayed recall, learning, attention & concentration, sequential memory, free recall, associative recall	5 to 59 years
<i>Wechsler Memory Scale—Fourth Edition (WMS-IV)</i>	Auditory memory, visual memory, visual working memory, immediate memory, delayed memory	16 to 65 years, 65 to 90 years (older adult)
<i>Wide Range Assessment of Memory and Learning (WRAML-2)</i>	Verbal memory, visual memory, attention/ concentration, recognition	5 to 90 years

The comprehensive batteries listed can be time-consuming to administer, typically taking an hour or longer to account for necessary time lapse due to delayed recall and recognition tasks. Shorter, more task-specific assessments are also available. Commonly used list-learning tasks include the *Rey Auditory-Verbal Learning Test (RAVLT; Rey, 1958)*, the *California Verbal Learning Test, Second Edition (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000)*, and the corresponding children’s version (*CVLT-C; Delis, Kramer, Kaplan, & Ober, 1994*). The *CVLT-II* is used with individuals aged 16 to 89 years. These tests examine episodic memory through an examination of the patient’s ability to learn and remember a list of words; however, there are some subtle differences. Both the *RAVLT* and *CVLT* include repeated trials and include an interference list to examine the effects of proactive interference (PI); that is, the degree to which prior learning interferes with new learning. Both also include delayed and/or recognition trials (Spren & Strauss, 1998). The *CVLT-II* and *CVLT-C*

precede in much the same manner as the *RAVLT*; however, the lists consist of semantically clustered groups so that the patient's ability to employ executive or organizing strategies to enhance learning can be examined. The *CVLT-II* also includes a forced-choice recognition subtest, completed 10 minutes after delayed recognition to specifically probe for the benefits of cuing on retrieval. Again, methods for examining semantic relative to serial clustering and the degree of intrusive errors can be determined to further understand factors affecting the memory deficit.

Commonly used visuospatial memory tests include the *Benton Visual Retention Test—Fifth Edition (BVRT; Sivan, 1992)* and the *Rey-Osterrieth Complex Figure Test (RCFT; Osterrieth, 1944; Rey, 1941)*. The *BVRT* can be used with individuals aged 8 through adult, and consists of 10 designs that are individually displayed for 10 seconds. After the 10-second display, the patient is asked to draw the design from memory. Other administration procedures, such as exposing the designs for only 5 seconds or allowing the patient to copy the designs without removing the stimulus cards, are also used to examine visual memory, perception, and visual-constructive abilities. Designs are scored based on specific criteria involving the number of correct reproductions and an error score that includes omission errors, distortions, perseverations, rotations, misplacements, and size errors (*Spreeen & Strauss, 1998*).

In addition to visual memory, the *RCFT* allows for assessment of planning and organization skills, and nonverbal learning (*Kasai et al., 2006; Waber & Holmes, 1986*). The test consists of a figure made up of 18 components, and can be used with individuals 6 to 89 years of age. In addition to a copy condition, both immediate and delayed recall (i.e., 30 minutes), as well as recognition trials, are administered. Several scoring systems are available for the *RCFT*, most of which provide criteria for copy accuracy and recall of details; however, some examiners use a more qualitative approach based on visual inspection (*Spreeen & Strauss, 1998*). This measure particularly pulls for the integration of perceptual–motor, executive, and visual memory abilities.

## **REHABILITATION OF MEMORY IMPAIRMENT**

The past 30 years of research has revealed that interventions are capable of improving attention, memory, and executive function. However, the degree to which “restorative” relative to compensatory intervention is of benefit is related to a host of factors and most support has surfaced primarily for compensatory approaches. The exception is found in the area of intervention efforts to improve

attentional deficits postinjury. Memory training is the most frequently prescribed form of rehabilitation (Helmick et al., 2010), and interventions for memory impairment fall into the larger domain of cognitive rehabilitation. Cognitive rehabilitation broadly refers to a multitude of treatments that aim to improve a person's cognitive and/or functional abilities following an injury or disease by retraining and/or teaching compensatory methods to enhance performance of lost skills, or by promoting performance of newly acquired abilities. The American Congress of Rehabilitation Medicine has provided the following definition of cognitive rehabilitation—"a systematic, functionally oriented service of therapeutic activities that is based on assessment and understanding of the patient's brain-behavioral deficits" (Cicerone et al., 2000, p. 1597). Interventions for memory rehabilitation focus on improving various aspects of memory performance, such as remembering past events, learning new information, recalling face-name associations, and remembering to perform a task (i.e., prospective memory). Whether in acquired or degenerative disorders, memory rehabilitation has aimed to enhance daily functioning through three primary strategies: (a) methods that facilitate residual explicit memory with support provided at both encoding and retrieval; (b) strategies that attempt to exploit the intact implicit memory system; and (c) interventions that enhance performance of daily functions through the use of external memory aids (De Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001). Table 6.2 provides a summary of commonly used methods of intervention that will be described in the subsequent sections.

Although the term "restorative" has often been associated with strategies to exploit implicit memory, and the other two approaches have been perceived as more or less compensatory, it is this author's opinion that all the methods employed are compensatory, as they require enhancement or facilitation of alternate cognitive systems and approaches to promote functioning. In addition, methods that attempt to exploit preserved implicit memory have more often been employed in the treatment of degenerative disorders, such as AD, given that the subcortical, implicit memory system is relatively preserved until late in the disease process.

## **TABLE 6.2 Summary of Commonly Used Methods of Intervention for Memory Impairment**



TYPE OF INTERVENTION	COMPENSATORY AND RELIES ON INTERNAL STRATEGY DEVELOPMENT	COMPENSATORY AND RELIES ON EXTERNAL DEVICES AND ENVIRONMENTAL AIDS	"RESTORATIVE" AND FOCUSES ON LEARNING DOMAIN-SPECIFIC KNOWLEDGE (E.G., FACE-NAME ASSOCIATIONS)
Mechanism of action	Facilitates residual explicit memory	Enhances function through external aids	Exploits intact implicit memory
Empirical support	Practice standard for mild memory impairment for TBI	Practice standard for mild memory impairment from TBI Practice guideline for severe memory impairment for TBI	Works best with severe impairment
Strategy	<ul style="list-style-type: none"> <li>• Verbal mnemonics: Story method</li> <li>Acronyms</li> <li>Sentences</li> <li>Chunking</li> <li>• Visual imagery mnemonic and imagery-based training</li> <li>Method of loci</li> <li>• Repetition and rehearsal strategies</li> <li>• Elaborative encoding</li> <li>• PQRST and metacognitive strategies</li> </ul>	<ul style="list-style-type: none"> <li>• Nonelectronic memory aids</li> <li>Memory notebooks</li> <li>• Electronic memory aids</li> <li>Personal data assistants (PDA)</li> <li>Cell Phone/Blackberry</li> <li>Memory glasses</li> <li>SenseCam</li> </ul>	<ul style="list-style-type: none"> <li>• Focus on domain-specific knowledge teaching</li> <li>• Error learning (EL)</li> <li>• Spaced retrieval (SR) and expanded rehearsal techniques</li> <li>• Method of VCs</li> <li>• Strategies to enhance motor and procedural memory</li> </ul>

## Methods That Facilitate Residual Explicit Memory or Enhance Function Through External Aids

These compensatory methods can include both *internal* and *external* strategies to help patients achieve their goals. Internal strategies are used when patients rely on residual memory skills by applying mnemonics, visual imagery, rehearsal strategies, and/or structuring and organizing information in a way that will enhance encoding and subsequent retrieval (Geusgens, Winkens, van Heugten, Jolles, & van den Heuvel, 2007). Mnemonics are verbal or visual strategies that one uses to help recall information more effectively. These methods are consciously learned and require substantial effort to develop (Harris, 1984). First-letter mnemonics are a very common strategy employed when attempting to learn new information. Without the mnemonic, “On Old Olympus’ Towering Top A Fin And German Viewed Some Hops,” it is unlikely that I would have ever learned the 12 cranial nerves—Olfactory, Optic, Oculomotor, Trochlear, Trigeminal, Abducens, Facial, Auditory/Vestibulocochlear, Glossopharyngeal, Vagus, Spinal Accessory, and Hypoglossal. Elaboration or turning isolated words into a meaningful story is also a commonly used verbal mnemonic. Visual

mnemonics or visual imagery can be defined as essentially using pictures to more effectively encode and subsequently retrieve information. A common example of the use of visual imagery occurs, when we attempt to learn the face–name association of someone that we’ve just met. We may associate a feature of her face with her name (e.g., Ms Smiley has large white teeth), or her name and face with a person who is already well known. Finally, the method of loci is a visual strategy that can be used to recall information by yoking the to-be-remembered information to a location or place, such as where a person is sitting. An elaboration of this visual imagery method is to link each of the objects to be remembered to the subsequent image or object. That is, the first image is linked to the second, the second to the third, third to the fourth, and so on. Although this method has been proven to be a successful mnemonic for those nonimpaired, support for its effectiveness in memory rehabilitation is more limited ([Wilson, 2009](#)).

In contrast, external methods include techniques such as memory notebooks, and the use of assistive technology, including personal computers and portable electronic devices such as personal digital assistants, pagers, and voice recorders. The vast majority of memory-training studies have explored the use of these tools as essentially serving as “memory prosthetics.” These devices have been used to prompt daily living tasks, such as taking medication, as well as for guidance systems with more complex tasks, such as vocational performance ([Kirsch, Levine, Lajiness-O’Neill, & Schnyder, 1992](#)). One of the most effective interventions is the memory notebook that typically includes sections for orientation, a memory log, a calendar, to-do lists, transportation, a feelings log, and names of those who may be personally relevant ([Tsaousides & Gordon, 2009](#)). Memory aids with tools, such as automated reminders, appear to have the potential to be significantly effective in helping those with prospective memory to compensate for their difficulty, particularly in mildly to moderately impaired patients ([Kapur, Glisky, & Wilson, 2004](#); [Wilson, Emslie, Quirk, & Evans, 2001](#)). The largest study conducted demonstrating the use of electronic memory aides was that reported by [Wilson \*et al.\* \(2001\)](#) using NeuroPage for which a text-based reminder was sent to the patient via a simple pager. This study revealed that participants increased their attainment of everyday goals by an average of 30% while using the pager, and the benefits persisted beyond the time that the pager was used in some patients. It has been suggested that the pager consolidated the intentions into a routine. One difficulty with automated reminders for tasks is the efficiency with which these systems can be marketed.

The risk that the platform on which a system runs becomes obsolete before it is able to reach the market is a legitimate concern for developers. For a comprehensive review of the use of external aids for managing memory deficits, see [Sohlberg et al. \(2007\)](#). A frequently encountered question by clinicians is whether an individual is a good candidate for a compensatory intervention, such as an external memory aid. [Evans and colleagues \(2003\)](#) reported that the following are important predictors of those who are likely to use and benefit from compensatory methods ([Wilson, 2009](#)):

1. Age—younger compared to older individuals compensate better.
2. Severity—the more severely impaired the individual is, the less likely he will benefit from compensatory methods.
3. Specificity of deficit—individuals with more diffuse cognitive impairment are less capable of compensating than those with more focal cognitive deficits.
4. Premorbid use of strategies—individuals who had used compensatory methods premorbidly were more likely to compensate more effectively postinjury or illness.

### **Interventions for Acquired Brain Injury**

Recently, a subcommittee of the Brain Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine completed a series of systematic reviews of the extant literature on cognitive rehabilitative methods for TBI and stroke to explore the evidence for cognitive rehabilitation and to provide clinical practice recommendations. Evidence-based *practice standards, guidelines, and options* for practitioners working in TBI care were established ([Cicerone et al., 2000, 2002, 2005](#)). *Practice standards* were based on evidence from class I, prospective, randomized, or quasi-randomized controlled studies that provided evidence for *strategy training to improve attention and memory*, as well as *external aides such as memory notebooks to address memory impairment* for individuals with mild memory impairment from TBI. *Practice guidelines* were based on class II evidence, including prospective, nonrandomized cohort or retrospective, and nonrandomized case controls or clinical series with controls, which affirmed the use of *external compensatory aides with direct application to functional activities* for individuals with severe memory impairment from TBI. Finally, *practice options* were based on class II and III evidence, including clinical series without controls or single-case designs, which provided support

for the interventions that promote the use of self-regulation strategies through self-instruction and self-monitoring to aid with memory function.

Similarly, a consensus conference for mTBI was conducted by the Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury and the Defense and Veterans Brain Injury Center to develop clinical guidance for the care of service members with persistent postconcussion symptoms, including cognitive rehabilitation ([Helmick et al., 2010](#)). Cognitive rehabilitation, including memory rehabilitation, is an important component of comprehensive rehabilitation for individuals with moderate and severe TBI, and is being used more consistently with individuals with persistent deficits following mTBI. Currently, there is no evidence to suggest that the cognitive deficits incurred from combat-related and other forms of injury resulting in mTBI differ or require alternative forms of intervention. Training in the use of external memory/organizational aids, the use of internal memory strategies, and in the use environmental aides are interventions with demonstrated efficacy in mTBI.

One of the most critical domains of memory impaired following injury is that of *prospective memory*, which refers to a series of processes involved in the formation, storage, and effective retrieval of an intention. Poor prospective memory is highly correlated with self-reported memory problems in individuals with and without neurological injury or impairment ([Ellis, 1996](#); [Fish, Wilson, & Manly, 2010](#)). If one is unable to act on the intention immediately, the individual must actively rehearse the intention or encode it in a manner such that it is likely to surface when enactment is possible. Numerous schemes for classifying prospective memory have been suggested. A common scheme is to distinguish between an event-based task (e.g., mail a letter when one encounters a mailbox), time-based tasks (e.g., call your doctor at 1:00), and an activity-based task (e.g., take your medication before bed) ([Einstein & McDaniel, 1996](#)). According to recent investigations, prospective memory should be understood as a product of many cognitive processes common to other tasks, rather than having a specific neural basis. There are no confirmed reported cases of pure prospective memory impairment. Indeed, prospective memory impairment arises in cases of more general memory, attentional, and executive problems. Prospective memory impairment is observed in a host of disorders, such as brain injury, dementia, depression, and schizophrenia.

[Fish, Wilson, and Manly \(2010\)](#) recently outlined methods to assist individuals with prospective memory impairment, alerting clinicians to address the deficits through individualized goals and within a holistic framework of

intervention. Four main areas of intervention were addressed: (a) retraining approaches, (b) supporting the retrospective component of the prospective memory tasks, (c) supporting the executive component of prospective memory tasks, and (d) supporting mnemonic and executive aspects of prospective memory tasks. Repeated practice of simple prospective memory tasks over increasing delays has been attempted and benefits reported in several case studies (Sohlberg, White, Evans, & Mateer, 1992b; Fleming, Shum, Strong, & Lightbody, 2005); however, variable generalization has been reported, and interpretations are limited by lack of control data. Impaired retrospective memory is likely to substantially interfere with prospective memory. Several studies involving patients with AD have used methods such as spaced retrieval (SR), EL, and elaborated encoding (discussed later) with some success to strengthen the memory trace to enhance the execution of that intention. As noted, prospective memory has a strong overlap with executive functions. Approaches to treat executive impairment have used systematic approaches to solve problems, and the use of these strategies has been of benefit in helping patients perform better on untrained tasks compared to controls.

### **Pediatrics With Acquired Injuries**

The use of cognitive rehabilitation methods in children is complicated by the fact that the loss is magnified by the cascading effects of disruption during a rapid phase of growth and development, coupled with the impact of the injury on future development, the so-called “growing into deficit” phenomenon (Fletcher, Miner, & Ewing-Cobbs, 1987). There are several reviews of cognitive rehabilitation in adults following brain injury (Cicerone et al., 2000, 2005; Eslinger, 2002; Kennedy et al., 2008; Rohling et al., 2009; Sohlberg & Mateer, 2001). In contrast, four reviews in the pediatric brain injury literature exist on the efficacy of cognitive interventions (Laatsch et al., 2007; Limond & Leeke, 2005; Michaud, 1995; Slomine & Locasio, 2009). With respect to memory rehabilitation, as with the adult literature, the focus has historically been restricted to the assessment and treatment of semantic and episodic memory. Interventions with children and adolescents have adopted similar strategies to address these difficulties (Ylvisaker, 1998), although limited studies have been conducted examining their efficacy. Interventions have focused on explicit instruction in the use of memory and rehearsal strategies, and training in the use of external memory aids. The memory strategies previously discussed for adults are also those that have been employed with children, such as organizational and

mnemonic methods and rehearsal strategies for improving the efficiency of encoding new information. As noted, this is a practice standard for memory deficits, as recommended by the American Congress of Rehabilitation Medicine (Cicerone et al., 2005). Franzen and Martin (1996) used elaborative encoding strategies (e.g., matching words to definition, formulating semantic relationships, or using words in self-generated sentences), as well as the PQRST (i.e., Preview passage, generate Questions relating to the passage, Read the passage, State answers to questions, and Test oneself) method described by Robinson (1970) to enhance the memory trace. Consistent with cognitive psychology literature in noninjured individuals, elaborative encoding has also been shown to enhance recall in children (Oberg & Turkstra, 1998). However, as with other techniques, generalization of this method to passages from academic texts was less successful. Enhanced memory performance has been reported with the use of external aids, and when interventions target domain-specific skills. There is also limited support for improved memory performance following “attention process training” (APT) (Sohlberg & Mateer, 1989). Specifically, in the Amsterdam Memory Training Program for children, improvement in delayed word memory and attention was noted following attention training, although no significant difference in immediate memory performance was reported. Multifaceted metacognitive approaches targeting attention and executive processes to treat memory deficits in teenagers have revealed significant improvements on task performance and memory scores, primarily attributed to enhanced verbal memory (Brett & Laatsch, 1998; Franzen & Martin, 1996). Research findings in children are perceived as equivocal, and interventions to date have not consistently assessed generalizability or conducted adequate follow-up (Slomine & Locascio, 2009). Consistent with the adult literature, general improvement in memory following the use of restorative strategies (Schacter & Glisky, 1986) has not been demonstrated in children.

### **Stroke Rehabilitation**

Consistent with the TBI literature, memory impairment is the most common cognitive consequence of stroke (Erikson, 1977). When internal devices such as mnemonics and rehearsal strategies have been employed in stroke, as might be expected, the use of methods that tap preserved hemispheric abilities has been reported to be of some success. Gasparrini (1979) found visual imagery mnemonics to be a significantly better strategy for improving memory function than verbal mediation in patients with mild aphasia following left-hemispheric

strokes. In general, few between-group studies have been conducted exploring the efficacy of memory retraining in stroke ([Majid, Lincoln, & Weyman, 2007](#)). [Wilson \(1992\)](#) examined the effectiveness of 3 weeks of problem-solving training and daily memory therapy in patients with stroke and head injury. No significant difference was found between the groups on measures of memory following intervention. Likewise, [Doornhein and de Haan \(1998\)](#) did not find significant group differences overall, when comparing a training program involving six memory strategies with repetitive practice in patients with stroke. However, following 4 weeks of training, a significant group difference was obtained on a test of face-name associations.

## Schizophrenia

Schizophrenia is one of the few psychiatric disorders for which the efficacy of cognitive rehabilitation has been examined. Neuropsychological investigations have revealed deficits in a broad range of abilities in schizophrenia, including (a) verbal ability, (b) complex perceptual skills, (c) abstraction and cognitive flexibility, (d) attention, (e) learning, (f) sensory abilities, and (g) motor skills ([Kurtz, Moberg, Gur, & Gur, 2001](#)). Nonetheless, the most significant decline is noted on measures of verbal learning and memory ([Saykin et al., 1994](#)), and the deficits are resistant to the effects of typical antipsychotic medication. Despite these findings, research investigating the benefits of cognitive rehabilitation approaches to remediate memory in schizophrenia has been more limited. [Medalia, Revheim, and Casey \(2000\)](#) examined the effectiveness of computerized exercises to target memory in 54 chronic inpatients, who were randomly assigned to (a) a memory remediation group, (b) a problem-solving remediation group, or (c) nonremediated control condition. The authors found that although memory performance on specific tasks could be enhanced through practice using a variety of compensatory methods, such as acronym creation, verbal-visual pairing, and so on, these methods did not generalize to other memory tasks. In a large, randomized, controlled study investigating the effects of an intensive remediation program on a broad battery of neurocognitive measures, [Bell and colleagues \(2001\)](#) reported improvement on measures of effect recognition, WM, and executive function following 50 hours of neurocognitive enhancement therapy (NET). Nonetheless, no effects were reported on secondary verbal or nonverbal memory. In a recent meta-analytic review of randomized controlled trials of psychological treatments in schizophrenia, specifically social skills training and cognitive remediation, the

authors concluded that cognitive rehabilitation had no benefit on attention, verbal memory, visual memory, planning, cognitive flexibility, or mental state (Pilling et al., 2002). More specifically, the authors reported four studies exploring verbal memory strategies in 117 patients (Benedict et al., 1994; Medalia et al., 2000; Tompkins et al., 1995; Wykes et al., 1999), and two studies exploring the efficacy of visual memory methods in 48 patients (Tompkins et al., 1995; Wykes et al., 1999). The reported effect sizes failed to reveal any benefit for cognitive remediation of memory deficits in the patients compared to matched controls.

## **STRATEGIES THAT ATTEMPT TO EXPLOIT THE INTACT IMPLICIT MEMORY SYSTEM**

### **Degenerative Disorders and Nondegenerative Forms of Brain Injury**

Given that functional outcome and quality of life are highly related to memory impairment in disorders such as cerebrovascular accident, TBI, or the degenerative disorders such as AD (Schalén, Hansson, Nordström, & Nordström, 1994), the understanding of which approaches are likely to be of benefit and generalizing to everyday tasks is a critical area of investigation. A number of cognitive rehabilitation programs have been developed over the past 30 or so years that have used computer programs to drill patients in the hopes of enhancing retrospective memory in the same way that a muscle is developed through exercise. Unfortunately, limited to no generalization has been reported with the majority of these memory practice programs (Wilson, 1997). As noted, other programs use internal strategies, such as mnemonics, or teach patients to use external aides, such as electronic aids, to compensate for their difficulties.

The three levels of memory rehabilitation strategies described earlier also have proven efficacy in the treatment of mild to moderate AD and the related dementias. Facilitation of residual explicit memory can be demonstrated in the early phase of AD under conditions that provide extensive assistance during both the acquisition of the material and retrieval. Encoding can be enhanced through multimodal input, by increasing the material's emotional salience, by providing guidance to engage self-generated semantic encoding, or through the use of activation of task- or event-related prior knowledge, or by anchoring the recall of material to personal life events (Acevedo & Loewenstein, 2007; De Vrees et al., 2001). Retrieval can be enhanced with the use of recall cues consistent with conditions used at encoding.



Alternatively, techniques have been developed based on cognitive neuropsychology and cognitive-experimental methods that attempt to access aspects of human memory that are often spared or relatively preserved in patients with amnesia. As previously noted, a distinction can be made between explicit and implicit memory (Schacter & Tulving, 1994). Whereas explicit memory refers to information that one is consciously aware of acquiring (e.g., facts, words), implicit memory does not require conscious retrieval and includes such memory components as procedural memory (e.g., memory for skills), conditioning and habituation (e.g., habits), and priming. Implicit memory relies on the integrity of subcortical structures and, as such, is often relatively intact in patients with severe amnesia from cortical degenerative disorders, such as AD. Individuals with severely impaired explicit memory display a relative preservation of implicit forms of learning, such as priming, conditioning, and procedural memory, revealing a clear dissociation between explicit and implicit memory within longterm episodic memory (Graf & Schacter, 1985; Tulving & Schacter, 1990).

Two of the most well-researched domains of implicit memory in AD include perceptual repetition priming and procedural memory. In the later, learning is measured as a change in the accuracy, speed, or bias for a stimulus due to prior exposure to that stimulus. Patients with mild to moderate AD display normal repetition priming on a number of tasks such as unfamiliar faces and for single word and text rereading. Procedural memory is measured by the improved accuracy in the execution of a task or ability over repeated trials with subjects having no or limited explicit recall of exposure, such as is evident in mirror-reading studies. Expanded rehearsal methods, the method of vanishing cues (VCs), and errorless learning (EL) are intervention strategies that capitalize on implicit abilities to aid mild to moderately impaired patients with AD or injury to acquire new or to relearn prior domain-specific knowledge, such as face–name associations (Baddeley & Wilson, 1994; Glisky et al., 1986a, 1986b; Kessels & de Haan, 2003). Glisky and colleagues (1986b) originally examined the utility of EL to treat patients with memory deficits secondary to brain injury and neurological illness. Recent meta-analytic and review articles have explored the utility of memory rehabilitation efforts based on the assumption of intact implicit learning capacity, such as EL and VC methods (Clare & Jones, 2008; Kessels & de Haan, 2003).

EL is based on the assumption that errors that are produced during learning interfere with the correct response (Baddeley, 1992). In humans, there is

evidence that these errors are stored and consolidated through implicit processes in amnesic patients. EL refers to a learning condition that involves the elimination of errors during the learning process (Clare & Jones, 2008). The theoretical and empirical origins of EL in memory rehabilitation can be reviewed in the seminal animal experiments of Terrace (1963), who revealed that discrimination previously thought to require exposure to both the reinforced (S+) and nonreinforced (S-) stimuli would be learned through exposure to the reinforced (S+) stimulus alone. Errors can be eliminated through a number of methods during memory rehabilitation, including (a) breaking the targeted task into small, discrete units; (b) providing sufficient models before the patient is asked to perform the task; (c) encouraging the patient not to guess; (d) immediately correcting errors; and (e) carefully fading prompts (Sohlberg et al., 2005). EL has typically been contrasted with trial-and-error or errorful learning (EF) that is based on error correction while training, and for which guessing is encouraged during acquisition. The literature has shown that amnesic patients learn better through EL compared to EF. EF appears to be less successful given that the errors occurring during the learning phase interfere with the accurate information to be learned, possibly at an implicit level. As such, the memory trace for wrong answers interferes with the accurate memory trace.

General support for EL methods has risen from results of investigations of patients with brain injury for which learning of word lists, verbal paired-associates, names, pictured objects, and general information were required. Learning and relearning face-name associations and personal information are often important goals in the rehabilitation of individuals with memory impairment, and EL has been shown to be a useful method to train these skills (Clare et al., 1999, 2002). However, when errorless and trial-and-error methods have been directly compared in trials of face-name associative learning in early-stage dementia, no advantage for errorless methods has been shown (Dunn & Clare, 2007). In addition, errorless methods have been shown to be more beneficial for more specific, low-level knowledge compared to more general, high-level information.

Recent investigations of EL methods in memory rehabilitation have attempted to enhance the benefits of EL through active processing, such as making evaluative judgments of the information to be acquired (Kalla et al., 2001). In this investigation, EL with preexposure to face-name pairs in which participants were required to make an evaluative judgment was more effective than EL alone, suggesting that pretraining and preexposure do appear to be of

benefit. Overall, the vast majority of the studies over the past decade have involved the learning of discrete, precise behaviors, such as words and names. These studies have generally shown that EL methods are superior to errorful methods, although learning may be very specific (Clare & Jones, 2008; Stark et al., 2005).

The method of VC is “a form of backward chaining that provides the client with progressively weaker cues following successful recall of targeted information” (Sohlberg et al., 2005). In contrast to true errorless methods, in the VC method, the patient is explicitly encouraged to guess the correct answer when cued. As such, this method is an adapted method that facilitates error-reduction. Moreover, many learning trials are required for this technique. However, the literature suggests that preventing the occurrence of errors during learning in amnesic patients is of benefit.

Another error reduction or elimination method that has been used in memory rehabilitation is the expanded rehearsal technique, which was originally described as SR (Landauer & Bjork, 1978). This method is based on the finding that the “time scheduling of retrieval attempts affects the degree to which benefits are observed as a result of retrieval practice, with most benefits accruing when test trials are spaced at gradually expanding intervals” (Clare & Jones, 2008; Landauer & Bjork, 1978). Practice guidelines for this method have been developed for individuals with dementia (Hopper et al., 2005). In this method, the patient repeatedly recalls information at short but gradually increasing time intervals (e.g., seconds). If an error occurs at a longer interval, it is immediately corrected and followed by a return to the previous interval at which success was achieved, followed by a reexposure to the target information. If the patient is still unsuccessful, the interval is halved. Intervals are filled with an interfering task, such as discussion. Successful storage is reported to occur if the individual is able to retrieve the information after 15 minutes to 1 hour (Camp et al., 2000). SR has been found to be effective in teaching individuals with brain injury (Melton & Bourgeois, 2005) and dementia (Bourgeois et al., 2003; Camp & Stevens, 1990; Camp et al., 1996). However, others have suggested that SR is no more effective than other schedules of distributed rehearsal in promoting longterm retention in individuals with dementia. There is no strong evidence of spontaneous generalization to nontargeted information noted with SR methods.

Limited information is currently available with respect to generalization of these methods. The results after long delays (i.e., weeks) are inconclusive, with some finding benefit 1 to 4 weeks after testing (Kern et al., 1996), whereas

others found large effect sizes up to 6 weeks after training with EL methods in head injury or encephalitis (Glisky et al., 1986a, 1986b). Finally, some investigations have reported that EL methods work best with individuals with severe amnesia, such as those in the later stages of AD (De Vreese et al., 2001), who have limited explicit memory capacity. However, other investigations have reported that while EL methods were more effective in teaching skills, such as face–name associations, in moderately impaired Alzheimer’s patients, when tested immediately, the difference between EL and EF methods was not evident after a 10-minute delay (Ruis & Kessels, 2005). Indeed the benefits of EL methods are not evident for all groups, some results are equivocal, and limitations exist with this method (Clare & Jones, 2008). In summary, EL methods appear to be of benefit in memory rehabilitation for teaching information to verbal individuals with memory deficits from brain injury, particularly those with severe impairment. It may be less advantageous to other types of tasks, and the benefits to those with mild and moderate dementia appear to require further investigation. In fact, Thone and Glisky (1995) have found that visual imagery methods and verbal elaboration are more effective than MVC in learning name–face associations in patients with mild AD.

As noted, while access to implicit memory mechanisms has been touted to explain the observed benefits of errorless methods in memory rehabilitation, others argue that the EL advantage may be enhanced by explicit memory, when there is sufficient residual explicit memory (Page et al., 2006). This may account for the more substantial benefit in those with severe explicit memory impairment compared to those with early-stage dementia, who do not gain as much from error-reduction methods. In the former, it is believed that errors generated during effortful learning lead to reduced performance, because the implicit system is not capable of distinguishing from correct responses. An additional benefit of EL methods is that they place fewer demands on attentional resources. If EL methods rely, at least partly, on implicit operations, such as priming, then attention is likely to be focused more heavily on the surface features, rather than contextual features (e.g., spatial or temporal context). As such, the responses can be primed, but may not be effectively encoded into episodic memory. Priming will reduce encoding variability given that the same set of discrete, stimulus features, but this will result in a sparse encoding experience (Wagner, 2002). As such, using methods such as elaboration may aid in improving the context or source memory. In general, effortful conditions that employ evaluative processes to improve encoding are more likely to enhance accurate source memory, and

these can be used more with those with some residual explicit memory functioning.

Several multimodal cognitive rehabilitation programs have been reported to be successful in enhancing functional outcome in patients with AD. [Quayhagen and colleagues \(1989, 1995\)](#) developed both caregiver-implemented as well as a cognitive stimulation program that consists of memory, problem-solving, and communication exercises. Patients displayed maintenance of performance on many of the neurocognitive measures for up to 8 and 9 months compared to control groups using this method. Goal management training (GMT) is a relatively new cognitive rehabilitation method used for older adults who experience normal cognitive decline ([Levine et al., 2011](#); [Winocur et al., 2007](#)). In GMT, participants are required to think about their unique experiences and discover which techniques (e.g., breaking down activities into goals and subgoals, using mental imagery, making a to-do list) are likely to work best for them. [Levine and colleagues \(2011\)](#) recently reported benefits of GMT using a randomized control trial of 12 weekly, 3-hour sessions that combined memory and psychosocial training in healthy older adults. Generalization and maintenance continue to be dependent on the spontaneous access to those skills in everyday life. As such, automated cueing systems have also been employed and investigated. For example, a tone or buzz might signal an individual to stop, pause, and think about where one is going and to consider one's intentions. [Manly and colleagues \(2002\)](#) have demonstrated the benefit of this method in a study for which randomly timed text messages were sent to braininjured subjects. The text read "STOP," a mnemonic for Stop, Think, Organize, and Plan. Prospective memory was found to be strongly superior on days for which cues were sent. Moreover, the strategies used extended over a period of at least 2 weeks following the training.

## MEMORY GROUPS

Orientation and reality-orientation groups have been in existence for over 40 years ([Folsom, 1968](#)). The goal of these groups has been primarily to enhance orientation, decrease confusion, and to promote socialization among geriatric patients in extended care facilities or for those with severe memory impairment following brain injury in residential treatment programs. Inpatient groups are typically conducted daily for 30 to 60 minutes. In addition to addressing orientation, these groups typically incorporate psychoeducation, including instruction on the use of both internal and external memory aides ([Berg et al.,](#)

1991). Similarly, outpatient programs can be developed for patients with mild–moderate memory impairment with the goal of individualized and group instruction on the use of various internal strategies, such as verbal and visual mnemonics and rehearsal methods, as well as external aids such as memory notebooks.

## **PARAMETERS OF INTERVENTION (DURATION, FREQUENCY, INTENSITY) AND MEASURED OUTCOMES**

The parameters of intervention regarding the required dosage to effect change have varied significantly from one study to the next, making interpretations difficult. In the adult-acquired brain injury literature, intervention sessions using a variety of techniques, such as imagery training or mnemonic methods, have reported sessions that occur on average for 1 hour per week, and from one to three times per week. The duration of treatment is from as low as 3 to 4 weeks (Boman et al., 2004; Doornhein & de Haan, 1998), to 8 to 10 weeks (Fleming et al., 2005; Kaschel et al., 2002), to upward of 11 to 27 weeks (Cicerone, 2002; Kime et al., 1996; Wade & Troy, 2001). In contrast, errorless methods are usually employed until some specific criterion is met, rather than based on some specific time frame. In pediatric brain injury, duration of treatment has been reported with more vague parameters (e.g., 15–30 minutes on nonconsecutive days) (Franzen & Martin, 1996), or again ranging anywhere from 6 to 7 weeks (Oberg & Turkustra, 1998; Wilson et al., 2001), to 17 to 19 weeks (Lawson & Rice, 1989; Hooft et al., 2005), or as high as 18 to 42 sessions (Brett & Laatsch, 1998). In the stroke literature, treatment duration has been equally variable with studies generally reporting 3 to 4 weeks of intervention (Doornhein & de Haan, 1998; Wilson, 1992).

One of the most critical determinants of the success of intervention is the degree to which the intervention transfers or generalizes to functional abilities, or the extent to which prior learning affects new learning (Geusgens, Winkens, van Heugten, Jolles, & van den Heuvel, 2007). Outcome has most often been reported by self-or relative-report of daily living tasks and by performance on neuropsychological measures of memory functioning. Overall, generalization of skills such as imagery training, elaborative encoding, or other mnemonic methods has been modest to absent or not statistically tested in disorders ranging from brain injury to epilepsy (Boman et al., 2004; Doornheim & de Haan, 1998; Kaschel et al., 2002; Ponds & Hendriks, 2006). Fester and Skinner (1957)

demonstrated a clear relationship between response effort and motivation, yet this is rarely considered or addressed in memory rehabilitation outcome and generalization. A clear example of the importance of this variable is reflected in the motivation of patients to learn or relearn the names of family members, but who may be less interested in learning lists of words.

## PHARMACOLOGIC AUGMENTATION

Over the past decade, there has been increased interest in exploring whether pharmacologic intervention has the ability to augment cognitive rehabilitation and training efforts in disorders such as schizophrenia (Friedman, Temporini, & Davis, 1999) and in programs for nondemented older adults (Yesavage et al., 2007). This would be analogous to the reported benefits of combining antidepressants with cognitive-behavioral interventions in the treatment of depression. The use of medication would hypothetically aid with improving encoding and subsequent retrieval by “priming the pump” (Yesavage et al., 2007), and these effects might occur through direct augmentation or indirectly through enhancement of attentional processes. Medications that increase cortical cholinergic activity such as AChE inhibitors (AChEIs) and M1/M4 muscarinic agonists have received the most support for their ability to enhance memory. AChEIs inhibit the enzyme acetylcholinesterase, thereby increasing the concentration of acetylcholine in synapses. Anticholinergics such as donepezil may assist older adults in their ability to assimilate and use mnemonic strategies. In a recent study by Saykin and colleagues (2004), WM performance was examined in MCI patients who had received donepezil for 6 weeks. WM improvement was positively related to frontal activity, as well as baseline hippocampal volume.

The next most plausible candidate to investigate with respect to its ability to enhance memory or augment the benefit of memory remediation is likely to be memantine, an N-methyl-D-aspartate (NMDA) receptor agonist that regulates glutamate. The NMDA receptor has been the primary pharmacological target to reduce glutamatergic activation, hypothesized to be central to several neuropsychological disorders (Muir, 2005). Glutamate is reported to play a role in memory functioning by regulating the amount of calcium flow into the nerve cells. Excessive glutamate results in excitotoxic events and eventually cell death. Under conditions of excessive glutamate exposure, as may be the case with dementia, TBI, and epilepsy, memantine enters the NMDA receptor, blocking its activity. Studies thus far have revealed the efficacy of memantine in the

treatment of both cognitive and clinical symptoms noted in moderate to severe AD (Areosa & Sherriff, 2003).

In addition, those medications that have indirect effects on memory through attentional mechanisms, specifically nicotinic receptor agonists and stimulants, are currently being investigated for their ability to improve memory and functional outcome. Pharmacotherapies targeting dopaminergic systems have shown benefits in attention, improved executive functioning, and enhanced performance on memory tasks in TBI (Bales et al., 2009; Chew & Zafonte, 2009).

### **FUTURE DIRECTIONS—ASSISTIVE TECHNOLOGY AND NEURAL REPAIR**

According to the Centers for Disease Control and Prevention, at least 1.7 million individuals sustain TBIs in the United States each year with about 52,000 deaths, 275,000 hospitalizations, and 1.365 million who are treated and released ([http://www.cdc.gov/traumaticbraininjury/tbi\\_ed.html](http://www.cdc.gov/traumaticbraininjury/tbi_ed.html)). In addition, an estimated 5.3 million Americans have AD. This number has doubled since 1980, and the number afflicted with AD and related dementias in the United States is projected to triple to 13 million by 2050. These are the two highest base rate disorders with memory impairment, and the statistics underscore an increasingly important area of research in memory rehabilitation—the need for intelligent cognitive devices (Bharucha et al., 2009). There are a multitude of commercially available time-based and task reminder systems. More recently, prospective memory aids have been developed that use artificial intelligence to provide procedural guidance for a task or determine whether and when a reminder is required. Aids such as Memory Glasses, MemoryClip, and Friedman can be used to enhance performance of many different tasks throughout the day. For example, Memory Glasses is an aid that is embedded in glasses and is context-aware. It is used to provide reminders to the patient to complete a task in a timely manner. Memory Glasses use a variety of computer-perception methods and captured visual images to capitalize on the context. For example, it may be able to provide a prompt to permit safe crossing of a street. Other intelligent systems, such as ISAAC, AutoMinder, and Friedman, are programmed to help improve a sequence of steps in simple or multiple step tasks. They can be customized to capitalize on the patients remaining cognitive abilities using text, audio, or visual displays. Unfortunately, none of the methods has yet undergone clinical trials with dementia patients.



Retrospective intelligent memory aids are also being explored. Microsoft's SenseCam is a digital camera that takes photographs passively, while worn by patients. It is capable of recording events that can be subsequently viewed by the patient and is thought to enhance autobiographic memory. A small number of clinical cases have been reported, including the reported benefit of SenseCam to aid in the formation of semantic memories in a 13-year-old child with profound episodic memory difficulties due to a metastatic intracranial germinoma (Pauly-Takacs, Moulin, & Estlin, 2010). However, as with the aforementioned, rigorous clinical trials have not yet been completed (Bharucha, 2009).

In addition to traditional cognitive therapies, the field of neural repair has made significant gains in the past decade in stroke research. Investigations have revealed that the adult brain is capable of forming new connections in the peri-infarct cortex, in projections from the brain opposite the stroke, and in signals for long-distance migration of new neurons to regions after the injury (Carmichael, 2001, 2010). In fact, approaches that specifically harness the molecular systems of memory and learning provide an avenue for stroke repair drugs.

Finally, it has been suggested that the next step for cognitive rehabilitation is to investigate the relationship between patient characteristics and treatment outcome (Cicerone et al., 2005). Future interventions are likely to become increasingly multimodal, including the use of instruction in traditional memory rehabilitation methods, the use of intelligent cognitive devices, and augmentation with a variety of pharmacologic agents. The efficacy of using these methods singularly or in combination will be based on successful functional outcome that must be examined beyond self-report, or through enhanced performance on psychometric indices. Generalization and maintenance must be systematically investigated in future studies. Neuroimaging has the potential to display a pattern of practice-related change in the form of: (a) activation decreases that are typically interpreted as increased neural efficiency; (b) activation increases, typically seen in practice-related expansions; and (c) reorganization of activation. Exploring the relationship between functional outcome and neural reorganization following intervention may help to expand our understanding of neural plasticity and functional reorganization across the developmental life span. Individual differences need to be investigated to determine who will most optimally benefit from a specific form of intervention. For example, individuals with a polymorphism related to higher levels of dopamine (Met allele) exhibit less task-specific brain activity during WM

compared to those with a polymorphism related to high levels of DA activity (Val allele). Individual differences in the catechol-O-methyltransferase (COMT) polymorphism affect levels of dopamine that in turn affect the efficiency of prefrontal cortical functioning. These individual differences are likely to interact with practice and treatment benefits (Kelly et al., 2006). WM is thought to be a basis for other cognitive control functions. As such, does targeting WM result in changes in flexible cognitive control? We will know in time.

## REFERENCES

- Acevedo, A., & Loewenstein, D. A. (2007). Nonpharmacological cognitive interventions in aging and dementia. *Journal of Geriatric Psychiatry and Neurology*, 20(4), 239–249.
- Alberini, C. M. (2005). Mechanisms of memory stabilization: Are consolidation and reconsolidation similar or distinct processes? *Trends in Neurosciences*, 28(1), 51–56.
- Alvarez, P., & Squire, L. R. (1994). Memory consolidation and the medial temporal lobe: A simple network model. *Proceedings of the National Academy of Sciences of the United States of America*, 91(15), 7041–7045.
- Anderson, J. R. (2005). *Cognitive psychology and its implications*. New York, NY: Worth.
- Areosa, A., & Sherriff, F. (2003). Memantine for dementia *Cochrane Database of Systemic Reviews*, (1), CD003154.
- Atkinson, R. C., & Shiffrin, R. M. (1968). Human memory: A proposed system and its control processes. In K. W. Spence & J. T. Spence (Eds.), *The psychology of learning and motivation* (Vol. 2). Austin, TX: Academic Press.
- Baddeley, A. (1992). Working memory. *Science*, 255(5044), 556–559.
- Baddeley, A. D. (2004). The psychology of memory. In A. D. Baddeley, M. Kopelman, & B. A. Wilson (Eds.), *The essential handbook of memory disorders for clinicians* (pp. 1–13). Hoboken, NJ: Wiley.
- Baddeley, A. (2000). The episodic buffer: a new component of working memory? *Trends in Cognitive Sciences*, 4(11), 417–423.
- Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. A. Bower (Ed.), *The psychology of learning and motivation*. New York, NY: Academic Press.
- Baddeley, A., & Wilson, B. A. (1994). When implicit learning fails: Amnesia and the problem of error elimination. *Neuropsychologia*, 32(1), 53–68.
- Bales, J. W., Wagner, A. K., Kline, A. E., & Dixon, C. E. (2009). Persistent cognitive dysfunction after traumatic brain injury: A dopamine hypothesis. *Neuroscience and Biobehavioral Reviews*, 33(7), 981–1003.
- Bayley, P. J., & Squire, L. R. (2005). Failure to acquire new semantic knowledge in patients with large medial temporal lobe lesions. *Hippocampus*, 15(2), 273–280.
- Bell, M. D., & Bryson, G. (2001). Work rehabilitation in schizophrenia: Does cognitive impairment limit improvement? *Schizophrenia Bulletin*, 27(2), 269–279.
- Benedict, R., Harris, A., Markow, T., McCormick, J., Nuechterlein, K., & Asarnow, R. (1994). Effects of attention training on information processing in schizophrenia. *Schizophrenia Bulletin*, 20, 537–546.
- Berg, I. J., Koning-Haanstra, M., & Deelman, B. G. (1991). Longterm effects of memory rehabilitation: A controlled study. *Neuropsychological Rehabilitation*, 1(2), 97–111.
- Bharucha, A. J., Anand, V., Forlizzi, J., Dew, M. A., Reynolds, C. F., Stevens, S., & Wactlar, H. (2009). Intelligent assistive technology applications to dementia care: Current capabilities, limitations, and future challenges. *American Journal of Geriatric Psychiatry*, 17(2), 88–104.
- Bigler, E. D. (1999). Neuroimaging in pediatric traumatic head injury: Diagnostic considerations and

- relationships to neurobehavioral outcome. *Journal of Head Trauma Rehabilitation*, 14(4), 406–123.
- Bigler, E. D., & Dodrill, C. B. (1997). Assessment of neuropsychological testing. *Neurology*, 49(4), 1180–1182.
- Boman, E., Enmarker, I., & Hygge, S. (2005). Strength of noise effects on memory as a function of noise source and age. *Noise & Health*, 7(27), 11–26.
- Boman, I. L., Lindstedt, M., Hemmingsson, H., & Bartfai, A. (2004). Cognitive training in home environment. *Brain Injury*, 18(10), 985–995.
- Bourgeois, C., & Tanchot, C. (2003). Mini-review CD4 T cells are required for CD8 T cell memory generation. *European Journal of Immunology*, 33(12), 3225–3231.
- Boxer, A. L., & Miller, B. L. (2005). Clinical features of frontotemporal dementia. *Alzheimer Disease and Associated Disorders*, 19(Suppl 1), S3–S6.
- Braak, H., & Braak, E. (1991). Neuropathological staging of Alzheimer-related changes. *Acta Neuropathologica*, 82(4), 239–259.
- Brett, A. W., & Laatsch, L. (1998). Cognitive rehabilitation therapy of braininjured students in a public high school setting. *Developmental Neurorehabilitation*, 2(1), 27–31.
- Broadbent, D. E. (1958). *Perception and communication*. New York, NY: Pergamon.
- Butters, N., Miliotis, P., Albert, M. S., & Sax, D. S. (1984). Memory assessment: Evidence of the heterogeneity of amnesic symptoms. *Advances in Clinical Neuropsychology*, 1, 127–159.
- Cabeza, R., Ciaramelli, E., Olson, I. R., & Moscovitch, M. (2008). The parietal cortex and episodic memory: An attentional account. *Nature Reviews Neuroscience*, 9(8), 613–625.
- Camp, C. J., Bird, M. J., & Cherry, K. E. (2000). Retrieval strategies as a rehabilitation aid for cognitive loss in pathological aging. In R. D. Hill, L. Backman, & A. S. Neely (Eds.), *Cognitive rehabilitation in old age* (pp. 224–248). New York: Oxford University Press.
- Camp, C. J., & Stevens, A. B. (1990). Spaced-retrieval: A memory intervention for dementia of the Alzheimer's type. *Clinical Gerontologist: Journal of Aging and Mental Health*, 10(1), 58–61.
- Camp, C. J., Foss, J. W., Stevens, A. B., & O'Hanlon, A. M. (1996). Improving prospective memory task performance in persons with Alzheimer's disease. In M. Brandimonte, G. Einstein, & M. A. McDaniel (Eds.), *Prospective memory: Theory and applications* (pp. 351–367). Mahwah, NJ: Lawrence Erlbaum Associates.
- Carmichael, S. T. (2010). Molecular mechanisms of neural repair after stroke. *Brain repair after stroke* (pp. 11–21). Cambridge, UK: Cambridge University Press.
- Carmichael, S. T., Wei, L., Rovainen, C. M., & Woolsey, T. A. (2001). New patterns of intracortical projections after focal cortical stroke. *Neurobiology of Disease*, 8(5), 910–922.
- Chew, E., & Zafonte, R. D. (2009). Pharmacological management of neurobehavioral disorders following traumatic brain injury—a state-of-the-art review. *Journal of Rehabilitation Research and Development*, 46(6), 851–879.
- Cicerone, K. D. (2002). Remediation of “working attention” in mild traumatic brain injury. *Brain Injury*, 16(3), 185–195.
- Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., Bergquist, T. F.,... Morse, P. A. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation*, 81(12), 1596–1615.
- Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., Kneipp, S.,... Catanese, J. (2005). Evidence-based cognitive rehabilitation: Updated review of the literature from 1998 through 2002. *Archives of Physical Medicine and Rehabilitation*, 86(8), 1681–1692.
- Clare, L., & Jones, R. S. (2008). Errorless learning in the rehabilitation of memory impairment: A critical review. *Neuropsychology Review*, 18(1), 1–23.
- Clare, L., Wilson, B. A., Breen, K., & Hodges, J. R. (1999). Errorless learning of face-name associations in early Alzheimer's disease. *Neurocase*, 5(1), 37–46.
- Clare, L., Wilson, B. A., Carter, G., Roth, I., & Hodges, J. R. (2002). Relearning face-name associations in early Alzheimer's disease. *Neuropsychology*, 16(4), 538–547.

- Cohen, M. (1997). *Children's Memory Scale*. San Antonio, TX: The Psychological Corporation.
- De Vreese, L. P., Neri, M., Fioravanti, M., Belloi, L., & Zanetti, O. (2001). Memory rehabilitation in Alzheimer's disease: A review of progress. *International Journal of Geriatric Psychiatry, 16*(8), 794–809.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, A. (1994). *The California Verbal Learning Test-Children's Version*. San Antonio, TX: The Psychological Corporation.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, A. (2000). *The California Verbal Learning Test-Second Edition*. San Antonio, TX: The Psychological Corporation.
- Desmond, D. W. (2004). The neuropsychology of vascular cognitive impairment: Is there a specific cognitive deficit? *Journal of the Neurological Sciences, 226*(1–2), 3–7.
- Dickerson, B. C., & Sperling, R. A. (2008). Functional abnormalities of the medial temporal lobe memory system in mild cognitive impairment and Alzheimer's disease: Insights from functional MRI studies. *Neuropsychologia, 46*(6), 1624–1635.
- Dickerson, B. C., & Sperling, R. A. (2009). Large-scale functional brain network abnormalities in Alzheimer's disease: Insights from functional neuroimaging. *Behavioural Neurology, 21*(1), 63–75.
- Dodrill, C. B. (1992). Neuropsychological aspects of epilepsy. *Psychiatric Clinics of North America, 15*(2), 383–394.
- Doornhein, K., & de Haan, E. H. F. (1998). Cognitive training for memory deficits in stroke patients. *Neuropsychological Rehabilitation, 8*(4), 393–400.
- Dudai, Y. (2000). Neurobiology. The shaky trace. *Nature, 406*(6797), 686–687.
- Dunn, J., & Clare, L. (2007). Learning face-name associations in early-stage dementia: Comparing the effects of errorless learning and effortful processing. *Neuropsychological Rehabilitation, 17*(6), 735–754.
- Einstein, G. O., & McDaniel, M. A. (1996). Retrieval processes in prospective memory: Theoretical approaches and some new empirical findings. In M. Brandimonte, G. Einstein, & M. McDaniel (Eds.), *Prospective memory: Theory and applications* (pp. 115–141). Mahwah, NJ: Lawrence Erlbaum Associates.
- Ellis, J. (1996). Prospective memory or the realization of delayed intentions: A conceptual framework for research. In M. A. Brandimonte, G. O. Einstein, & M. A. McDaniel (Eds.), *Prospective memory: Theory and applications* (pp. 1–22). Mahwah, NJ: Lawrence Erlbaum Associates.
- Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science, 11*, 19–23.
- Eguia, M. C., Rabinovich, M. I., & Abarbanel, H. D. (2000). Information transmission and recovery in neural communications channels. *Physical Review. E, Statistical Physics, Plasmas, Fluids, and Related Interdisciplinary Topics, 62*(5 Pt B), 7111–7122.
- Eslinger, P. J. (Ed.). (2002). *Neuropsychological interventions: Clinical research and practice* (No. 494). New York, NY: Guilford Press.
- Evans, J. J., Wilson, B. A., Needham, P., & Brentnall, S. (2003). Who makes good use of memory aids? Results of a survey of people with acquired brain injury. *Journal of the International Neuropsychological Society, 9*(6), 925–935.
- Fester, C. B., & Skinner, B. F. (1957). *Schedules of reinforcement*. New York: Appleton-Century-Crofts.
- Fish, J., Wilson, B. A., & Manly, T. (2010). The assessment and rehabilitation of prospective memory problems in people with neurological disorders: A review. *Neuropsychological Rehabilitation, 20*(2), 161–179.
- Fleming, J. M., Shum, D., Strong, J., & Lightbody, S. (2005). Prospective memory rehabilitation for adults with traumatic brain injury: A compensatory training programme. *Brain Injury, 19*(1), 1–10.
- Fletcher, J. M., Miner, M. E., & Ewing-Cobbs, L. I. N. D. A. (1987). Age and recovery from head injury in children: Developmental issues. In H. S. Levin, J. Grafman, & H. M. Eisenberg (Eds.), *Neurobehavioral recovery from head injury* (pp. 279–291). New York, NY: Oxford University Press.
- Folsom, J. C. (1968). Reality orientation for the elderly mental patient. *Journal of Geriatric Psychiatry, 1*(1), 1–10.

- 1(2), 291–307.
- Frankland, P. W., & Bontempi, B. (2005). The organization of recent and remote memories. *Nature Reviews. Neuroscience*, 6(2), 119–130.
- Frankland, P. W., O'Brien, C., Ohno, M., Kirkwood, A., & Silva, A. J. (2001). Alpha-CaMKII-dependent plasticity in the cortex is required for permanent memory. *Nature*, 411(6835), 309–313.
- Franzen, M. D., & Martin, N. (1996). Do people with knowledge fake better? *Applied Neuropsychology*, 3(2), 82–85.
- Friedman, J. I., Temporini, H., & Davis, K. L. (1999). Pharmacologic strategies for augmenting cognitive performance in schizophrenia. *Biological Psychiatry*, 45(1), 1–16.
- Gale, S. D., Johnson, S. C., Bigler, E. D., & Blatter, D. D. (1995). Nonspecific white matter degeneration following traumatic brain injury. *Journal of the International Neuropsychological Society*, 1(1), 17–28.
- Gasparrini, B. (1979). A treatment for memory problems in left hemisphere CVA patients. *Journal of Clinical and Experimental Neuropsychology*, 1(2), 137–150.
- Geusgens, C. A., Winkens, I., van Heugten, C. M., Jolles, J., & van den Heuvel, W. J. (2007). Occurrence and measurement of transfer in cognitive rehabilitation: A critical review. *Journal of Rehabilitation Medicine*, 39(6), 425–439.
- Gilboa, G., Chen, R., & Brenner, N. (2005). History-dependent multiple-time-scale dynamics in a single-neuron model. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 25(28), 6479–6489.
- Gleissner, U., Helmstaedter, C., Quiske, A., & Elger, C. E. (1998). The performance-complaint relationship in patients with epilepsy: A matter of daily demands? *Epilepsy Research*, 32(3), 401–409.
- Glisky, E. L., & Schacter, D. L. (1988). Longterm retention of computer learning by patients with memory disorders. *Neuropsychologia*, 26(1), 173–178.
- Glisky, E. L., Schacter, D. L., & Tulving, E. (1986a). Computer learning by memory-impaired patients: Acquisition and retention of complex knowledge. *Neuropsychologia*, 24(3), 313–328.
- Glisky, E. L., Schacter, D. L., & Tulving, E. (1986b). Learning and retention of computer-related vocabulary in memory-impaired patients: Method of vanishing cues. *Journal of Clinical and Experimental Neuropsychology*, 8(3), 292–312.
- Gold, P. E. (2008). Protein synthesis inhibition and memory: Formation vs amnesia. *Neurobiology of Learning and Memory*, 89(3), 201–211.
- Graf, P., & Schacter, D. L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. *Journal of Experimental Psychology. Learning, Memory, and Cognition*, 11(3), 501–518.
- Graham, D. I., McIntosh, T. K., Maxwell, W. L., & Nicoll, J. A. (2000). Recent advances in neurotrauma. *Journal of Neuropathology and Experimental Neurology*, 59(8), 641–651.
- Harris, J. E. (1984). Remembering to do things: A forgotten topic. In J. E. Harris & P.E. Morris, *Everyday memory: Actions and absentmindedness* (pp. 71–92). London: Academic Press.
- He, H., Dong, W., & Huang, F. (2010). Anti-amyloidogenic and anti-apoptotic role of melatonin in Alzheimer disease. *Current Neuropharmacology*, 8(3), 211–217.
- Hebb, D. O. (1949). *The organization of behavior: A neuropsychological theory*. New York, NY: Psychology Press.
- Helmick, K., Members of Consensus Conference (2010). Cognitive rehabilitation for military personnel with mild traumatic brain injury and chronic postconcussional disorder: Results of April 2009 consensus conference. *NeuroRehabilitation*, 26(3), 239–255.
- Hermann, B. P., Seidenberg, M., & Bell, B. (2002). The neurodevelopmental impact of childhood onset temporal lobe epilepsy on brain structure and function and the risk of progressive cognitive effects. *Progress in Brain Research*, 135, 429–438.
- Hof, P. R., & Morrison, J. H. (1999). Quantitative analysis of a vulnerable subset of pyramidal neurons in Alzheimer's disease: II. Primary and secondary visual cortex. *Journal of Comparative Neurology*, 301, 55–64.

- Holloway, M. (2003). The mutable brain. *Scientific American*, 289, 79–85.
- Hooft, I. V., Andersson, K., Bergman, B., Sejersen, T., Von Wendt, L., & Bartfai, A. (2005). Beneficial effect from a cognitive training programme on children with acquired brain injuries demonstrated in a controlled study. *Brain Injury*, 19(7), 511–518.
- Hopper, T., Mahendra, N., Kim, E., Azuma, T., Bayles, K. A., Cleary, S. J., & Tomoeda, C. K. (2005). Evidence-based practice recommendations for working with individuals with dementia: Spaced-retrieval training. *Journal of Medical Speech Language Pathology*, 13(4), xxvii–xxxiv.
- Hyman, B. T., Van Hoesen, G. W., Damasio, A. R., & Barnes, C. L. (1984). Alzheimer's disease: Cell-specific pathology isolates the hippocampal formation. *Science (New York, N.Y.)*, 225(4667), 1168–1170.
- Izquierdo, I., Vianna, M. R., Izquierdo, L. A., Barros, D. M., Szapiro, G., Coitinho, A. S., . . . Medina, J. H. (2002). Memory retrieval and its lasting consequences. *Neurotoxicity Research*, 4(5–6), 573–593.
- Jankowitz, B. T., & Adelson, P. D. (2006). Pediatric traumatic brain injury: Past, present and future. *Developmental Neuroscience*, 28(4–5), 264–275.
- Kalla, T., Downes, J. J., & vann de Broek, M. (2001). The preexposure technique: Enhancing the effects of errorless learning in the acquisition of face–name associations. *Neuropsychological Rehabilitation*, 11(1), 1–16.
- Kapur, N., Glisky, E. L., & Wilson, B. A. (2004). External memory aids and computers in memory rehabilitation. In A. D. Baddeley, M. Kopelman, & B. A. Wilson (Eds.), *The essential handbook of memory disorders for clinicians* (pp. 301–328). New York: Wiley.
- Kasai, M., Meguro, K., Hashimoto, R., Ishizaki, J., Yamadori, A., & Mori, E. (2006). Nonverbal learning is impaired in very mild Alzheimer's disease (CDR 0.5): Normative data from the learning version of the Rey-Osterrieth Complex Figure Test. *Psychiatry and Clinical Neurosciences*, 60(2), 139–146.
- Kasai, H., Fukuda, M., Watanabe, S., Hayashi-Takagi, A., & Noguchi, J. (2010). Structural dynamics of dendritic spines in memory and cognition. *Trends in Neurosciences*, 33(3), 121–129.
- Kaschel, R., Della Sala, S., Cantagallo, A., Fahlböck, A., Laaksonen, R., & Kazen, M. (2002). Imagery mnemonics for the rehabilitation of memory: A randomised group controlled trial. *Neuropsychological Rehabilitation*, 12(2), 127–153.
- Kelly, C., Foxe, J. J., & Garavan, H. (2006). Patterns of normal human brain plasticity after practice and their implications for neurorehabilitation. *Archives of Physical Medicine and Rehabilitation*, 87(12 Suppl 2), S20–S29.
- Kennedy, M. R., Coelho, C., Turkstra, L., Ylvisaker, M., Sohlberg, M., Yorkston, K., . . . Kan, P. F. (2008). Intervention for executive functions after traumatic brain injury: A systematic review, meta-analysis and clinical recommendations. *Neuropsychological Rehabilitation*, 18(3), 257–299.
- Kern, R. S., Wallace, C. J., Hellman, S. G., Womack, L. M., & Green, M. F. (1996). A training procedure for remediating WCS deficits in chronic psychotic patients: An adaptation of errorless learning principles. *Journal of Psychiatric Research*, 30(4), 283–294.
- Kessels, R. P., & de Haan, E. H. (2003). Implicit learning in memory rehabilitation: A meta-analysis on errorless learning and vanishing cues methods. *Journal of Clinical and Experimental Neuropsychology*, 25(6), 805–814.
- Kime, S. K., Lamb, D. G., & Wilson, B. A. (1996). Use of a comprehensive programme of external cueing to enhance procedural memory in a patient with dense amnesia. *Brain Injury*, 10(1), 17–25.
- Kirsch, N. L., Levine, S. P., Lajiness-O'Neill, R., & Schnyder, M. (1992). Computer-assisted interactive task guidance: Facilitating the performance of a simulated vocational task. *Journal of Head Trauma Rehabilitation*, 7, 13–25.
- Kurtz, M. M., Moberg, P. J., Gur, R. C., & Gur, R. E. (2001). Approaches to cognitive remediation of neuropsychological deficits in schizophrenia: A review and meta-analysis. *Neuropsychology Review*, 11(4), 197–210.
- Laatsch, L., Harrington, D., Hotz, G., Marcantuono, J., Mozzoni, M. P., Walsh, V., & Hersey, K. P. (2007). An evidence-based review of cognitive and behavioral rehabilitation treatment studies in children with

- acquired brain injury. *Journal of Head Trauma Rehabilitation*, 22(4), 248–256.
- Landauer, T. K., & Bjork, R. A. (1978). Optimum rehearsal patterns and name learning. *Practical Aspects of Memory*, 1, 625–632.
- Lashley, K. S. (1930). Basic neural mechanisms in behavior. *Psychological Review*, 37(1), 1.
- Lawson, M. J., & Rice, D. N. (1989). Effects of training in use of executive strategies on a verbal memory problem resulting from closed head injury. *Journal of Clinical and Experimental Neuropsychology*, 11(6), 842–854.
- Lechner, H. A., Squire, L. R., & Byrne, J. H. (1999). 100 years of consolidation-remembering Müller and Pilzecker. *Learning & Memory*, 6(2), 77–87.
- Levin, H. S., Wilde, E. A., Chu, Z., Yallampalli, R., Hanten, G. R., Li, X.,... Hunter, J. V. (2008). Diffusion tensor imaging in relation to cognitive and functional outcome of traumatic brain injury in children. *Journal of Head Trauma Rehabilitation*, 23(4), 197–208.
- Levine, B., Schweizer, T. A., O'Connor, C., Turner, G., Gillingham, S., Stuss, D. T., Manly, T., & Robertson, I. H. (2011). Rehabilitation of executive functioning in patients with frontal lobe brain damage with goal management training. *Frontiers in Human Neuroscience*, 5, 9. doi: 10.3389/fnhum.2011.00009.
- Limond, J., & Leeke, R. (2005). Practitioner review: Cognitive rehabilitation for children with acquired brain injury. *Journal of Child Psychology and Psychiatry*, 46(4), 339–352.
- Lisman, J., & Morris, R. G. (2001). Memory. Why is the cortex a slow learner? *Nature*, 411(6835), 248–249.
- Loftus, E. F. (2005). Planting misinformation in the human mind: A 30-year investigation of the malleability of memory. *Learning & Memory*, 12(4), 361–366.
- Loftus, E. F., & Davis, D. (2006). Recovered memories. *Annual Review of Clinical Psychology*, 2, 469–498.
- Mackenzie, I. R., & Rademakers, R. (2007). The molecular genetics and neuropathology of frontotemporal lobar degeneration: recent developments. *Neurogenetics*, 8(4), 237–248.
- Majid, M. J., Lincoln, N. B., & Weyman, N. (2007). Cognitive rehabilitation for memory deficits following stroke. *Cochrane Database of Systematic Reviews*, (3), CD002293.
- Manly, T., Robertson, I. H., & Kapur, N. (2002). Paradoxes in neurorehabilitation. In N. Kapur, O. Sacks, A. Pascual-Leone, & V. Ramachandran (Eds.) *The paradoxical brain*. (ch. 4, pp. 74–93). Cambridge, UK: Cambridge University Press.
- McCauley, S. R., & Levin, H. S. (2004). Prospective memory in pediatric traumatic brain injury: A preliminary study. *Developmental Neuropsychology*, 25(1–2), 5–20.
- McClelland, J. L., McNaughton, B. L., & O'Reilly, R. C. (1995). Why there are complementary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. *Psychological Review*, 102(3), 419–457.
- McGaugh, J. L. (1966). Time-dependent processes in memory storage. *Science*, 153(3742), 1351–1358.
- McGaugh, J. L. (2000). Memory—A century of consolidation. *Science*, 287(5451), 248–251.
- McHugh, P. R., & Folstein, M. F. (1975). Psychiatric syndromes of Huntington's chorea: A clinical and phenomenological study. *Psychiatric Aspects of Neurologic Disease*, 1, 267–286.
- Medina, J. H., Bekinschtein, P., Cammarota, M., & Izquierdo, I. (2008). Do memories consolidate to persist or do they persist to consolidate? *Behavioural Brain Research*, 192(1), 61–69.
- Medalia, A., Revheim, N., & Casey, M. (2000). Remediation of memory disorders in schizophrenia. *Psychological Medicine*, 30(6), 1451–1459.
- Meeter, M., & Murre, J. M. (2004). Consolidation of longterm memory: Evidence and alternatives. *Psychological Bulletin*, 130(6), 843–857.
- Melton, A., & Bourgeois, M. (2005). Training compensatory memory strategies via the telephone for persons with TBI. *Aphasiology*, 19(3–5), 353–364.
- Michaud, L. J. (1995). Evaluating efficacy of rehabilitation after pediatric traumatic brain injury. In S. H. Broman & M. E. Michel (Eds.), *Traumatic head injury in children* (pp. 247–257). New York, NY:

- Oxford University Press.
- Milner, B. (1965). Visually-guided maze learning in man: Effects of bilateral hippocampal, bilateral frontal, and unilateral cerebral lesions. *Neuropsychologia*, 3(4), 317–338.
- Milner, B. (1968). Visual recognition and recall after right temporal-lobe excision in man. *Neuropsychologia*, 6(3), 191–209.
- Moscovitch, M., Chein, J. M., Talmi, D., & Cohn, M. (2007). Learning and memory. In B. J. Baars & N. M. Gage (Eds.), *Cognition, brain and consciousness*. London, UK: Elsevier.
- Moscovitch, M., Nadel, L., Winocur, G., Gilboa, A., & Rosenbaum, R. S. (2006). The cognitive neuroscience of remote episodic, semantic and spatial memory. *Current Opinion in Neurobiology*, 16(2), 179–190.
- Moscovitch, M., Rosenbaum, R. S., Gilboa, A., Addis, D. R., Westmacott, R., Grady, C., . . . Nadel, L. (2005). Functional neuroanatomy of remote episodic, semantic and spatial memory: A unified account based on multiple trace theory. *Journal of Anatomy*, 207(1), 35–66.
- Mottram, L., & Donders, J. (2006). Cluster subtypes on the California verbal learning test-children's version after pediatric traumatic brain injury. *Developmental Neuropsychology*, 30(3), 865–883.
- Muir, K. W., & Teal, P. A. (2005). Why have neuro-protectants failed? Lessons learned from stroke trials. *Journal of Neurology*, 252(9), 1011–1020.
- Müller, G. E., & Pilzecker, A. (1900). Experimentelle Beiträge zur Lehre von Gedächtnis. *Zeitschrift für Psychologie, Ergänzungsband*, 1, 1–300.
- Murray, E. A., & Bussey, T. J. (2001). Consolidation and the medial temporal lobe revisited: Methodological considerations. *Hippocampus*, 11(1), 1–7.
- Nadel, L., & Moscovitch, M. (1997). Memory consolidation, retrograde amnesia and the hippocampal complex. *Current Opinion in Neurobiology*, 7(2), 217–227.
- Nadel, L., Samsonovich, A., Ryan, L., & Moscovitch, M. (2000). Multiple trace theory of human memory: Computational, neuroimaging, and neuropsychological results. *Hippocampus*, 10(4), 352–368.
- Nayak, T. K., & Sikdar, S. K. (2007). Time-dependent molecular memory in single voltage-gated sodium channel. *Journal of Membrane Biology*, 219(1–3), 19–36.
- Oberg, L., & Turkstra, L. S. (1998). Use of elaborative encoding to facilitate verbal learning after adolescent traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 13(3), 44–62.
- Osterrieth, P. A. (1944). Le test de copie d'une figure complexe: Contribution à l'étude de la perception et de la mémoire. *Archives de Psychologie*, 30, 286–356.
- Oyegbile, T. O., Dow, C., Jones, J., Bell, B., Rutecki, P., Sheth, R., . . . Hermann, B. P. (2004). The nature and course of neuropsychological morbidity in chronic temporal lobe epilepsy. *Neurology*, 62(10), 1736–1742.
- Page, M., Wilson, B. A., Shiel, A., Carter, G., & Norris, D. (2006). What is the locus of the errorless-learning advantage? *Neuropsychologia*, 44(1), 90–100.
- Pauly-Takacs, K., Moulin, C. J., & Estlin, E. J. (2012). Benefits and limitations of errorless learning after surviving pediatric brain tumors: A case study. *Journal of Clinical and Experimental Neuropsychology*, 34(6), 654–666.
- Pilling, S., Bebbington, P., Kuipers, E., Garety, P., Geddes, J., Orbach, G., & Morgan, C. (2002). Psychological treatments in schizophrenia: I. Meta-analysis of family intervention and cognitive behaviour therapy. *Psychological Medicine*, 32(5), 763–782.
- Ponds, R. W., & Hendriks, M. (2006). Cognitive rehabilitation of memory problems in patients with epilepsy. *Seizure*, 15(4), 267–273.
- Quayhagen, M. P., & Quayhagen, M. (1989). Differential effects of family-based strategies on Alzheimer's disease. *Gerontologist*, 29(2), 150–155.
- Quayhagen, M. P., Quayhagen, M., Corbeil, R. R., Roth, P. A., & Rodgers, J. A. (1995). A dyadic remediation program for care recipients with dementia. *Nursing Research*, 44(3), 153–159.
- Rabinovici, G. D., & Miller, B. L. (2010). Frontotemporal lobar degeneration: Epidemiology, pathophysiology, diagnosis and management. *CNS Drugs*, 24(5), 375–398.



- Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique [Psychological examination of traumatic encephalopathy]. *Archives de Psychologie*, 28, 286–340.
- Rey, A. (1958). *L'examen clinique en psychologie*. Paris: Presse Universitaire de France.
- Reynolds, C., & Voress, J. K. (2007). *The test of Memory and Learning-Second Edition (TOMAL-2)*. Austin, TX: Pro-Ed.
- Reynolds, C. R., & Bigler, E. D. (1994). *Test of memory and learning. Examiner's manual*. Austin, TX: Pro-Ed.
- Robinson, F. P. (1970). *Effective study*. New York, NY: Harper & Row.
- Rohling, M. L., Faust, M. E., Beverly, B., & Demakis, G. (2009). Effectiveness of cognitive rehabilitation following acquired brain injury: A meta-analytic re-examination of Cicerone et al.'s (2000, 2005) systematic reviews. *Neuropsychology*, 23(1), 20–39.
- Ruis, C., & Kessels, R. P. (2005). Effects of errorless and errorful face-name associative learning in moderate to severe dementia. *Aging Clinical and Experimental Research*, 17(6), 514–517.
- Rutland-Brown, W., Langlois, J. A., Thomas, K. E., & Xi, Y. L. (2006). Incidence of traumatic brain injury in the United States, 2003. *Journal of Head Trauma Rehabilitation*, 21(6), 544–548.
- Salmon, D. P. (2000). Disorders of memory in Alzheimer's disease. In L. S. Cermak (Ed.), *Memory and its disorders. Handbook of neuropsychology* (2nd edn, Vol. 2, pp. 155–195). Amsterdam, Netherlands: Elsevier Science.
- Salmon, D. P., Bondi, M. W. (1999). Neuropsychology of Alzheimer's disease. In R. D. Terry, R. Katzman, K. L. Bick, & S. S. Sisodia (Eds.), *Alzheimer disease* (2nd edn, pp. 39–56). Philadelphia, PA: Lippincott Williams & Wilkins.
- Saykin, A. J., Shtasel, D. L., Gur, R. E., Kester, D. B., Mozley, L. H., Stafiniak, P., & Gur, R. C. (1994). Neuropsychological deficits in neuroleptic naive patients with first-episode schizophrenia. *Archives of General Psychiatry*, 51(2), 124–131.
- Saykin, A. J., Wishart, H. A., Rabin, L. A., Flashman, L. A., McHugh, T. L., Mamourian, A. C., & Santulli, R. B. (2004). Cholinergic enhancement of frontal lobe activity in mild cognitive impairment. *Brain: A Journal of Neurology*, 127(Pt 7), 1574–1583.
- Schacter, D. L., & Glisky, E. L. (1986). Memory remediation: Restoration, alleviation, and the acquisition of domain-specific knowledge. In B. Uzzell & Y. Gross (Eds.), *Clinical neuropsychology of intervention* (pp. 257–282). Boston, MA: Martinus Nijhoff.
- Schacter, D. L., & Tulving, E., (1994). Memory systems 1994. *What are the memory systems of 1994?* (pp. 1–38). Cambridge, MA: MIT Press.
- Schalén, W., Hansson, L., Nordström, G., & Nordström, C. H. (1994). Psychosocial outcome 5–8 years after severe traumatic brain lesions and the impact of rehabilitation services. *Brain Injury*, 8(1), 49–64.
- Sheslow, D., & Adams, W. (2003). *Wide Range Assessment of Memory and Learning-Second Edition (WRAML-2)*. Wilmington, DE: Wide Range.
- Sivan, A. B. (1992). *Benton visual retention test*. San Antonio, TX: Psychological Corporation.
- Slomine, B., & Locascio, G. (2009). Cognitive rehabilitation for children with acquired brain injury. *Developmental Disabilities Research Reviews*, 15(2), 133–143.
- Sohlberg, M. M., & Mateer, C. A. (1989). *Introduction to cognitive rehabilitation: Theory and practice*. New York, NY: Guilford Press.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach*. New York, NY: Guilford Press.
- Sohlberg, M. M., Ehlhardt, L., & Kennedy, M. (2005, November). Instructional techniques in cognitive rehabilitation: A preliminary report. *Seminars in Speech and Language*, 26(4), 268–279.
- Sohlberg, M. M., Kennedy, M. R. T., Avery, J., Coelho, C., Turkstra, L., Ylvisaker, M., & Yorkston, K. (2007). Evidence-based practice for the use of external aids as a memory compensation technique. *Journal of Medical Speech Language Pathology*, 15(1), xv–li.
- Sohlberg, M. M., White, O., Evans, E., & Mateer, C. (1992). Background and initial case studies into the effects of prospective memory training. *Brain Injury*, 6(2), 129–138.
- Sperling, R. A., Dickerson, B. C., Pihlajamaki, M., Vannini, P., LaViolette, P. S., Vitolo, O. V., . . .

- Johnson, K. A. (2010). Functional alterations in memory networks in early Alzheimer's disease. *Neuromolecular Medicine*, 12(1), 27–43.
- Spreen, O., & Strauss, E. (1998). Memory. In *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed., pp. 260–422). New York, NY: Oxford University Press.
- Squire, L. R. (1987). *Memory and brain*. New York, NY: Oxford University Press.
- Squire, L. R. (2004). Memory systems of the brain: A brief history and current perspective. *Neurobiology of Learning and Memory*, 82(3), 171–177.
- Squire, L. R., & Alvarez, P. (1995). Retrograde amnesia and memory consolidation: A neurobiological perspective. *Current Opinion in Neurobiology*, 5(2), 169–177.
- Squire, L. R., & Bayley, P. J. (2007). The neuroscience of remote memory. *Current Opinion in Neurobiology*, 17(2), 185–196.
- Stark, C., Stark, S., & Gordon, B. (2005). New semantic learning and generalization in a patient with amnesia. *Neuropsychology*, 19(2), 139–151.
- Starr, J. M., Loeffler, B., Abousleiman, Y., Simonotto, E., Marshall, I., Goddard, N., & Wardlaw, J. M. (2005). Episodic and semantic memory tasks activate different brain regions in Alzheimer disease. *Neurology*, 65(2), 266–269.
- Stavitsky, K., Brickman, A. M., Scarneas, N., Torgan, R. L., Tang, M. X., Albert, M., . . . Stern, Y. (2006). The progression of cognition, psychiatric symptoms, and functional abilities in dementia with Lewy bodies and Alzheimer disease. *Archives of Neurology*, 63(10), 1450–1456.
- Sutherland, R. J., Lehmann, H., Spanswick, S. C., Sparks, F. T., & Melvin, N. R. (2006). Growth points in research on memory and hippocampus. *Canadian Journal of Experimental Psychology = Revue Canadienne de Psychologie Expérimentale*, 60(2), 166–174.
- Terrace, H. S. (1963). Errorless transfer of a discrimination across two continua. *Journal of the Experimental Analysis of Behavior*, 6, 223–232.
- Thone, A. I. T., & Glisky, E. L. (1995). Learning of name-face associations in memory impaired patients: A comparison of different memory training procedures. *Journal of the International Neuropsychological Society*, 1, 29–38.
- Tompkins, L. M., Goldman, R. S., & Axelrod, B. N. (1995). Modifiability of neuropsychological dysfunction in schizophrenia. *Biological Psychiatry*, 38(2), 105–111.
- Tsaousides, T., & Gordon, W. A. (2009). Cognitive rehabilitation following traumatic brain injury: Assessment to treatment. *Mount Sinai Journal of Medicine*, 76(2), 173–181.
- Tse, D., Langston, R. F., Kakeyama, M., Bethus, I., Spooner, P. A., Wood, E. R., . . . Morris, R. G. (2007). Schemas and memory consolidation. *Science*, 316(5821), 76–82.
- Tulving, E. (1972). Episodic and semantic memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory* (pp. 381–402). New York, NY: Academic Press.
- Tulving, E., & Schacter, D. L. (1990). Priming and human memory systems. *Science*, 247(4940), 301–306.
- Vitali, P., Migliaccio, R., Agosta, F., Rosen, H. J., & Geschwind, M. D. (2008). Neuroimaging in dementia. *Seminars in Neurology*, 28(4), 467–483.
- Waber, D. P., & Holmes, J. M. (1986). Assessing children's memory productions of the Rey-Osterrieth Complex Figure. *Journal of Clinical and Experimental Neuropsychology*, 8(5), 563–580.
- Wade, T. K., & Troy, J. C. (2001). Mobile phones as a new memory aid: A preliminary investigation using case studies. *Brain Injury*, 15(4), 305–320.
- Wagner, A. D. (2002). Cognitive control and episodic memory: Contributions from prefrontal cortex. In L. R. Squire & D. L. Schacter (Eds.), *Neuropsychology of memory* (3rd edn, pp. 174–192). New York, NY: Guilford Press.
- Ward, H., Shum, D., Wallace, G., & Boon, J. (2002). Pediatric traumatic brain injury and procedural memory. *Journal of Clinical and Experimental Neuropsychology*, 24(4), 458–470.
- Ward, H., Shum, D., McKinlay, L., Baker, S., & Wallace, G. (2007). Prospective memory and pediatric traumatic brain injury: Effects of cognitive demand. *Child Neuropsychology*, 13(3), 219–239.
- Waugh, N. C., & Norman, D. A. (1965). Primary memory. *Psychological Review*, 72, 89–104.

- Wechsler, D., Holdnack, J. A., & Drozdick, L. W. (2009). *Wechsler Memory Scale: Fourth Edition*. San Antonio, TX: Pearson.
- Wilson, B. A., Baddeley, A. D., Evans, J., & Shiel, A. (1994). Errorless learning in the rehabilitation of memory impaired people. *Neuropsychological Rehabilitation*, 4, 307–326.
- Wilson, B. A., Greenfield, E., Clare, L., Baddeley, A., Cockburn, J., Watson, P., Tate, R., Sopena, S., & Nannery, R. (2008). *The Rivermead Behavioural Memory Test—Third Edition (RBMT-3)*. San Antonio, TX: Pearson Assessment.
- Wilson, B. A. (1992). Memory therapy in practice. In B. A. Wilson & N. Moffat (Eds.), *Clinical management of memory problems* (2nd edn, pp. 120–153). London: Chapman & Hall.
- Wilson, B. A. (1997). Cognitive rehabilitation: How it is and how it might be. *Journal of the International Neuropsychological Society*, 3(5), 487–496.
- Wilson, B. A., Emslie, H., Foley, J., Shiel, A., Watson, P., Hawkins, K., Groot, Y., & Evans, J. J. (2005). *The Cambridge Prospective Memory Test (CAMPRMPT)*. London: Harcourt Assessment.
- Wilson, B. A. (2009). *Memory rehabilitation: Integrating theory and practice*. New York, NY: Guilford Press.
- Wilson, B. A., Emslie, H. C., Quirk, K., & Evans, J. J. (2001). Reducing everyday memory and planning problems by means of a paging system: A randomised control crossover study. *Journal of Neurology, Neurosurgery, and Psychiatry*, 70(4), 477–482.
- Wilson, B. A., Emslie, H., Evans, J. J., Quirk, K., Watson, P., & Fish, J. (2009). The NeuroPage system for children and adolescents with neurological deficits. *Developmental Neurorehabilitation*, 12(6), 421–426.
- Wilson, B., Cockburn, J., & Baddeley, A. D. (1985). *The Rivermead Behavioural Test*. Bury St. Edmunds, Suffolk, England: Thames Valley Test.
- Wilson, C., & Robertson, I. H. (1992). A home-based intervention for attentional slips during reading following head injury: A single case study. *Neuropsychological Rehabilitation*, 2(3), 193–205.
- Winocur, G., Craik, F. I., Levine, B., Robertson, I. H., Binns, M. A., Alexander, M.,... Stuss, D. T. (2007). Cognitive rehabilitation in the elderly: Overview and future directions. *Journal of the International Neuropsychological Society*, 13(1), 166–171.
- Wykes, T., Reeder, C., Corner, J., Williams, C., & Everitt, B. (1999). The effects of neurocognitive remediation on executive processing in patients with schizophrenia. *Schizophrenia Bulletin*, 25(2), 291–307.
- Yesavage, J., Hoblyn, J., Friedman, L., Mumenthaler, M., Schneider, B., & O'Hara, R. (2007). Should one use medications in combination with cognitive training? If so, which ones? *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, 62(Special issue 1), 11–18.

# Rehabilitation of Visuospatial Deficits

*Justin H. Ory, Mandi Wilkes-Musso, Alyse Ann Barker,  
Daniel Proto, and Wm. Drew Gouvier*

## SEQUELAE AND NEUROPATHOLOGY: WHAT ARE VISUOSPATIAL DEFICITS?

Visuospatial deficits exist on a spectrum ranging from purely sensory based to purely cognitive in nature (Sacks, 1970) and deficits may be innate or acquired. Although not as obvious or as likely to adversely impact social interaction skills as aphasic language disorders, the presence of visuospatial deficits can markedly impact a person's diagnostic status and prognosis for recovery. Depending on the specific symptom manifestations, the presence of certain visuospatial deficits in particular is often used to satisfy the requirement for checking potential agnosia in diagnosing dementia. Their presence often markedly impairs an individual's ability to ambulate and navigate in his environment, as well as limit abilities to function safely and effectively in basic and advanced activities of daily living. Visuospatial deficits include partial or hemifield sensory losses often known as visual field cuts, which occur in the contralesional hemifield (Cohen, 2011). Hemifield sensory distortions in the absence of field cuts have also been reported (Gouvier, Cottam, Webster, Beissel, & Woffard, 1984). Hemifield suppression (i.e., the tendency to consistently ignore or fail to respond to stimuli on one side of the body) is commonly observed in testing, and has been attributed to lesions in the visual association area (Mendoza, 2011). Attentional and hemiattentional disorders (e.g., failure to detect, respond to, or represent contralesional stimuli despite intact motor and sensory functioning)

can occur on either the right side or left side when stroke or injury damages the contralateral temporal lobe. However, hemineglect is most common after disruption of the right hemisphere and subsequent left hemineglect is more severe (Ogden, 1987). Finally, pure visual agnosia for specific objects, words, or letters (Sacks, 1970) is seen following right-lateralized or bilateral occipital and ventral temporal lobe areas, in the case of agnosia for faces, and left-lateralized lesions in occipito-temporal areas, for letter-word prosopagnosia (Righi & Tarr, 2011). These visuospatial deficits are rarely seen alone, but are often comorbid with other deficits, such as aphasia, apraxia, and memory and executive dysfunctions. Visuospatial deficits impact functioning in a number of domains, including activities of daily living such as dressing, grooming, ambulation, and household activities; reading and other skills necessary for work or school; and finally, patients with visuospatial deficits are at increased risk for sustaining further disabling injuries, such as falls and collisions as a direct result of their impairments.

Most patients demonstrating visuospatial deficits due to brain injury or stroke have diffuse damage to multiple cortical and subcortical regions of the brain, making it difficult to isolate regions responsible for specific deficits. However, the inferior parietal lobe (Driver & Vuilleumier, 2001), the parietal temporal junction (Milner & McIntosh, 2005), and the temporal-occipital junction (Lee et al., 2010) are commonly implicated cortical areas associated with visuospatial neglect. Additional research suggests that visuospatial neglect results from a “cortico-subcortical anatomical network” involving the putamen, pulvina, caudate nucleus, insula, and superior temporal gyrus (Karnath, Berger, Kuker, & Rorden, 2004; Karnath, Himmelbach, & Rorden, 2002). A frontoparietal network consisting of the intraparietal sulcus, superior parietal lobule, and the frontal eye fields has been suggested as the neuronal basis of visuospatial attention (Corbetta, Kincade, & Shulman, 2002; Corbetta, Patel, & Shulman, 2008) and plays a role in other partial field cuts as well. Lesions of the paraventricular white matter have also been associated with neglect (Verdon, Schwartz, Lovblad, Hauert, & Vuilleumier, 2010). Animal studies suggest that the superior colliculus may play a role in orienting behaviors resulting in visuospatial neglect due to hyperactivity and hypoactivity of the contralesional and ipsilesional hemispheres, respectively (Payne & Rushmore, 2004; Sprague, 1966).

It is widely accepted that attention, or lack of attention, plays a large role in visuospatial neglect. Posner and Petersen (1990) named three primary networks

involved in visuospatial attention: alerting, orienting, and executive control. Evidence has been found for deficits in ability to perceive (alert to) objects (Bonato, Priftis, Marenzi, Umiltà, & Zorzi, 2010) and disengage attention (orient) once fixated (Posner, Walker, Friedrich, & Rafal, 1984; Ptak, Schneider, Golay, & Muri, 2007). Other theories about visuospatial neglect focus on the role of spatial working memory (executive functioning). Deficits in spatial working memory are believed to be responsible for repeated visitation of previously addressed stimuli (Milner & McIntosh, 2005) and have been associated with lesions of the parietal and insular cortex (Pisella, Berberovic, & Mattingley, 2004; Rossit et al., 2009). Danckert and Ferber (2006) postulate that visual neglect of contralesional stimuli is best explained by a combination of deficits in spatial working memory and ipsilateral biases in spatial attention and exploratory motor control. Spatial working memory is particularly relevant in rehabilitation settings where patients who demonstrate some degree of mastery of compensatory strategies during rehabilitation exacerbate deficits in visuospatial neglect during activities of daily living due to increased attentional demands at home (Bonato et al., 2010).

It has been posited that the right hemisphere is capable of ipsilateral and contralateral allocation of visual attention, whereas the left hemisphere is solely responsible for directing attention to the contralateral visual field (Heilman's hemispatial theory; Heilman & Van Den Abell, 1980; Mesulam, 1981, 1999). One problem with this theory is that hemineglect is not an all-or-nothing phenomenon but typically presents as a graded neglect toward the contralateral visual field. To account for this, Kinsbourne (1977) proposed the interhemispheric competition theory, which suggests that both hemispheres play roles in visual attentional processing of contralateral visual fields but that a lesion in the right hemisphere results in loss of respective attention allocation and a gradient of impaired attentional functioning from the contralateral to the ipsilateral hemisphere. Data from healthy patients provide support for interhemispheric competition, that is, both hemispheres contribute to spatial attention over the visual field with biases toward the contralateral hemisphere. The hemispheres are balanced by neuronal mechanisms located in the right superior parietal lobe, left frontal eye field, and left posterior parietal cortex (Szczepanski, Konen, & Kastner, 2010).

A more complicated theory suggests that there are two relatively independent neural systems involved in visual attention representing both "top-down" (cognitive, endogenous, voluntary; dorsal, parietal, and frontal cortices)

and “bottom-up” (sensory, exogenous, automatic; temporoparietal and ventral frontal cortices) mechanisms (Corbetta & Shulman, 2002). Although both neural networks may contribute to visuospatial deficits, the bottom-up neural network is postulated to be lateralized to the right hemisphere and to result in the lion’s share of neglect symptoms, including target detection and orienting. It appears that neglect is related to automatic (bottom-up) attention shifting rather than a voluntary (top-down) act of biased attention distribution to the ipsilateral hemisphere (Bartolomeo & Chokron, 2002; Smania et al., 1998). Although intact in spatial neglect patients, voluntary attention is unable to compensate for deficits in automatic attention because the exogenous system supplies the endogenous system with incoming information (Natale, Posteraro, Prior, & Marzi, 2005). Other evidence suggests that both exogenous and endogenous orienting to stimuli may be controlled by the same mechanisms located in the frontal and parietal lobes (Peelen, Heslenfeld, & Theeuwes, 2004).

Although there are multiple theories about attentional mechanisms involved in visuospatial neglect and concomitant neurological underpinnings, no answer is complete; there is likely a complex interaction between brain structures and attentional deficits. As knowledge about these components is expanded, assessment and rehabilitation strategies will be refined and improved.

### **ASSESSMENT: HOW CAN WE TELL THE DEFICITS ARE THERE?**

Visuospatial deficits have been identified as a symptom of various neurological conditions and insults, including subcortical dementia, traumatic brain injury, and stroke. Visuospatial deficits can be evaluated with neuropsychological, analog reality (curriculum-based assessment), and in vivo assessment strategies. A fundamental step in the rehabilitation of visuospatial deficits is proper assessment to aid in treatment planning and track rehabilitative progress using a process that Anastasi (1954) referred to as testing the limits, and subsequently refined and incorporated into the “process approach” (Kaplan, 1988) to neuropsychological assessment. The goal is to first identify deficit areas and then identify the props and supports that can allow the patient to successfully function in the area of impairment either through skill enhancement or via compensation strategies.

Various neuropsychological measures assess visuospatial skills in children, adolescents, and adults. These measures include but are not limited to the *Benton Judgment of Line Orientation Test (JLOT)*, *Visual Object and Space Perception Battery*, *Differential Ability Scales-Second Edition spatial subtests*, *NEPSY-II*

visual spatial processing subtests, Wechsler Adult Intelligence Scales-Fourth Edition Perceptual Reasoning subtests, Wechsler Intelligence Scale for Children-Fourth Edition Perceptual Reasoning subtests, Stanford-Binet Intelligence Scales-Fifth Edition Visual Spatial Processing subtest, Woodcock Johnson-III Test of Cognitive Abilities Spatial Relations and Picture Recognition subtests, Test of Visual Motor Integration, Rey-Osterrieth Complex Figure Test, Benton Visual Form Discrimination Test, Hooper Visual Organization Test, Clock Drawing Test, and Bender-Gestalt II. Also, [Kerkhoff and Marquardt \(1998\)](#) have developed a computerized test of visuospatial abilities in which various components of the task can be modified (e.g., stimuli size, stimuli color). Visuospatial neglect is a specific type of visuospatial deficit that can be evaluated using traditional neuropsychological measures, as well as specialized measures, including letter cancellation and line bisection tasks, visual scanning for proximal and distant stimuli, and somatosensory awareness and localization activities ([New York University, Institution of Rehabilitation Medicine, 1983](#)).

Analog reality or curriculum-based assessment measures of visuospatial neglect include more ecologically valid behavioral tasks, such as picture scanning, reading, and writing tasks. *The Behavioural Inattention Test (BIT; Wilson, Cockburn, & Halligan, 1987)* is a measure used to assess visuospatial neglect, which employs behavioral tasks. The Two Part Picture test has also been used to detect neglect, and it has demonstrated greater sensitivity than the picture scanning tasks included in the BIT ([Brunila, Jalas, Lindell, Tenovuo, & Hamalainen, 2003](#); [Lindell et al., 2007](#)). Computerized assessment tasks employing virtual environments have also been developed to assess visual spatial deficits ([Allahyar & Hunt, 2003](#); [Stirk & Foreman, 2005](#)).

Visuospatial functioning and rehabilitative outcomes can also be examined through neuroimaging ([Laatsch, Jobe, Sychra, Lin, & Blend, 1997](#); [Laatsch, Thulborn, Krisky, Shobat, & Sweeney, 2004](#)). In a study by [Thiyagesh et al. \(2009\)](#), individuals with Alzheimer's disease looking at visuospatial stimuli demonstrated less activation of the superior parietal lobe and parieto-occipital cortex, but more activation of the inferior parietal lobe than control participants. Using single photon emission computerized tomography, researchers found increases in cerebral blood flow following cognitive rehabilitation therapy for visuospatial neglect ([Pantano et al., 1992](#)).

The visuospatial domain encompasses various skills, including visual-perceptual and visual-constructional abilities. Because the visuospatial domain is multifaceted, often different measures are needed to comprehensively assess for



deficits. Also, clinicians should consider that many measures that assess visuospatial skills also require that other cognitive skills, such as executive functioning, are intact for tasks to be successfully completed. When deficits in the visuospatial domain are observed, identifying the “communalities among the failed tasks” can elucidate the cause of task failures (Gouvier, O’Jile, & Ryan, 1998, p. 188). Determining the reason for task failure is an essential step in identifying cognitive interventions, which will target neuropsychological and functional impairments in the client’s everyday environment.

The specifics of the referral question guide the selection of measures that are most appropriate for assessment. Because performance on many neuropsychological measures can be influenced by practice effects (Miller et al., 2009), clinicians should use caution when choosing measures to track rehabilitative progress to ensure the tracking measures do not confound or bias the outcome measures used to evaluate pre/post follow-up changes in performance. Erez, Katz, Ring, and Soroker (2009) developed Visual Spatial Search Task (VISSTA), a computerized program used to evaluate visual spatial attention and diagnose unilateral spatial neglect. The authors suggest the newly developed computerized test may be less susceptible to practice effects and thus more appropriate for tracking rehabilitative progress than traditional paper-and-pencil tests. Many traditional measures are available in parallel forms, and the use of curriculum-based measures, derived by the clinician for the specific target behaviors under investigation are the ideal measures for tracking incremental changes in performance without compromising core neuropsychological measures.

### **Role of Assessment in Visuospatial Rehabilitation**

Using an understanding of the brain–behavior relationship, neuropsychologists can identify which brain areas are most likely affected by the lesions, as well as any deficits that may impact the effectiveness of a specific rehabilitation plan. Subsequently, an approach to rehabilitation can be chosen—which utilizes the more intact mechanism while also taking advantage of the patient’s strengths. Neuropsychological assessment also provides quantitative information to guide hypotheses about cognitive status and capacity, thus providing baseline data whereby recovery progress may be tracked (Shutter & Jallo, 1998).

## **REMEDICATION STRATEGIES: PARTICULAR FOCUS ON HEMINEGLECT AND RELATED PROBLEMS**

One way to categorize the theoretical approaches to the rehabilitation of visuospatial neglect is by their modality—whether environmental, behavioral, or cognitive compensation (in which the goal is to develop habits or strategies to cover areas of weakness), or restoration (in which the goal is to improve deficits). Within each modality, there are techniques to address visuospatial deficits through either top-down (e.g., deliberate, conscious), or bottom-up (e.g., which rely more on attenuating automatic processes) approaches (Corbetta & Shulman, 2002). Many of these techniques may address both apraxic and agnosic issues together as they often share a common underpinning. The reader is reminded that apparent apraxic or agnosic problems might have a basis in sensory or motor deficit. A sensory-based apraxia appears when the patient's movements are disrupted because of poor positional sensors advising them about the movement and limb position coordinates, whereas a motor-based agnosia might appear when the patient's movement disorder prevents him or her from bringing the appropriate sensory receptors to bear on the relevant stimuli to be apprehended and responded to.

## COMPENSATION

A number of studies suggest that at least some degree of spontaneous recovery may reflect compensation rather than a genuine reduction in underlying spatial distortion (Bartolomeo, 2000; Goodale, Milner, Jakobson, & Carey, 1990). Rehabilitation techniques intended to facilitate this compensation (whether behaviorally or cognitively) have been supported for both top-down and bottom-up modalities.

### Environmental Compensation

The traditional model of environmental engineering has focused on teaching the patient to capitalize on areas of strength for functional, everyday activities, adding specific rehabilitative training exercises to promote reacquisition and refinement of weak skills. When visuospatial skills are the target, and the deficient skills are preserved sufficiently, a shift in training strategies may be appropriate, with the majority of training activities focused solely on the restoration of lost function via forced disuse of the intact side (LeVere, 1975). The success of this approach suggests that behavioral restitution is the result of continued and refined operation of spared neural mechanisms rather than a functional reorganization of neural tissue. Taking LeVere's findings a step further, Taub (2004) found that brain plasticity was greater for recovery than is

generally recognized. Other environmental compensation techniques function by placing some of the workload from deficit systems onto external stimuli, such as the use of posted reminders to aid memory and orientation cues for visual deficits. Such techniques are usually developed with input from an occupational therapist as part of an interdisciplinary team and are applied on a patient-specific basis.

### **Behavioral Compensation**

Behavioral compensatory strategies for remediating hemispatial or hemiinattentional deficits involve training a patient habitually to scan from left to right, using intact right peripheral vision to identify salient and biologically significant stimuli (Manly, 2002). This visual scanning training (VST) is a top-down approach that is typically accomplished by using a highly salient cue (e.g., a red bookmark or verbalization: “anchor left”) as an additional “anchor” at the left of the sheet—and systematically fading this cue as performance improves. When used while reading, this technique can increase the number of words the patient detects (Diller & Weinberg, 1977). However, Lawson (1962) describes how a patient’s improved reading did not generalize well—even between different editions of the same book, proving once again the behaviorists’ mantra about the need to program generalization into any training program (Stokes & Baer, 1977).

For some neglect patients, simply using their left hand to perform a task produces a marked reduction in neglect (Luaute, Halligan, Rode, Rossetti, & Boisson, 2006). Limb activation training (LAT) is a bottom-up strategy that uses the left hand in the left visual space as a left-oriented visuosomatic cue. Theoretically, LAT is based on the hypothesis that engaging the contralesional limb results in increased activation of the damaged hemisphere, which is otherwise inhibited by overactivation in the contralesional lobe. Findings support this hypothesis as the benefits appear even when the movements are outside the visual field, indicating that they are not due to simple visual cuing (Manly, 2002). Although LAT has produced substantial improvements in single case studies, many patients with visual neglect also have some degree of hemiplegia and display a bias toward using their right limbs over their left. Although at least one randomized clinical trial has found reductions of hemiplegia using LAT (Robertson, McMillan, MacLeod, Edgeworth, & Brock, 2002), other studies have found no effect (Cubelli, Paganelli, Achilli, & Pedrizzi, 1999). Thus, it is questionable whether LAT is an effective therapeutic option for this population.

Assorted physical interventions have been attempted over the years, including eye patching and the use of prism spectacles, which shift left-sided visual information to be inputted into the favored left hemiretina. Rossi, Kheyfets, and Reding (1990) performed a 39-subject randomized clinical trial using such glasses and reported significant improvements on five neurological assessments of visual perception after 4 weeks when compared to controls, but there were no significant differences in an assessment of activities of daily living.

### Neuropsychological Compensation

Sustained attention training (SAT) and computerized alertness training are both rooted in connectionist models of neuropsychological function and are hypothesized to work by strengthening the connections between spared neurons in the right hemisphere to accommodate for the lesioned area (Proto, Pella, Hill, & Gouvier, 2009; Thimm, Fink, Kust, Karbe, & Sturm, 2006). Eight patients with left spatial neglect improved in their performance on cancellation tasks following treatment with SAT (Robertson, Tegner, Tham, Lo, & Nimmo-Smith, 1995). Similarly, Kim *et al.* (2007) found that virtual-reality (VR) based computerized alertness programs produced improvements on a variety of tasks, including several functional skills, within the VR environment.

The phenomenon of blindsight and Zihl's (1981) demonstration of scotoma reduction work most effectively when the sensory modality merely shows bilateral simultaneous suppression error and not full exclusion errors when unilateral stimulation only is used. One example of how to use this for rehabilitating visuospatial neglect is through errorless trial training. A patient with visual suppressions but not exclusions can benefit from this protocol in which they practice bilateral detection by doing modified simultaneous bilateral stimulation trials as follows: Prepare patient for bilateral visual trials. Present stimulus to affected half-field 1 second before presenting to the intact field. As client becomes increasingly adept at detecting, fade the time lag between the presentations to  $\frac{1}{2}$  second,  $\frac{1}{4}$  second, and 0 second and then beyond the "ordinary requirements of the real world" by presenting to the strong field first and lag the presentation to the affected field.

### Cognitive Compensation

A more cognitive approach to remediating visuospatial deficits is to teach the client rules to apply to guide his use of behavioral scanning. One way to

accomplish this is to teach clients verbal compensations to remind them of their hemiperceptual deficits. For example, following a stroke, Mr. L. M., a retired sailor, leaned way to his right, but felt like he was straight. Consequently, he could not bear weight on his left leg and was unable to walk. When Mr. L. M. was taught to remind himself that the feeling of “listing to port” (i.e., leaning to the left) was “normal trim” (i.e., upright posture), he could keep his left leg perpendicular to the ground and was able to walk normally. Beyond verbal strategies, the client’s mindful awareness is sometimes enough to compensate for visual deficits (e.g., a client judges distance on his right correctly, but what looks like a foot on the left, is less than an inch. If he wants to clear the door by a foot, he has to know that it should look like 5 feet); (Gouvier, Bua, Blanton, & Urey, 1987).

Feedback training is a bottom-up cognitive technique that aims to reduce anosognosia, a condition in which patients with a disability are unaware of the existence of their disability, which often is often reported among spatial neglect patients. Because the patient does not recognize her visuospatial neglect, anosognosia can affect a patient’s motivation for treatment if not overcome (Luaute et al., 2006). Results from clinical trials have been generally favorable. Soderback, Bengtsson, Ginsburg, and Ekholm (1992) video-recorded four patients to provide feedback on their neglect behavior prior to their participation in training, with subsequent improvements on a cancellation task and three household tasks. The largest and most recent published feedback training controlled trial used a challenging visuomotor modality, which the author acknowledges may have contributed to his null treatment effect (Harvey, Hood, North, & Robertson, 2003).

Experimentations with sensory stimulation as a rehabilitation technique for lateral visual neglect have included caloric vestibular stimulation (Rubens, 1985), optokinetic stimulus (OKS; Pizzamiglio, Frasca, Guariglia, Incoccia, & Antonucci, 1990), and vibration (Karnath, Christ, & Hartje, 1993) or electrical stimulation to the neck muscles (Rossetti & Rode, 2002) or extracranially over the parietal cortex (Luaute et al., 2006). Caloric stimulation of the horizontal ear canal, usually with cold water, produced a substantial reduction of neglect as measured by segment cancelation and reading tests. However, the effects only lasted from 6 to 10 minutes. OKS has demonstrated decreased leftward neglect when the stimulus moves toward the left and an increased rightward bias when the stimulus moves to the right (Pizzamiglio, 2003).

Similarly, contralesional posterior neck muscle vibration (NMV) has

shown short-term benefits in a variety of areas ([Kerkhoff, 2003](#)), and these benefits have been shown to persist at least 2 weeks after therapy, although additional studies are necessary to further support these findings. Contralateral transcutaneous electrical nerve stimulation (TENS), in which electrical stimulation is applied to the neck, and repetitive transcranial magnetic stimulation (rTMS), in which electrical stimulation is applied extracranially over the parietal cortex, have been demonstrated to improve a variety of visuospatial deficits ([Brighina, et al., 2003](#); [Vallar, Rusconi, & Bernardini, 1996](#)). In one recent randomized clinical trial, OKS and TENS have proved more effective than controls at producing improvements on standard neglect tests, as well as measures of everyday performance. However, the TENS group experienced a drop in improvement on the neglect tests at 1-week retest when compared to the OKS group. Both groups showed improvement in reading and writing ability at the end of therapy and at the 1-week follow up when compared to the control group ([Schroder, Wist, & Homberg, 2008](#)).

Another technique to reduce left visual field neglect is eye patching. Monocular eye patching (i.e., patching the ipsilesional eye) was theorized to decrease superior collicular activity in the contralesional hemisphere and was first identified by [Sprague \(1966\)](#) while studying cats. [Barrett, Crucian, Beversdorf, and Heilman \(2001\)](#) reported that monocular eye patching may worsen left-field neglect in some instances. Their conclusions, which supported the Sprague effect, were based on a single case study and conflict with some prior research, which showed that monocular eye patching of the left eye could encourage right-eye dominance.

Right half-field eye patching is a newer adaptation of eye patching in which particular hemifields are blocked. [Tsang, Sze, and Fong \(2009\)](#) investigated the efficacy of right half-field eye patching in a randomized clinical trial. Right half-field eye patching involves blocking the right visual field from both eyes. They found that patients treated with right half-field eye patching displayed significantly more improvement on the BIT and the Functional Independence Measure (FIM) after 4 weeks. However, the evaluation period may have been too close to the original neurological insult (admittance vs. 4 weeks post stroke), and potential benefits in impairment tests were not confirmed by improvements in function. Overall, eye patching continues to receive support for its efficacy, but its functional effectiveness requires additional support, and the actual mechanism of change needs to be identified.

Prismatic adaptation therapy (PA) is a corrective technique that uses

prisms. It is based on the theory of neuronal plasticity. The prisms shift the visual field laterally so that visual targets appear at a displaced position. Training then involves perceptual–motor pointing tasks. Initial pointing movements approximate the virtual position of the target, whereas each subsequent pointing movement corrects for error and ensures that subjects can readily point to the real target position. Following the removal of the prisms, further pointing movements are required to reinforce the sensory motor adaptation and obtain more robust negative aftereffects (Weiner, Hallett, & Funkenstein, 1983). These negative aftereffects are the result of a compensatory shift in the direction opposite to the visual shift produced by the prisms. Thus, right PA with leftward negative aftereffect would improve left-spatial neglect. In a seven-sample clinical trial, improvements over a control sample were achieved on such tasks as line bisection, cancellation, naming, and general visual perception for six out of seven patients (with the seventh patient failing to achieve adaptation). Interestingly, all six patients who showed improvement at the end of the 2 weeks of therapy had maintained those improvements at the 5-week follow-up, indicating potential long-term usefulness of this therapy technique (Frassinetti, Angeli, Meneghello, Avanzi, & Ladavas, 2002). However, generalizability to activities of daily living is yet to be demonstrated in depth and, although promising, the long-term benefits require a great deal more confirmatory research. For example, Striemer and Danckert (2010) propose an alternative explanation for the effects of PA whereby many of the beneficial effects arise through the influence of adaptation on circuits in the dorsal visual stream controlling attention and visuomotor behaviors.

## Restoration

Beyond compensation strategies, rehabilitation techniques that encourage new neuronal associations have also been developed. Such therapies are most effective when the damage to the brain is relatively localized rather than diffuse, allowing intact neurons to form associations with surrounding healthy neurons, bypassing the damaged areas. This can be accomplished through a more cognitive sensory training or through pharmacotherapy with neurotransmitters associated with the damaged pathways.

### Cognitive Restoration

One theory as to why lesions of the right hemisphere produce more severe contralateral biases is that they disrupt the bilateral cerebral activity mediating

the body-centered representations of the body and of the external space (Pizzamiglio, 2003) resulting in a rightward egocentric bias of body and space perception. This bias can be partially overcome by techniques, which shift attention to the left. Although hemispheric visual neglect can operate on an object's coordinates (i.e., left half of object ignored), for many patients, the body's midline appears to be a crucial marker of what is left and what is right. Karnath, Schenkel, and Fischer (1991) demonstrated that simply instructing patients to rotate their trunk 15° so that more of the visual scene falls on the right side of the body promises to significantly reduce neglect. However, neglect patients are unlikely to spontaneously make such rotations, preferring to orient the eyes, head, and trunk to the right.

Mental imagery training is a top-down cognitive rehabilitation technique, which is inspired by the representational theory of left-spatial neglect. In one study, Smania, Bazoli, Piva, and Guidetti (1997) used mental imagery exercises with two right-brain-lesioned patients and showed positive results, including improvements on six neuropsychological tests of left-field neglect with improvements remaining stable over 6 months indicating the possibility of long-term improvements. However, this was a two-subject study and mental imagery training requires more research to determine if these results are typical or exceptional.

Space remapping training is a relatively new bottom-up cognitive therapy that originated from experimental clinical trials. It was observed that an elongated stick could produce a virtual extension of body and space perception, resulting in a remapping of far space as near space. Space remapping therapy attempts to generalize this effect toward the neglected left space. In a clinical trial, patients with left-space neglect were instructed to grasp for objects in their right visual field while simultaneously observing a virtual hand grasp a virtual object in their left visual field using VR (Castiello, Lusher, Burton, Glover, & Disler, 2004). The trials revealed significantly improved grasping accuracy for the left side of the visual space following space remapping.

“Virtual reality” is a term used to describe highly immersive three-dimensional environments, typically created using realistic computer-generated imaging (CGI), as well as the equipment and technology required to produce them (Heim, 1994). VR training has been used in a variety of fields in which in vivo practice is hazardous or expensive, including flight simulators (Platt, Dahn, & Amburn, 1991) and laparoscopic surgery (Seymour et al., 2002). Over the past 10 years, there has been a push to incorporate VR into patient assessment



protocols, VR classrooms have been developed to assess for attention deficit hyperactivity disorder (ADHD; [Rizzo et al., 2004](#)), and driving simulators have been developed to assess for visuospatial deficits (Cherniack, in press). These simulators have been used to train skills that otherwise would be dangerous to train in vivo, such as street crossing, and the outcomes have been promising regarding generalizability to the real world ([Kim et al., 2004, 2007](#)). However, VR-based assessments and treatments are prohibitively more expensive, complex, and difficult to use than the current range of assessment and rehabilitation protocols, and virtual environments require extensive programming and are therefore difficult to tailor to a client's specific needs.

## Pharmacotherapy

Pharmacological rehabilitation for left-visuospatial neglect follows one of the two theories ([Greene, Robertson, Gill, & Bellgrove, 2010](#)). The earlier of the two therapies, treatment with dopamine agonists, was proposed in 1987 when Fleet, Valenstein, Watson, and Heilman observed that the brain structures related to left-visuospatial neglect were all related by a common dopaminergic bond. In a four-case study, [Mukand et al. \(2001\)](#) examined the performance of four female stroke patients with left-visuospatial neglect using an abbreviated version of the BIT and FIM before and after 1 week of treatment with carbidopa L-dopa. After 1 week, three out of four of patients displayed significant improvements in their modified BIT scores and their functional status on the FIM. However, not every study has been so promising. [Grujic et al. \(1998\)](#) found that treatment with bromocriptine, a dopamine agonist, actually reoriented visual exploration away from the neglected visual hemispace. Despite mixed results in case studies, there have been no rigorous clinical trials evaluating the effectiveness of dopamine agonist therapy for left-visuospatial neglect, and more conclusive results are needed before its potential can be truly understood.

The more recent development in pharmacotherapy for visuospatial neglect is based on the hypothesis that norepinephrine modulates attentional processes, which may interact with spatial neglect ([Greene et al., 2010](#)). As such, this top-down treatment entails using a noradrenergic agonist, Guafacine. To date, there have been only limited-sample case studies testing the effectiveness of Guafacine. [Singh-Curry, Malhotra, Farmer, and Husain \(2011\)](#) examined the effects of Guafacine on attentional ability in a 38-year-old patient with acute disseminated encephalomyelitis (ADEM), with subsequent improvement reported in attentional abilities relevant to spatial neglect, including line

bisection, and to a lesser extent, line cancellation. Another case study demonstrated improvements on writing tasks as well as increased visual exploration for two patients whose lesion spared the dorso-lateral prefrontal cortex ([Malhotra, Parton, Greenwood, & Husain, 2006](#)), whereas a third patient whose lesion included damage to the dorso-lateral prefrontal cortex did not improve after treatment. As with treatment with dopamine agonists, there is insufficient empirical data to support the use of Guafacine to treat left-visuospatial neglect. However, the results thus far look promising.

### **Rehabilitation of Children**

The “Kennard Principle” holds that the younger a patient is at the time of initial brain injury, the better the likely outcome. However, recent investigation has demonstrated that this principle may be oversimplified. Recent work by [Laurent-Vannier, Brugel, and Agostini \(2000\)](#) indicates that under certain conditions (e.g., if the lesions are diffuse) recovery is no greater in children versus adults, or younger children versus older children. In addition, sequelae may be present even when intelligence is spared. However, despite these findings, children continue to be underrepresented in the visuospatial neglect research ([Dennis, 2010](#)).

Despite this neglect in the treatment literature, children do suffer from visuospatial neglect. [Laurent-Vannier, Pradat-Diehl, Chevignard, Abada, and De Agostini \(2003\)](#) assessed 12 children in an inpatient pediatric rehabilitation unit specializing in acquired brain lesions. Of the 12 children assessed, all exhibited either left (nine cases) or right (three cases) visuospatial neglect. [Brown, Gouvier, and Blanchard-Fields \(1990\)](#) discuss three categories of cognitive therapy, which have been supported for children through empirical literature. They are cognitive modeling, problem-solving, and self-instructional training. These cognitive therapies have not been evaluated for the treatment of visuospatial neglect in children, but may be used to orient children with visuospatial neglect to therapy, as well as to assist with implementing individualized functional strategies to help young patients regain a greater degree of independence. However, research needs to be done to evaluate the effectiveness of intervention for visuospatial neglect with children.

### **FUTURE DIRECTIONS**

Researchers and clinicians know more about visual neglect now than they did just a half-decade ago. The implementation of novel technologies in imaging and

statistical analysis has informed newly posited theories regarding the phenomenon of neglect. These theories have, in turn, influenced the development of new treatments. However, with so many varying ideas about underlying pathology, proper assessment methods, and appropriate interventions, much work remains to be done to bring greater clarity and consensus to this topic. Of paramount importance are the refinement of neuropathological disease models and the identification of critical impacted neurophysiological mechanisms. Without this information, the development of comprehensive neglect theories and accompanying interventions remains elusive.

Much like the progression of the concept of classifying “organic versus inorganic” factors early in the history of neuropsychology, knowledge of visuospatial neglect now appears to be nearing a level of sophistication sufficient to warrant greater diagnostic classification specificity. Multiple processes and structures thought to be impacted in visual neglect have been implicated and studied (e.g., top-down/bottom-up and endogenous/exogenous processes, temporoparietal and occipitotemporal junctions, visuoperception and spatial working memory, and frontoparietal control systems; [Bonato et al., 2010](#); [Corbetta & Shulman, 2002](#); [Danckert & Ferber, 2006](#); [Lee et al., 2010](#); [Milner & McIntosh, 2005](#); [Natale et al., 2005](#); [Szczepanski et al., 2010](#)). Now researchers must synthesize these various pieces into larger scale disease models that allow for identification of which processes and structures might be implicated in the case of each individual patient. Promising work by [Verdon and colleagues \(2010\)](#) has moved in this direction, identifying three specific neuropsychological deficit factors present in visual neglect and then mapping the underlying neuroanatomical correlates of each. Such multilevel, cohesive efforts are the necessary next step in theory development. This work allows for greater refinement in the assessment and classification of visual neglect based on the presence or absence of identified deficit factors, which are each indicative of unique neuropathologies. Clinicians can then better choose and implement appropriate interventions based on which factors are, and are not, affected.

The development of such appropriate interventions is also an area requiring further study. Although various techniques have been studied (e.g., TENS, rTMS, OKS, PAT, VST, half-field eye patching), few of these measures have been examined in the context of large-scale randomized controlled clinical trials (RCCTs). Additionally, although improvements on specific neuropsychological assessment measures have been observed, generalization of these deficits to activities of daily living has often been minimal or nonexistent (e.g., [Frassinetti](#)

et al., 2002; Lawson, 1962; Rossi et al., 1990). Finally, few studies have evaluated long-term retention of observed improvements. Encouragingly, recent research has begun addressing these absences—Kim *et al.* (2007) found some transfer of VR alertness training to functional skills; Tsang and colleagues (2009) used randomized clinical trial methods to evaluate right-hemifield eye patching and noted improvement in functional skills; and Luukkainen-Markkula, Tarkka, Pitkänen, Sivenius, and Hämäläinen (2009) also used randomized clinical trial methodology to compare gains from left-arm activation and VST at up to 6 months postintervention. As these and other treatments develop, the need for RCCTs complete with long-term efficacy and outcome measures becomes more appropriate and necessary. Additionally, the potential benefits of case-study meta-analyses in rehabilitation research, such as those discussed by Barrett and Gonzalez-Rothi (2005), have gone largely unexplored in visual neglect research. Given the large number of single-case and small-group studies already available, rigorous meta-analyses of these data could be just as informative as RCCT results while requiring a smaller investment of experimental resources, and are thus equally crucial.

Beyond patient-centered interventions, the recruitment of family members in the rehabilitation process is another area that requires more research. The severity of visual neglect has been shown to predict scores on measures of family burden (Buxbaum et al., 2004), which could lead to greater levels of caregiver and patient distress. Conversely, early research has tentatively found that family member involvement in the rehabilitation process is related to improved performance on tests of inattention/neglect and mobility (Osawa & Maeshima, 2010). In light of these encouraging results, much more work is needed to examine optimal treatment roles and distress coping strategies for patients' families during the rehabilitation process.

Despite this current scarcity of comprehensive multilevel disease models and refined assessment and classification systems, and the need for further development of long-term RCCT-based intervention outcome evaluations involving patients in families, the trends in the field of visual neglect are promising. As noted, recent work has increasingly addressed these areas. Optimistic results suggest that affected patients and their loved ones may soon begin realizing the benefits of this research, including greater levels of functional independence and significantly improved quality of life.

## REFERENCES

- Anastasi, A. (1954). *Psychological testing*. New York, NY: Macmillan.
- Allahyar, M., & Hunt, E. (2003). The assessment of spatial orientation using virtual reality techniques. *International Journal of Testing*, 3(3), 263–275.
- Barrett, A. M., Crucian, G. P., Beversdorf, D. Q., & Heilman, K. M. (2001). Monocular patching may worsen sensory-attentional neglect: A case report. *Archives of Physical Medicine and Rehabilitation*, 82(4), 516–518.
- Barrett, A. M., & Gonzalez-Rothi, L. J. (2005). Theoretical bases of neuropsychological interventions. In P. J. Elsinger (Ed.), *Neuropsychological interventions: Clinical research and practice* (pp. 16–37). New York, NY: Guilford Press.
- Bartolomeo, P. (2000). Inhibitory processes and spatial bias after right hemisphere damage. *Neuropsychological Rehabilitation*, 10, 511–526.
- Bartolomeo, P., & Chokron, S. (2002). Orienting of attention in left unilateral neglect. *Neuroscience and Biobehavioral Reviews*, 26(2), 217–234.
- Bonato, M., Priftis, K., Marenzi, R., Umiltà, C., & Zorzi, M. (2010). Increased attentional demands impair contralesional space awareness following stroke. *Neuropsychologia*, 48(13), 3934–3940.
- Brighina, F., Bisiach, E., Oliveri, M., Piazza, A., La Bua, V., Daniele, O., & Fierro, B. (2003). 1 Hz repetitive transcranial magnetic stimulation of the unaffected hemisphere ameliorates contralesional visuospatial neglect in humans. *Neuroscience Letters*, 336(2), 131–133.
- Brown, L., Gouvier, W. D., & Blanchard-Fields, F. (1990). Cognitive interventions across the life span. In J. M. Williams (Ed.), *Cognitive neuropsychology* (pp. 133–153). New York, NY: Springer Publishing Company.
- Brunila, T., Jalas, M., Lindell, J. A., Tenovuo, O., & Hamalainen, H. (2003). The two part picture in detection of visuospatial neglect. *Clinical Neuropsychologist*, 17(1), 45–53.
- Buxbaum, L. J., Ferraro, M. K., Veramonti, T., Farne, A., Whyte, J., Ladavas, E., . . . & Coslett, H. B. (2004). Hemispatial neglect: Subtypes, neuroanatomy, and disability. *Neurology*, 62(5), 749–756.
- Castiello, U., Lusher, D., Burton, C., Glover, S., & Disler, P. (2004). Improving left hemispatial neglect using virtual reality. *Neurology*, 62(11), 1958–1962.
- Cherniack, E. P. (in press). Not just fun and games: Applications of virtual reality in the identification and rehabilitation of cognitive disorders of the elderly. *Disability and rehabilitation: Assistive technology*, Retrieved from <http://informahealthcare.com/doi/pdf/10.3109/17483107.2010.542570>
- Cohen, R. A. (2011). Quadrantanopia. In J. S. Kreutzer, J. DeLuca, & B. Caplan, (Eds.), *Encyclopedia of clinical neuropsychology* (pp. 2095–2097), New York, NY: Springer Publishing Company.
- Corbetta, M., Kincade, J. M., & Shulman, G. L. (2002). Neural systems for visual orienting and their relationships to spatial working memory. *Journal of Cognitive Neuroscience*, 14(3), 508–523.
- Corbetta, M., Patel, G., & Shulman, G. L. (2008). The reorienting system of the human brain: from environment to theory of mind. *Neuron*, 58(3), 306–324.
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews. Neuroscience*, 3(3), 201–215.
- Cubelli, R., Paganelli, N., Achilli, D., & Pedrizzi, S. (1999). Is one hand always better than two? A replication study. *Neurocase*, 5, 143–151.
- Danckert, J., & Ferber, S. (2006). Revisiting unilateral neglect. *Neuropsychologia*, 44(6), 987–1006.
- Dennis, M. (2010). Margaret Kennard (1899–1975): not a “principle” of brain plasticity but a founding mother of developmental neuropsychology. *Cortex*, 46(8), 1043–1059.
- Diller, L., & Weinberg, J. (1977). Hemiinattention in rehabilitation: The evolution of a rational remediation program. *Advances in Neurology*, 18, 63–82.
- Driver, J., & Vuilleumier, P. (2001). Perceptual awareness and its loss in unilateral neglect and extinction. *Cognition*, 79(1–2), 39–88.
- Erez, A. B., Katz, N., Ring, H., & Soroker, N. (2009). Assessment of spatial neglect using computerised feature and conjunction visual search tasks. *Neuropsychological Rehabilitation*, 19(5), 677–695.
- Fleet, W. S., Valenstein, E., Watson, R. T., & Heilman, K. M. (1987). Dopamine agonist therapy for neglect

- in humans. *Neurology*, 60, 1826–1829.
- Frassinetti, F., Angeli, V., Meneghello, F., Avanzi, S., & Ladavas, E. (2002). Long-lasting amelioration of visuospatial neglect by prism adaptation. *Brain*, 125, 608–623.
- Goodale, M. A., Milner, A. D., Jakobson, L. S., & Carey, D. P. (1990). Kinematic analysis of limb movements in neuropsychological research: subtle deficits and recovery of function. *Canadian Journal of Psychology*, 44(2), 180–195.
- Gouvier, W. D., Bua, B. G., Blanton, P. D., & Urey, J. R. (1987). Behavioral changes following visual scanning training: Observations of five cases. *International Journal of Neuropsychology*, 9, 74–80.
- Gouvier, W. D., Cottam, G., Webster, J. S., Beissel, G. F., & Woffard, J. D. (1984). Behavioral interventions with stroke patients for improving wheelchair navigation. *International Journal of Clinical Neuropsychology*, 6, 186–190.
- Gouvier, W. D., O’Jile, J. R., & Ryan, L. M. (1998). Neuropsychological assessment for planning cognitive interventions. In G. Goldstein & S. R. Beers (Eds.), *Rehabilitation* (pp. 181–199). New York, NY: Plenum.
- Greene, C. M., Robertson, I. H., Gill, M., & Bellgrove, M. A. (2010). Dopaminergic genotype influences spatial bias in healthy adults. *Neuropsychologia*, 48(9), 2458–2464.
- Grujic, Z., Mapstone, M., Gitelman, D. R., Johnson, N., Weintraub, S., Hays, A., . . . Mesulam, M. M. (1998). Dopamine agonists reorient visual exploration away from the neglected hemispace. *Neurology*, 51(5), 1395–1398.
- Harvey, M., Hood, B., North, A., & Robertson, I. H. (2003). The effects of visuomotor feedback training on the recovery of hemispatial neglect symptoms: assessment of a 2-week and follow-up intervention. *Neuropsychologia*, 41(8), 886–893.
- Heilman, K. M., & Van Den Abell, T. (1980). Right hemisphere dominance for attention: the mechanism underlying hemispheric asymmetries of inattention (neglect). *Neurology*, 30(3), 327–330.
- Heim, M. (1994). *The metaphysics of virtual reality*. New York, NY: Oxford.
- Kaplan, E. (1988). A process approach to neuropsychological assessment. In T. Boll & B. K. Bryant (Eds.), *Clinical neuropsychology and brain function: Research, measurement, and practice* (pp. 125–168). Washington, DC: American Psychological Association.
- Karnath, H. O., Berger, F. M., Küker, W., & Rorden, C. (2004). The anatomy of spatial neglect based on voxel-wise statistical analysis: a study of 140 patients. *Cerebral cortex*, 14(10), 1164–1172.
- Karnath, H. O., Christ, K., & Hartje, W. (1993). Decrease of contralateral neglect by neck muscle vibration and spatial orientation of trunk midline. *Brain*, 116, 383–396.
- Karnath, H. O., Himmelbach, M., & Rorden, C. (2002). The subcortical anatomy of human spatial neglect: Putamen, caudate nucleus and pulvinar. *Brain*, 125(Pt 2), 350–360.
- Karnath, H. O., Schenkel, P., & Fischer, B. (1991). Trunk orientation as the determining factor of the ‘contralateral’ deficit in the neglect syndrome and as the physical anchor of the internal representation of body orientation in space. *Brain*, 114, 1997–2014.
- Kerkhoff, G. (2003). Modulation and rehabilitation of spatial neglect by sensory stimulation. *Progress in Brain Research*, 142, 257–271.
- Kerkhoff, G., & Marquardt, C. (1998). Standardised analysis of visual-spatial perception after brain damage. *Neuropsychological Rehabilitation*, 8(2), 171–189.
- Kim, J., Kim, K., Kim, D. Y., Chang, W. H., Park, C. I., Ohn, S. H., . . . Kim, S. I. (2007). Virtual environment training system for rehabilitation of stroke patients with unilateral neglect: crossing the virtual street. *Cyberpsychology & Behavior*, 10(1), 7–15.
- Kim, K., Kim, J., Ku, J., Kim, D. Y., Chang, W. H., Shin, D. I., . . . Kim, S. I. (2004). A virtual reality assessment and training system for unilateral neglect. *Cyberpsychology & Behavior*, 7(6), 742–749.
- Kinsbourne, M. (1977). Hemineglect and hemisphere rivalry. *Advances in Neurology*, 18, 41–49.
- Laatsch, L., Jobe, T., Sychra, J., Lin, Q., & Blend, M. (1997). Impact of cognitive rehabilitation therapy on neuropsychological impairments as measured by brain perfusion SPECT: A longitudinal study. *Brain Injury*, 11(12), 851–863.

- Laatsch, L. K., Thulborn, K. R., Krisky, C. M., Shobat, D. M., & Sweeney, J. A. (2004). Investigating the neurobiological basis of cognitive rehabilitation therapy with fMRI. *Brain Injury, 18*(10), 957–974.
- Laurent-Vannier, A., Brugel, D. G., & De Agostini, M. (2000). Rehabilitation of brain-injured children. *Child's Nervous System, 16*(10–11), 760–764.
- Laurent-Vannier, A., Pradat-Diehl, P., Chevignard, M., Abada, G., & De Agostini, M. (2003). Spatial and motor neglect in children. *Neurology, 60*(2), 202–207.
- Lawson, I. R. (1962). Visual-spatial neglect in lesions of the right cerebral hemisphere. A study in recovery. *Neurology, 12*, 23–33.
- Lee, B. H., Kang, E., Cho, S. S., Kim, E. J., Seo, S. W., Kim, G. M., . . . Na, D. L. (2010). Neural correlates of hemispatial neglect: a voxel-based SPECT study. *Cerebrovascular Diseases, 30*(6), 573–583.
- LeVere, T. E. (1975). Neural stability, sparing, and behavioral recovery following brain damage. *Psychological Review, 82*(5), 344–358.
- Lindell, A. B., Jalas, M. J., Tenovuo, O., Brunila, T., Voeten, M. J., & Hamalainen, H. (2007). Clinical assessment of hemispatial neglect: Evaluation of different measures and dimensions. *The Clinical Neuropsychologist, 21*(3), 479–497.
- Luaute, J., Halligan, P., Rode, G., Rossetti, Y., & Boisson, D. (2006). Visuospatial neglect: a systematic review of current interventions and their effectiveness. *Neuroscience and Biobehavioral Reviews, 30*(7), 961–982.
- Luukkainen-Markkula, R., Tarkka, I. M., Pitkänen, K., Sivenius, J., & Hämäläinen, H. (2009). Rehabilitation of hemispatial neglect: A randomized study using either arm activation or visual scanning training. *Restorative Neurology and Neuroscience, 27*(6), 663–672.
- Malhotra, P. A., Parton, A. D., Greenwood, R., & Husain, M. (2006). Noradrenergic modulation of space exploration in visual neglect. *Annals of Neurology, 59*(1), 186–190.
- Manly, T. (2002). Cognitive rehabilitation for unilateral neglect: Review. *Neuropsychological Rehabilitation, 12*(4), 289–310.
- Mendoza, J. E. (2011). Suppression. In J. S. Kreutzer, J. DeLuca, & B. Caplan, (Eds.), *Encyclopedia of clinical neuropsychology* (p. 2439). New York, NY: Springer.
- Mesulam, M. M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10*(4), 309–325.
- Mesulam, M. M. (1999). Spatial attention and neglect: parietal, frontal and cingulate contributions to the mental representation and attentional targeting of salient extrapersonal events. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences, 354*(1387), 1325–1346.
- Miller, J. C., Ruthig, J. C., Bradley, A. R., Wise, R. A., Pedersen, H. A., & Ellison, J. M. (2009). Learning effects in the block design task: a stimulus parameter-based approach. *Psychological Assessment, 21*(4), 570–577.
- Milner, A. D., & McIntosh, R. D. (2005). The neurological basis of visual neglect. *Current Opinion in Neurology, 18*(6), 748–753.
- Mukand, J. A., Guilmette, T. J., Allen, D. G., Brown, L. K., Brown, S. L., Tober, K. L., & Vandyck, W. R. (2001). Dopaminergic therapy with carbidopa L-dopa for left neglect after stroke: a case series. *Archives of Physical Medicine and Rehabilitation, 82*(9), 1279–1282.
- Natale, E., Posteraro, L., Prior, M., & Marzi, C. A. (2005). What kind of visual spatial attention is impaired in neglect? *Neuropsychologia, 43*(7), 1072–1085.
- New York University, Institution of Rehabilitation Medicine. (1983). *Working approaches to remediation of cognitive deficits in brain damaged persons* (Supplement to the 11th Annual Workshop for Rehabilitation Professionals). New York, NY: NYUIRM.
- Ogden, J. A. (1987). The “neglected” left hemisphere and its contribution to visuospatial neglect. In J. A. Ogden & M. Jeannerod (Eds.), *Neurophysiological and neuropsychological aspects of spatial neglect* (pp. 215–233). New York, NY: Elsevier Science.
- Osawa, A., & Maeshima, S. (2010). Family participation can improve unilateral spatial neglect in patients with acute right hemispheric stroke. *European Neurology, 63*(3), 170–175.

- Pantano, P., Di Piero, V., Fieschi, C., Judica, A., Guariglia, C., & Pizzamiglio, L. (1992). Pattern of CBF in the rehabilitation of visuospatial neglect. *The International Journal of Neuroscience*, 66(3-4), 153–161.
- Payne, B. R., & Rushmore, R. J. (2004). Functional circuitry underlying natural and interventional cancellation of visual neglect. *Expérimental Brain Rresearch. Expérimentelle Hirnforschung. Expérimentation cérébrale*, 154(2), 127–153.
- Peelen, M. V., Heslenfeld, D. J., & Theeuwes, J. (2004). Endogenous and exogenous attention shifts are mediated by the same large-scale neural network. *NeuroImage*, 22(2), 822–830.
- Pisella, L., Berberovic, N., & Mattingley, J. B. (2004). Impaired working memory for location but not for colour or shape in visual neglect: a comparison of parietal and non-parietal lesions. *Cortex*, 40(2), 379–390.
- Pizzamiglio, L. (2003). Rehabilitation of visual neglect. *Cognitive Processing*, 8(11), 8–11.
- Pizzamiglio, L., Frasca, R., Guariglia, C., Incoccia, C., & Antonucci, G. (1990). Effect of optokinetic stimulation in patients with visual neglect. *Cortex*, 26(4), 535–540.
- Platt, P. A., Dahn, D. A., & Amburn, P. (1991). *Low-cost approaches to virtual flight simulation*. Proceedings of the IEEE 1991 National Aerospace and Electronics Conference, NAECON 1991, 23, 940–946. New York, NY: IEEE.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, 13, 25–42.
- Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience*, 4(7), 1863–1874.
- Proto, D., Pella, R. D., Hill, B. D., & Gouvier, W. D. (2009). Assessment and rehabilitation of acquired visuospatial and proprioceptive deficits associated with visuospatial neglect. *Neuro Rehabilitation*, 24(2), 145–157.
- Ptak, R., Schnider, A., Golay, L., & Muri, R. (2007). A non-spatial bias favouring fixated stimuli revealed in patients with spatial neglect. *Brain*, 130, 3211–3222.
- Righi, G., & Tarr, M. J. (2011). Visual agnosia. In J. S. Kreutzer, J. DeLuca, & B. Caplan, (Eds.), *Encyclopedia of clinical neuropsychology* (pp. 2623–2625). New York, NY: Springer Publishing Company.
- Rizzo, A. A., Buckwalter, J. G., Bowerly, T., Van Der Zaag, C., Humphrey, L., Neumann, U., . . . Sisemore, D. (2004). The virtual classroom: A virtual reality environment for the assessment and rehabilitation of attention deficits. *CyberPsychology & Behavior*, 3(3), 489–499.
- Robertson, I. H., McMillan, T. M., MacLeod, E., Edgeworth, J., & Brock, D. (2002). Rehabilitation by limb activation training reduces left-sided motor impairment in unilateral neglect patients: A single-blind randomised control trial. *Neuropsychological Rehabilitation*, 12, 439–454.
- Robertson, I. H., Tegner, R., Tham, K., Lo, A., & Nimmo-Smith, I. (1995). Sustained attention training for unilateral neglect: theoretical and rehabilitation implications. *Journal of Clinical and Experimental Neuropsychology*, 17(3), 416–430.
- Rossetti Y., & Rode, G. (2002). Reducing spatial neglect by visual and other sensory manipulations: Noncognitive (physiological) routes to the rehabilitation of a cognitive disorder. In H. O. Karnath, D. Milner, & G. Vallar (Eds.), *The cognitive and neural bases of spatial neglect* (pp. 375–396). New York, NY: Oxford University Press.
- Rossi, P. W., Kheyfets, S., & Reding, M. J. (1990). Fresnel prisms improve visual perception in stroke patients with homonymous hemianopia or unilateral visual neglect. *Neurology*, 40(10), 1597–1599.
- Rossit, S., Muir, K., Reeves, I., Duncan, G., Birschel, P., & Harvey, M. (2009). Immediate and delayed reaching in hemispatial neglect. *Neuropsychologia*, 47(6), 1563–1572.
- Rubens, A. B. (1985). Caloric stimulation and unilateral visual neglect. *Neurology*, 35(7), 1019–1024.
- Sacks, O. (1970). *The man who mistook his wife for a hat and other clinical tales*. New York, NY: Simon & Schuster.
- Schroder, A., Wist, E. R., & Homberg, V. (2008). TENS and optokinetic stimulation in neglect therapy after cerebrovascular accident: a randomized controlled study. *European Journal of Neurology*, 15(9),



922–927.

- Seymour, N. E., Gallagher, A. G., Roman, S. A., O'Brien, M. K., Bansal, V. K., Andersen, D. K., & Satava, R. M. (2002). Virtual reality training improves operating room performance: results of a randomized, double-blinded study. *Annals of Surgery, 236*(4), 458–463.
- Shutter, L. A., & Jallo, J. (1998). Outcome from traumatic brain injury. *Trauma Quarterly, 14*(1), 61–100.
- Singh-Curry, V., Malhotra, P., Farmer, S. F., & Husain, M. (2011). Attention deficits following ADEM ameliorated by guanfacine. *Journal of Neurology, Neurosurgery, & Psychiatry, 82*, 688–690.
- Smania, N., Bazoli, F., Piva, D., & Guidetti, G. (1997). Visuomotor imagery and rehabilitation of neglect. *Archives of Physical Medicine and Rehabilitation, 78*(4), 430–436.
- Smania, N., Martini, M. C., Gambina, G., Tomelleri, G., Palamara, A., Natale, E., & Marzi, C. A. (1998). The spatial distribution of visual attention in hemineglect and extinction patients. *Brain, 121*, 1759–1770.
- Soderback, I., Bengtsson, I., Ginsburg, E., & Ekholm, J. (1992). Video feedback in occupational therapy: its effects in patients with neglect syndrome. *Archives of Physical Medicine and Rehabilitation, 73*(12), 1140–1146.
- Sprague, J. M. (1966). Interaction of cortex and superior colliculus in mediation of visually guided behavior in the cat. *Science, 153*(3743), 1544–1547.
- Stirk, J. A., & Foreman, N. (2005). Assessment of visual-spatial deficits in patients with Parkinson's disease and closed head injuries using virtual environments. *Cyberpsychology & Behavior, 8*(5), 431–440.
- Stokes, T. F., & Baer, D. M. (1977). An implicit technology of generalization. *Journal of Applied Behavioral Analysis, 10*, 349–367.
- Striener, C. L., & Danckert, J. A. (2010). Through a prism darkly: Re-evaluating prisms and neglect. *Trends in Cognitive Sciences, 14*(7), 308–316.
- Szczepanski, S. M., Konen, C. S., & Kastner, S. (2010). Mechanisms of spatial attention control in frontal and parietal cortex. *Journal of Neuroscience, 30*(1), 148–160.
- Taub, E. (2004). Harnessing brain plasticity through behavioral techniques to produce new treatments in neurorehabilitation. *American Psychologist, 59*(8), 692–704.
- Thimm, M., Fink, G. R., Kust, J., Karbe, H., & Sturm, W. (2006). Impact of alertness training on spatial neglect: A behavioural and fMRI study. *Neuropsychologia, 44*(7), 1230–1246.
- Thiyagesh, S. N., Farrow, T. F., Parks, R. W., Accosta-Mesa, H., Young, C., Wilkinson, I. D., . . . Woodruff, P. W. (2009). The neural basis of visuospatial perception in Alzheimer's disease and healthy elderly comparison subjects: An fMRI study. *Psychiatry Research, 172*(2), 109–116.
- Tsang, M. H., Sze, K. H., & Fong, K. N. (2009). Occupational therapy treatment with right half-field eye-patching for patients with subacute stroke and unilateral neglect: a randomised controlled trial. *Disability and Rehabilitation, 31*(8), 630–637.
- Vallar, G., Rusconi, M. L., & Bernardini, B. (1996). Modulation of neglect hemianesthesia by transcutaneous electrical stimulation. *Journal of the International Neuropsychological Society, 2*(5), 452–459.
- Verdon, V., Schwartz, S., Lovblad, K. O., Hauert, C. A., & Vuilleumier, P. (2010). Neuroanatomy of hemispatial neglect and its functional components: a study using voxel-based lesion-symptom mapping. *Brain, 133*, 880–894.
- Weiner, M. J., Hallett, M., & Funkenstein, H. H. (1983). Adaptation to lateral displacement of vision in patients with lesions of the central nervous system. *Neurology, 33*(6), 766–772.
- Wilson, B., Cockburn, J., & Halligan, P. (1987). Development of a behavioral test of visuospatial neglect. *Archives of Physical Medicine and Rehabilitation, 68*(2), 98–102.
- Zihl, J. (1981). Recovery of visual functions in patients with cerebral blindness. Effect of specific practice with saccadic localization. *Experimental Brain Research. Experimentelle Hirnforschung. Expérimentation cérébrale, 44*(2), 159–169.

# Educational and Occupational Rehabilitation and Intervention

*Sarah C. Connolly, Eric E. Pierson, and Chad A. Noggle*

The rehabilitation of individuals with neurological and psychiatric disorders is a complex task requiring the coordination of health service providers, family members, agencies, state agencies, employers, and educators (Shigaki, 2001). The neuropsychologist or psychologist engaging in rehabilitation services is oftentimes the ideal individual to integrate the diverse health groups and their unique goals and agendas into a complementary plan. The types of individuals necessary to such a treatment team will vary depending upon the specific referral for rehabilitation and educational services, the location, and the goals for treatment. Individuals in need of educational and rehabilitation services include those with a mood disorder, anxiety disorder, psychotic disorder, personality disorder, or a disorder typically first diagnosed in childhood. Individuals with primarily neurological disorders also benefit from the use of educational and rehabilitation services to complement treatment. Examples of neurological conditions that may benefit include tic disorder, Tourette's syndrome, seizure disorder, traumatic brain injury (TBI), stroke, aphasia, or other injury.

Regardless of the type of disorder, the treating psychologist or neuropsychologist has a unique set of skills that may be used to communicate effectively with different members of the medical and rehabilitation staff and also with the patient and family (Braga, Da Paz, & Ylvisaker, 2005; Murray, Maslany, & Jeffery, 2006). Training in the biological and medical bases for treatment allows for communication with nurses and physicians. Knowledge of psychometrics and the testing essential in clinical evaluations by psychologists,

occupational therapists, physical therapists, and speech and hearing pathologists allows for an ability to translate results for patients (Mitrushina, Boone, & D'Elia, 1999). Added to this is the knowledge of psychological processing and group dynamics that enable psychologists to appreciate and connect with the family and patient in ways other professionals may not. The training enables us to have a view of the situation that moves us from the diagnostic question to the heart of rehabilitation, helping the individual to move from a condition of disease to one of purposeful action, providing a sense of belonging, and giving meaning to one's life.

Although the immediate treatment and recovery of functioning from many illnesses must require specialized care in a hospital or outpatient clinic (van Heugten et al., 2006), the goals and objectives of rehabilitation and educational services require the psychologist or neuropsychologist to connect with the family, teachers, or employers of the patient (Braga et al., 2006; Murray et al., 2006). As the psychologist moves from the hospital setting into the residential and occupational settings of the patient's life and encounters environmental tasks and challenges that were not present in the health setting, a new appraisal of the individual's ability to function may emerge (Klonoff et al., 2006). The psychologist may also gain a greater perspective of the patient, the resources available to him or her, and cultural factors that may alter the progression of treatment that otherwise would not be seen. As a result of the need for the rehabilitation professional to work with the family and employers, the current chapter is organized around specific disability conditions with guidance for educational, occupational, and family interventions included.

## **EDUCATIONAL AND OCCUPATIONAL REHABILITATION OF NEUROLOGICAL DISORDERS**

### **Seizure Disorder**

An individual with a seizure disorder is likely to require a broad range of rehabilitation services depending upon the nature of the disorder, level of functioning of the individual, response to medication, and frequency of seizures. Seizures are categorized depending on whether the seizure has a localized point of initiation or if it is a seizure that has an initiation in multiple broad regions of the brain. Although information from clinical observations, third-party reports, and the subjective reports of patients may be helpful in correctly classifying the type of seizure, the use of electroencephalograms (EEGs) is often necessary

(Weinstein & Gaillard, 2007). Generalized seizures, absence seizures, and partial seizures can all lead to changes in consciousness. Individuals experiencing any type of seizure activity are likely to present with problems in memory, alertness, and attention of which those around them need to be made aware. Transient periods of confusion and disorientation proximal to seizure activity are common manifestations (Weinstein & Gaillard, 2007).

A seizure by its very nature involves the rapid firing of neurons in the brain to a point at which the death of neurons may occur. As a result, most medications designed to treat epilepsy and/or seizures must, in some fashion, govern the rate of neuronal firing between cells. The cellular activity of learning in the brain is connected to the ability of neurons to fire and the ability of cells to alter the rate of fire. Given the nature of antiseizure medications, they often have side effects that may include disruptions in one's ability to learn or recall new material.

Working with those who have seizures may be a difficult task. Individuals who are not at therapeutic or effective levels of medication may have slower rates of learning because of difficulties with attention and concentration as a result of the ongoing seizure activity. Individuals who are receiving appropriate levels of medication and are not currently experiencing seizure activity may still have slower than typical rates of learning as a result of the medicinal side effects.

In addition to changes in consciousness, teachers and parents also must be aware whether there is a history of seizures with motor activity. Examples include myoclonic, atonic, and tonic-clonic seizures. Individuals working with these patients need to be made aware of common first aid steps that should be taken in the event of a seizure, such as clearing the space around the individual and providing her with water following the end of the seizure. Individuals with atonic seizures are at risk of striking the ground and, as a result, may need to wear a helmet or other protective headgear (Weinstein & Gaillard, 2007).

## Educational Interventions

In addition to taking precautions to promote the physical safety of individuals with seizure disorders, school personnel may educate themselves on the related symptoms and implement procedures for supporting these students who may experience frequent absenteeism, academic underachievement, and problems with peer relations within the school setting. One way in which a school system may become better informed of the individual's needs could be through a

comprehensive evaluation conducted by a multidisciplinary team to assess functioning across domains (e.g., intellectual functioning, adaptive behavior, sensory functioning, and speech/language functioning). By gaining a better understanding of the student's potential deficits through an evaluation, the multidisciplinary team can then determine how to best support the student by determining possible special education eligibility or necessary classroom modifications.

When it comes to the effects of seizure disorders within the classroom, educators should be aware of how seizures interrupt student learning. For example, recurring petit mal seizures (which may occur hundreds of times per day) or partial seizures may cause the student to experience difficulties paying attention to verbally presented directions, decreased work pace, or an inability to demonstrate competence in multistep problem-solving skills ([Wodrich & Cunningham, 2008](#)). Further, some complex partial seizures are followed by periods of decreased arousal, as well as difficulties encoding new material and recalling crystallized information. Classroom modifications for assisting students whose consciousness may be temporarily interrupted as a result of seizures include simplifying and slowing the pace of verbally presented information, pairing the student with a peer to clarify directions, and providing written directions ([Wodrich & Cunningham, 2008](#)). Those individuals who experience partial seizures may have specific difficulties with declarative memory and/or expressive and receptive language. In the classroom, this may manifest as difficulties memorizing factual information, which can become increasingly important as students progress through school. To address the student's difficulties with memory, [Wodrich and Cunningham \(2008\)](#) suggest that teachers (a) assess these students' knowledge through tasks of recognition, rather than recall (e.g., multiple-choice tests, rather than essay or free-recall tests), (b) provide several short exams rather than one long exam, (c) assess the students' general understanding of a concept rather than specific, related facts, (d) and teach mnemonic strategies to enhance memorization.

Individuals with seizure disorders may also experience difficulties in social and peer relationships within the classroom as a secondary result of these disorders. Seizure activity may lead to events that have negative social repercussions such as the loss of bowel and bladder control; loss of motor movement; and unusual behavior exhibited prior to, during, or after seizure. As a result, individuals working on rehabilitation with these individuals and their families need to consider common sense steps to reduce embarrassment. These

include the use of class lessons on seizures and steps to keep changes of clothing at school ([Weinstein & Gaillard, 2007](#)).

The desire to be involved in sports and extracurricular activities by individuals with seizure disorders needs to be carefully considered. Generally, engaging in physical activity that promotes overall health is encouraged. However, those with a history of seizure disorders may need to take extra precautions in regard to hydration and nutrition and coordinating medication. It is important that the family and patient consider whether or not the activity that is being discussed will increase the likelihood of a seizure. Activities that would involve physical blows to the head (football, wrestling, or boxing) should be discouraged, whereas those such as running and swimming should be encouraged.

### Occupational Interventions

It is also important to consider the implications for the future of individuals with seizure disorders, who will likely transition from a school environment to an employment setting. Although many individuals with epilepsy demonstrate the ability to maintain employment and sufficiently perform work-related tasks, unemployment rates have consistently been higher for people with epilepsy as compared to the general population ([de Boer, 2005](#)). Research conducted on employment rates finds individuals with epilepsy who are seeking employment are at a disadvantage when compared to their nonepileptic peers, but several clinical variables, such as the number of antiepileptic drugs (AEDs) used, frequency of seizures, and seizure type, further impact their ability to obtain employment ([de Boer, 2005](#)). Family members or caretakers of those with epilepsy are also subjected to workplace discrimination, which may lead to decreased opportunities for retention or promotion ([Parfene, Stewart, & King, 2009](#)). To minimize the stigma associated with epilepsy that frequently occurs in the workplace, it is recommended that employers utilize organization-wide education programs that address the discrimination of individuals with epilepsy, or those associated with an individual with epilepsy (e.g., family members or caregivers; [Parfene, Stewart, & King, 2009](#)). Such educational programs may minimize stereotypes related to seizure disorders, thus promoting employment opportunities for family members and individuals with epilepsy. To improve opportunities for employment, [de Boer \(2004\)](#) emphasized the use of vocational reintegration programs for people with epilepsy who may have cognitive difficulties or restrictions resulting from a history of seizures. Through a

vocation integration program, a vocational consultant examines the individual's strengths and weaknesses through interviews, review of background history, neuropsychological assessments, matching the individual with a potential employer with the intention of preparing the employee for a permanent employment placement (de Boer, 2004). The vocational integration program described by de Boer is much like the aforementioned evaluation process in the school, during which the individual's strengths and limitations are examined, with the overarching goal of promoting workplace success.

## Family Support

In addition to the educational, occupational, and social-emotional effects that seizures may have on the individual, family members are likely to play an integral role in the development of the individual with epilepsy. Caring for an individual who experiences seizures can be a trying experience for family members, who are likely to devote emotional and financial support toward caring for the medical and developmental needs of the individual. Parents themselves may experience their own psychological and social difficulties as a result of their child's conditions, such that they may feel stigmatized, restricted, and stressed. A high degree of family problems may be detrimental to the child with epilepsy, with research suggesting that family conflict and poor cohesion may be associated with the frequency and severity of seizures, as well as the child's psychosocial adjustment (McCusker, Kennedy, Anderson, Hicks, & Hanrahan, 2002). To minimize the effects of family problems on seizure disorder, it is important to promote a positive emotional and psychological climate within the family, which may be achieved through increased cohesiveness and expressiveness among family members, as well as decreased conflict.

## Traumatic Brain Injury

As is the case with individuals with seizure disorders, those persons with TBI require varying degrees and types of support in the educational or work environment depending upon the nature, location, and extent of the injury. It is critical that those conducting rehabilitation efforts with an individual with a history of TBI obtain an evaluation of the individual's preserved and impaired areas of functioning. A thorough neuropsychological evaluation may help to provide important information as to broad areas of deficits and specific skills with which the individual may need support. It is important to recognize that

much rehabilitation work may involve problem solving and skill teaching in areas that are not typically assessed with traditional measures. As a result, rehabilitation psychologists may find it necessary to employ the use of functional skill assessment techniques to readily identify where the individual is having difficulty in the accomplishment of a common, but complex activity.

Work with individuals with a history of TBI may also benefit from an incorporation of the use of family and systems theory, as well as neuropsychological and behavioral approaches to intervention. Individuals with TBI may exhibit difficulty in emotional regulation, a reduction in impulse control, and impaired social skills judgment leading to higher levels of familial strife. Although family members may find it difficult to accept losses in cognition, memory, and attention that correspond with a head injury, they may be more accepting of these changes than those that lead to changes in mood, behavior, and personality.

Individuals with a history of TBI may also experience symptoms of posttraumatic stress related to the events surrounding the injury. As a result, those working with individuals with a history of head injury may wish to learn more about the details surrounding the incident and determine whether or not it is necessary to remove specific stimuli to avoid triggering anxiety.

Interventions with those involved in head injury will need to vary depending upon the nature, location, and severity of the injury. Individuals with head injuries are likely to have difficulties with attention. This is the most common difficulty seen in individuals with a TBI given the tendency for injury to typically occur to the prefrontal and posterior regions of the brain (Lezak et al., 2004). As a result of difficulties with attention, it may help to minimize distractions within the workspace or educational environment of those with a head injury. Reducing the number of choices an individual needs to make and lengthening the amount of time an individual has to make choices can be beneficial.

As it is important to consider the details surrounding a traumatic event to help consider whether specific stimuli in the environment may elicit symptoms of anxiety, it is also important to recognize that individuals with a history of TBI may also have a history of or are currently involved in substance abuse. Those working with the individual need to consider whether additional professionals or other key figures in the individual's life need to be included in the treatment team in those cases.

## Educational Interventions



## Educational Interventions

Current trends in the fields of special education and school psychology have led to the widespread adoption of single-subject case design studies. This makes the practicing school psychologist an ideal collaborator for those working with children in need of rehabilitation. School psychologists are familiar and well versed in the use of tools for the repeated measurement of important academic achievement constructs such as reading, writing, and mathematics performance. The school psychologist may be less familiar with tools appropriate for the evaluation of neuropsychological disability such as disinhibition and/or the dysregulation of emotions.

School psychologists may also be helpful in working to isolate and analyze the specific skills with which individuals in need of rehabilitation are having difficulty. The use of a functional analysis, when properly conducted, can be critical in discriminating which skills or tasks are beyond the ability of the individual and which tasks are being avoided because of discomfort or difficulty. Further data can be generated to identify what aspect of the tasks at hand may require specific alternative strategies or support beyond instruction to assist with rehabilitation ([Sattler & Hogue, 2006](#)).

Oftentimes, children who have sustained a head injury may miss a significant amount of time in school. Families may wish to consider a school reentry process, during which the child can receive individualized tutoring from a special education teacher who is familiar with possible cognitive deficits and can develop rehabilitative goals that are consistent with the academic curriculum ([Marcantuono & Prigatano, 2008](#)).

## Occupational Interventions

Similar to individuals with a history of seizure disorder, those with a TBI may report experiencing cognitive fatigue more quickly, which can impede job performance. As a result, it may be beneficial to provide several breaks during the workday to allow the individual to relax and mentally regroup.

Supported employment has been a widely utilized and researched intervention for developing the vocational skills of individuals with disabilities, specifically those with TBI. Unlike a traditional work setting, a supported employment program incorporates on-site training with support from a professional for as long as necessary, monetary compensation for work, and a goal of assisting the individual to transition to a competitive work setting in which the individual can take on age-appropriate work responsibilities ([Gamble](#)

& Moore, 2003). Work environments in which an individual has the opportunity to improve work performance through positive, constructive feedback and prosocial support and without the competitive conditions found in traditional work environments predict better vocational outcomes for patients with TBI (Kendall, 2003).

## Family Support

The effects of TBI extend beyond the individual and have a significant impact on family members and caregivers who also are likely to experience unexpected lifestyle adjustments and may be required to devote more care toward the individual than before the injury. As a result, family dynamics may shift and parental stress may increase, thus leading to negative outcomes for the child's progress (Braga, Da Paz, & Ylvisaker, 2005). Still, with the proper support from professionals, families may have the transformative power of promoting healing in children who experience impairments following brain injury. Family-supported interventions that incorporate parent training for delivering rehabilitation services in the home may even yield more positive improvements than those delivered within a clinical setting (Braga, Da Paz, & Ylvisaker, 2005). A family-supported treatment plan should include frequent collaboration between family members and multidisciplinary team members (physicians, psychologists, occupational therapists, physical therapists, and speech/language pathologists) through scheduled meetings and home visits, as well as intensive parent training for delivering the exercises and modifications at home, including mentoring from a clinician and graphic illustrations for implementing the intervention (Braga, Da Paz, & Ylvisaker, 2005). The overall family environment further plays a role in the behavioral adjustment of the child following injury. Generally, better family functionality, including high levels of parental support, acceptance, and structure, predicts improved behavior outcomes (Yeates et al., 2010). Despite the challenges of raising a child whose development has been impacted by a TBI, parents are encouraged to feel empowered as their involvement in treatment is an imperative factor in the promotion of positive outcomes.

## Stroke

Stroke refers to a sudden disruption in cerebral blood flow, during which brain cells do not receive adequate amounts of oxygen and glucose necessary for proper functioning. (Kempler, 2005). If the levels of oxygen and glucose are not

quickly restored, the cells will die and as a result, a patient's neurological functioning is impaired due to cell death within a particular region of the brain (Kempler, 2005). Just like TBI, the symptoms of stroke differ based on the portion of the brain that has been damaged. The long-term effects also differ from person to person based on the location of the stroke. Although strokes may occur at any age, the risk of experiencing a stroke increases with age, which may be further increased with both treatable and untreatable risk factors. Though untreatable risk factors, such as age, gender, and heredity may not be addressed through intervention programs, reducing treatable risk factors, such as smoking, obesity, hypertension, or heart disease may be an effective means of stroke prevention.

In addition to the physical effects of stroke on functioning, individuals may experience neurological deficits and cognitive impairments as well as behavioral and psychological changes that may impair their ability to cope with problems. Following a stroke, patients may experience elevated levels of depression, irritability, apathy, decreased sexual activity, reductions in social and leisure activities, and increased family conflict (Angeleri, Angeleri, Foschi, Giaquinto, & Nolfe, 1993).

## Educational Interventions

The intervention developed to meet the needs of a stroke victim will be largely dependent on the location and severity of the stroke. Stroke is considered highly uncommon in children and the causes vary from those of adult stroke patients. For some children who experience a stroke the causes are idiopathic, other children may have preexisting medical conditions that predispose them to the occurrence of stroke, such as sickle cell anemia, collagen-vascular disorders, and increased risks for blood clots (Gabis, Yangala, & Lenn, 2002). Some research has been devoted to the outcomes following a stroke in a child with sickle cell anemia, as well as in children who experienced silent stroke. Again, many of the impairments differ based on the location of the stroke, but individuals who experienced a stroke with a diffuse lesion tended to demonstrate impairments in spatial tasks, whereas individuals who experience strokes with anterior lesions more commonly demonstrated impairments in attention (Craft, Schatz, Glauser, Lee, & DeBaun, 1993). Recognizing the possible neurological deficits in children who have experienced stroke will likely have strong implications for delivering classroom instruction to the child. For example, children who have spatial deficits may demonstrate specific difficulties in reading, characterized by

poor skills in arranging letters, words, or sentences on a page, or difficulty with construction tasks in which the individual is asked to reproduce a model by writing or drawing it or reconstructing the model using parts. Here, it may be useful to provide the student with multiple verbal prompts rather than exclusively providing a visual prompt. For students who experience attention problems as a result of stroke, several classroom modifications may be implemented to create a well-structured and predictable environment. The classroom environment may also be modified to minimize distractions for students. This can be achieved by providing the student with preferential seating, in which the student is seated at the front of the classroom, near the teacher's areas of instruction, and away from windows and doors, or other areas in which he or she may be distracted by external stimuli. The goal is to reduce any potential distraction, while increasing the opportunity to attend to appropriate stimuli, such as verbal and visual input. It may also be useful to create clear goals and expectations regarding work completion. For example, to encourage attention and effort during nonpreferred tasks, students with attention problems should be provided with a mixture of high-and low-interest tasks, as well as a goal for the amount of work to be completed before taking a break. To assist with this it may be helpful to use incremental teaching strategies. These strategies employ a high rate of success (90%) to failure in the structure of learning situations (Coddling, Hilt-Panahon, Panahon, & Benson, 2009). Again, because the outcomes of stroke differ based on the location and severity, the interventions implemented in the classroom will likely be modified to best meet the individual needs of the student.

## Occupational Interventions

One's ability to return to work following a stroke may be impacted by physical or psychological changes that may have occurred. For example, impaired communication may affect one's ability to fulfill social components of the job, whereas physical or cognitive impairments may cause the individual to experience difficulty with carrying out the physical work demands (e.g., reduced work pace). Still, Angleri *et al.* (1993) reported the outcome of many individuals who are able to return to work following stroke, such that returning to work results in a more positive outlook on life, increased involvement in leisure activities, and an improved financial situation. Though the work involvement appears to yield positive results for stroke patients, vocational rehabilitation services may be necessary and useful to assist the patient in gradually returning

to work and developing a productive work pace, as well as feelings of satisfaction toward one's job (Angleri et al., 1993).

## Family Support

Similar to the aforementioned neurological disorders, it is important to examine the role of family support in measuring stroke patient outcomes. The research regarding the effects of family support on improved stroke patient functioning has been mixed, with some studies reporting that high levels of family support result in significantly improved functioning (Tsouna-Hadjis, Vemmos, Zakopoulos, & Stamatelopoulos, 2000), and other studies indicating that though caregivers may experience increased life satisfaction and social activities, few changes occur on the part of the stroke patients (Mant, Carter, Wade, & Winner, 2000). Those studies that reported positive outcomes as a result of family support emphasized the need for social support (especially from a spouse), instrumental help, emotional support, and encouragement, as well as adherence to treatment plan procedures (Tsouna-Hadjis et al., 2000). Additionally, providing caregivers with appropriate support and education can be useful in promoting positive outcomes for patients, as well as the family members. Specifically, implementing education programs and counseling services for caregivers assisted the family functioning as a whole (Evans, Matlock, Bishop, Stranahan, & Pederson, 1988). Effective caregiver education programs have included information regarding typical consequences of stroke (e.g., language impairments, cognitive impairments, and physical impairments), as well as individualized treatment programs and opportunities for caregivers to collaborate with professionals (Evans et al., 1988). Caregiver counseling programs have been effective when including education programs as well as the integration of cognitive behavioral therapy (CBT) (Evans et al., 1988).

## PSYCHIATRIC PROBLEMS

Although psychiatric manifestations are not conceptualized as falling within the parameters of a traditional rehabilitation setting, they are worthy of discussion as they are commonly associated with functional deficits that require intervention. Though objectively identifiable deficits in cognition have been inconsistently reported in the literature, there has been greater consensus regarding the impact of psychiatric problems on school and occupational performance. For the majority of psychiatric presentations, the *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision (*DSM-IV-TR*; American

Psychiatric Association [APA], 2000) specifically indicates the potential for problems at school and at work. In the following section, we discuss some of the psychiatric presentations, their impact on educational and occupational performance, and means by which rehabilitation can be facilitated.

## Anxiety

Individuals with clinical levels of anxiety may have a wide range of difficulties in the school or occupational setting. Oftentimes individuals with other conditions may have secondary problems with anxiety as a result of other areas of disability or due to an intense fear about limited capabilities in the educational or employment setting. Regardless of whether or not anxiety is the primary area or secondary area of concern, the goal of reducing the presence of anxiety-provoking stimuli should be accompanied by the teaching of skills to improve an individual's ability to manage stress and the anxiety he or she experiences.

When working with individuals experiencing symptoms of anxiety, it is important to work with family, coworkers, and significant others in developing strategies for intervention. The incorporation of third parties into the rehabilitation and treatment of anxiety disorders are helpful for several reasons. First, the development of an increased support net enables the individual patient to more clearly communicate to others the problems they have and areas in which they may need help. Second, the patient is capable of turning to a colleague or colleagues for support in instances when anxiety or panic ensues. Third, the colleague or family member has a specific plan and strategy in place for addressing the anxiety.

One of the key features of anxiety and worry is a sense of a loss of control or lack of ability to control the situation. As such it is essential for those working with the individual to help the identified patient begin to gain control. A good starting point is the individual's breathing and heartbeat. The coworker or family member may be specifically taught controlled breathing exercises to work with the patient when a panic attack arises. It is important to have both the family member and coworker practice the breathing technique with the patient on a regular basis in both mildly stressful and calm situations to allow both to become familiar with the techniques.

In addition to the use of breathing techniques, the coworker and family member can be trained to help the individual use progressive relaxation techniques as well. The coworker and family member who employs both activities together will likely experience a sense of relaxation and calm within

themselves as a reflection of the activity. As a result, the coworker or family member may become more accommodating and supportive of the anxiety felt by the individual.

The client may find it helpful to keep a regular journal or daily frequency count of the number of times that anxiety-management strategies needed to be employed. Coworkers or family members may contribute independent ratings to indicate the frequency with which they are being accessed for help. The use of recorded data may help in not only symptom management but also increase the fidelity of interventions by allowing the clinician to query and verify the techniques as they were employed in specific situations. Additionally, the clinician may revisit the appropriate use of the techniques if the strategies reveal a decreased ability to manage symptoms.

Coworkers and family members may also provide critical information as to cues and triggers of anxiety reactions by the patient. The coworker and family member can also be helpful in identifying roadblocks or impediments to the use of the relaxation techniques that the patient or client may not be able to provide. Family and/or friends may inform the clinician as to why the techniques are not being employed when the patient may not have insight into the particular situation at hand. In some instances, the ability for family or friends to collect data through the use of video can be extremely powerful and helpful for allowing the practitioner to review and improve the use of relaxation techniques.

The incorporation of family and coworkers into the rehabilitation team may also help in terms of medication management. Oftentimes the use of medication is successful in reducing the anxiety felt by individuals with anxiety disorders. However, many of the medications have a high level of addiction potential. The ability of family members to include information regarding the correct and regular use of medication is essential.

## Educational Interventions

Within the classroom, teachers may play an important role in reducing students' anxiety by first creating a well-structured classroom environment with clear expectations, smooth transitions, open communication, and soothing atmosphere. Teachers may assist anxious students by establishing a predictable structure where possible, such as by establishing a routine by which students are given cues when the structure is about to change. Instructional success for most students begins with good classroom management (Kampwirth, 2004; Rathvon, 2010), which children with anxiety will benefit from themselves. Some students

worry excessively about making mistakes in school or about what teachers or parents may think of them. Students who feel especially anxious about their school performance may further benefit from increased positive attention from adults at school, or being provided with praise or tangible reinforcement as they attempt to approach challenging academic tasks.

The use of CBT techniques may be another way of assisting individuals to reduce feelings of anxiety. The use of CBT may be especially useful in school and can be implemented as a universal intervention that serves as a prevention treatment for all students and can be included as part of the school's curriculum (Mayer, Van Acker, Lochman, & Greshman, 2009; Weissman, Antinoro, & Chu, 2008). Through CBT, individuals learn to recognize negative cognitions surrounding their emotions and feelings of anxiety and develop more adaptive problem-solving and coping skills, thus minimizing negative beliefs and associated feelings of anxiety. As described earlier, relaxation techniques may be a useful method for reducing anxiety in children. In a school-wide prevention program, teachers, school counselors, or school psychologists may teach anxious children to recognize physical responses to anxiety then train the child to progressively relax a muscle group. As the child learns to tense and relax muscles, the child becomes more cognizant of physical symptoms of anxiety and learns to recognize these symptoms as a cue to relax. Other CBT strategies to be used in a school setting may include adult modeling of effective coping strategies, role playing to practice coping skills aloud, cognitive restructuring, exposure therapy through the use of a fear hierarchy (which has been especially useful for minimizing test anxiety), and the use of reinforcement contingencies (Weisman et al., 2009).

In addition to the development and use of relaxation techniques to support the individual with anxiety, there has been—over the years—repeated recommendation by those doing evaluations to provide the individual in testing situations with extended time. The rationale underlying this accommodation stems from the idea that the person will have a reduced sense of pressure and feel more at ease when taking the test. Unfortunately this accommodation fails to consider the possibility that the individual will be experiencing the same level of anxiety and fear about the testing situation for a prolonged period. As a result, it is necessary to teach the individual relaxation techniques for managing the stress of the testing situation and the additional time the child/adolescent will spend in the testing environment.



## Occupational Interventions

The workplace may be a setting that is particularly anxiety provoking for some individuals. Consequences of anxiety experienced by employees may include poor work performance, lack of assertiveness, decreased possibilities for promotion, or even work avoidance. The demands of some occupations may cause employees to experience anxiety as a result of pressure to increase work performance and time spent on the job. Because anxiety is an unavoidable aspect of functioning, it is not possible to completely eliminate anxiety, though it is possible to teach individuals strategies for managing and reducing anxiety (Richardson & Rothstein, 2008). As a preventative strategy for reducing anxiety at work, employers can develop an atmosphere designed to reduce work stress, which may include creating support groups, providing employees with increased opportunities to make decisions, or facilitating supportive relationships among colleagues (Richardson & Rothstein, 2008). More intensive interventions used to target individual's specific anxieties at work can be modified based on the individual's symptoms and cognitions. Many of the individualized interventions to reduce workplace anxiety are similar to that of the previously discussed interventions to manage anxiety in school-aged children. For example, CBT may be a useful method of reducing anxiety during which individuals learn to recognize their negative thoughts while developing more adaptive ways of modifying and managing their thoughts during stressful situations. Additional methods for learning to reduce stress may include meditation techniques, exercise programs, biofeedback, or time-management and goal-setting techniques (Richardson & Rothstein, 2008).

## Family Support

Environmental factors play an imperative role in the development and reduction of anxiety. As such, many factors of the home environment may contribute to an individual's experiences of anxiety. Like teachers and employers, parents can be a facilitator of intervention by working to reduce the amount of stress in the home environment. Specifically, parent-child relationships characterized by an insecure attachment and high parental anxiety may increase symptoms of anxiety in children (Ginsburg & Schlossberg, 2002). Though parent anxiety is also unavoidable and may be the result of occupational stress, financial problems, marital problems, lack of social support, or a variety of other problems, the development of parent-child relationships that are secure, adaptable, and trusting tend to be important qualities for creating a supportive and low-stress

environment for the child.

As discussed, the use of CBT has been a well-supported strategy for reducing anxiety in children and adults. Though CBT may be used as an individualized intervention, it may also serve as a family-based treatment. With family involvement during CBT, parents may collaborate with the therapist and be viewed by the child as a “team member” who assists that child by rewarding positive behaviors, providing support, and modeling coping skills ([Ginsburg & Schlossberg, 2002](#)). Further, it may also be useful for parents to participate in support groups to collaborate with others on dealing with a child’s anxiety.

## Depression

Interventions with individuals with depression are capable of dramatically improving quality of life for patients. Current recommendations for the treatment of moderate to severe depression include the combination of psychotherapy and medication. In the rehabilitation environment, it is important to understand that unlike typical aspects of mental health where the presence of a depressed mood may be related to an underlying cognitive distortion or biochemical disorder, the triggering episode may reflect an individual’s realistic appraisal of a new position in life.

It is the responsibility of the practitioner in the rehabilitation setting to help the patient shift focus from what she does not have control over to what she does have control over.

## Educational Interventions

Like anxiety, CBT has been a widely utilized approach to treating depression in children, as well as adults ([Lofthouse & Fristad, 2004](#)). The use of CBT to treat a child with depression is not limited to a clinic setting, but can also be implemented as a school-based intervention, occurring in individual or group sessions. In the classroom, teachers may create additional opportunities for positive adult attention or tangible rewards. Additional CBT techniques, according to [Lofthouse and Fristad \(2004\)](#), include activity-scheduling logs; instruction in self-monitoring and increasing pleasant activities; changing objective conditions to increase rewards; group problem-solving discussions; relaxation training; positive imagery; cognitive restructuring; skills generalization; problem solving; watching videotapes of self displaying nondepressed behaviors; education about thoughts, feelings, and behaviors; social skills training; setting realistic standards and goals; and increasing self-

reinforcement. It is possible for the mental health professional within the school (e.g., school psychologist or school counselor) to develop a consultative relationship during which the techniques used in CBT can extend beyond the therapy session and into the classroom (Kampwirth, 2004; [Martens & Erchul, 2010](#)).

## Occupational Interventions

Individuals with depression oftentimes report struggling to remain productive at work. They may exhibit poor motivation in completing work assignments and duties and even frequently call off from work, which over time can limit opportunities for advancement and possibly even lead to termination. As individuals struggle in job performance, they may manifest additional reduction in self-esteem and self-confidence, which may worsen their depression. Consequently, this may also contribute to reactionary anxiety. Similar to those options discussed as treatment for anxiety, employers may develop support groups and facilitate supportive relationships among colleagues. Scheduled time throughout the day where employees can engage in activities such as walking can be helpful as it breaks up the day, and exercise is known to help improve mood. CBT can help identify negative and ruminating thoughts. Goal-setting techniques can help improve productivity.

## Family Support

Family therapy may further promote positive outcomes in individuals with depression. Parents of young children may learn to shift their own behaviors related to parenting style. Specifically, parents may learn to take on a more authoritative parent style, which emphasizes empathy, listening, positive-reinforcement, a soothing environment, and a reduction in coercive discipline ([Lofthouse & Fristad, 2004](#)). In therapy, families should discuss the effects of depression on the family as a unit, identify potential ongoing or past problems, and openly communicate to problem solve and develop a strong, supportive family environment.

## Bipolar

Individuals with bipolar disorder are often in need of substantial support from family and coworkers. Individuals with bipolar disorder tend to challenge the social support network that is in existence around them. Their day-to-day

presentation can be difficult to predict. In addition, individuals who are bipolar often experience negative side effects to the commonly prescribed medications that help them to regulate and moderate their emotions. Individuals with bipolar disorder may experience feedback from colleagues that they are more predictable but less fun or exciting when they are on their medication. As a result, an implicit message may be sent to the individual that he or she would be better off stopping the medications that they are prescribed.

When working on the rehabilitation of someone with bipolar disorder, it is important to remember that the individual is at increased risk for relapse as a nature of the progression of the disorder. Individuals with bipolar disorder may experience manic, depressive, or mixed episodes and all have serious consequences for the intervention team. Although depressive episodes may cause an individual to be late to work, withdrawn in social situations, and slow in the completion of tasks in the work environment, the manic employee may be prone to careless mistakes, risky behavior, irritability, or aggressiveness. Mixed episodes may involve increased irritability and increased emotional lability. As a result, it is important that family and colleagues regularly check in with the individual. Similar to individuals with personality disorders, the rehabilitation patient with a bipolar disorder may both push and pull the treatment team away as a result of the shift in moods.

Individuals with bipolar disorder are also more prone to abuse substances, particularly during manic phases. As a result, these individuals may have greater difficulties in the workplace or educational environment. Rehabilitation and the training of new skills may be slowed for these individuals in situations in which substance use has returned or is being exhibited on a problematic basis.

Individuals with bipolar disorder may be prone to aggressiveness and irritability. They are more likely to engage in gambling or reckless spending. These behaviors may lead the individual to have problems with law enforcement and incarceration. The rehabilitation team needs to be prepared for the possibility of requests for records from law enforcement officials.

## Educational Interventions

Though psychopharmacological interventions are traditionally the primary approach to treating bipolar disorder, it is important to consider interventions related to the individual's environment and daily functioning, which may be used as a supplement or alternative to the use of medication. It is important for educators to be aware of symptoms of bipolar disorder, which may be

manifested in the classroom by maladaptive internalizing or externalizing behaviors. Specifically, children with bipolar disorder may demonstrate symptoms of depression, such that they cry excessively or display increased irritability or social withdrawal. However, children with bipolar disorder may also display manic symptoms, which may resemble symptoms associated with attention deficit hyperactivity disorder (ADHD). In this case, the child may be hyperactive, talk excessively and rapidly, and report bizarre or grandiose ideas. Unlike children with ADHD, those with bipolar disorder rapidly cycle between extreme emotional states (depressive and mania), which may be observed through the students' interaction with others and their ability to complete academic assignments. To support the educational needs of children with bipolar disorder, it may be appropriate in some cases to conduct a psychoeducational evaluation or functional behavior assessment to determine the impact of the disorder on academic achievement or to determine whether an aspect of the environment can be altered to reduce problem behaviors and better promote academic success. Further, school psychologists and school counselors may expand their knowledge of bipolar disorder and work with teachers and students to develop an environment that supports the needs of the students. Specifically, school psychologists or school counselors may assist teachers by developing a behavior plan in the classroom or by working with the student individually to develop social skills, self-management skills, and to build self-esteem and self-concept (Bardick & Bernes, 2005). To best accommodate the educational needs of the child with bipolar disorder, multidisciplinary collaboration among parents, teachers, and mental health professionals within the school is encouraged.

## Occupational Interventions

For an individual with bipolar disorder, occupational skills will be largely dependent on the individual's current functioning level, his need for support, and the magnitude and management of his depressive or manic symptoms. Similar to procedures conducted in school to assess one's current functional capacities, the administration of a neuropsychological assessment may provide individuals and caretakers with a better understanding of one's vocational abilities. Research on occupational outcomes for individuals with bipolar disorder has indicated that cognitive abilities are a strong predictor of employment status, over and beyond the predictive value of severity of bipolar symptoms (Dickerson et al., 2004). This suggests that strong cognitive abilities serve as a protective factor for individuals with bipolar disorder who are seeking to establish and maintain

employment. Obtaining information regarding an individual's cognitive or neuropsychological functioning may be useful in selecting vocational options that are consistent with the individual's level of functioning. Vocational support programs, such as those described in previous sections, may also be an option for individuals struggling with vocational adjustment who are in need of additional instruction and monitoring to complete work-related tasks.

## Family Support

Perhaps the most well-researched and implemented approach to promote family support for bipolar disorder has been the use of family-focused treatment (FFT; Mikolwitz & Goldstein, 1997; Miklowitz & Otto, 2006). Using FFT, family members play an integral role in promoting mental health, as well as in relapse prevention. Parents, spouses, siblings, and caregivers experience a series of therapeutic and educational sessions during which they are provided with psychoeducation regarding bipolar disorder (knowledge of risk and protective factors, symptoms, treatments, and relapse prevention), communication enhancement training to develop skills in active listening and assertiveness or positive and negative feelings, and problem-solving skills training to assist the individual with bipolar disorder in her daily functioning (Mikolwitz & Goldstein, 1997). Additional family support for those caring for an individual with bipolar disorder may include multifamily or individual family psychoeducation groups during which families are educated on associated symptoms and treatments of bipolar disorder and work to develop problem-solving and communication skills in an individual or multifamily format (Young & Fristad, 2007). In addition to engagement in the aforementioned educational programs, it may also be useful for family members to be provided with resources instruction regarding crisis management. Should the individual with bipolar disorder be under the care of a physician or therapist, collaboration between these family members and these professionals may assist in medication management, as well as progress toward treatment goals.

## Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is characterized by a disturbance in psychological functioning following exposure to an extreme traumatic stressor, characterized by intense fear, helplessness, or horror (APA, 2000). Individuals with PTSD persistently re-experience the traumatic event through intrusive thoughts, flashbacks, nightmares, or hallucinations, persistently avoid stimuli

associated with the trauma, and display an increased sense of arousal characterized by sleep difficulties, hypervigilance, or disturbances in mood or concentration. These symptoms are present for a period of at least 1 month and significantly impair social, occupational, or academic functioning (APA, 2000). Though diagnostic criteria for children and adolescents experiencing PTSD are consistent with the aforementioned criteria, the symptoms of PTSD may be manifested and observed in a manner that is different from that of adults. Children may repetitively reenact play situations that reflect a specific aspect of the trauma rather than reporting flashbacks or numbing experiences (APA, 2000; Mazza & Overstreet, 2000). Responding to and supporting individuals with PTSD may be a difficult task as they may display additional psychological distress (e.g., depression, anxiety or suicidal ideation), developmental difficulties, and problems with daily functioning, characterized by academic or occupational difficulties.

## Educational Interventions

Children experiencing PTSD may struggle in the classroom as they may have difficulty focusing attention, become socially withdrawn, or demonstrate poor academic performance (e.g., poor grades and grade retention). In addition to academic difficulties, children with PTSD may display additional psychological symptoms at school, such as anxiety, depression, or suicidal ideation. Positive school characteristics, such as high-quality schools with connections to prosocial community organizations, may serve as a protective factor in minimizing the effects of environmental stressors and promoting resilience (Mazza & Overstreet, 2000). Still, children most vulnerable to being negatively impacted by environmental trauma and at risk for developing PTSD are likely exposed to multiple risk factors such as traumatic experiences, poverty, minority status, low family cohesiveness and poor parenting, and limited community support and resources.

It is important for educators to recognize protective factors (such as family environment) and work with the students and their family members to increase support and encourage resiliency. Rehabilitation professionals may play a particularly important role in supporting the educational needs of children with PTSD by educating parents and school personnel on the warning signs of traumatic exposure (e.g., difficulty concentrating, separation anxiety, memory impairment, increased aggressiveness, apathy, or diminished interest) and utilizing brief self-report or screening measures to identify these children (Mazza

& Overstreet, 2000). Following the identification of children who are experiencing PTSD, educators may work to develop interventions to target the negative impact in the classroom, which generally include strategies for improving coping skills, self-esteem, and building protective strategies (Mazza & Overstreet, 2000).

## Occupational Interventions

For some individuals with PTSD, their psychological distress may have evolved from a traumatic situation in the workplace (e.g., work-related injury), making one's return to work following a traumatic event, or attitudes and productivity in the workplace, difficult. Therapeutic techniques may be one way of developing strategies to assist the individual in reducing distress surrounding the trauma and to facilitate a successful return to work. Specifically, CBT techniques such as desensitization, flooding, anxiety hierarchy, and coping skills training may be applied to reduce avoidant behaviors and assist the individual in gradually returning to work. Here, the individual learns to become gradually desensitized to the work setting and demands by visiting the work site or working and developing coping skills (MacDonald, Colotla, Flamer, & Karlinsky, 2003). Vocational rehabilitation programs may be another occupational intervention option for PTSD. By working with a vocational rehabilitation consultant, the individual with PTSD may be matched with an employer and work site with the same values and goals as the individual who is willing to provide job modifications and accommodations to promote vocational success.

## Family Support

High levels of family support and positive family environments generally serve as protective factors in children and adolescents who have been exposed to trauma. Specifically, when adequate family support is present, children feel they can discuss the traumatic event with their primary caregiver, thus reducing levels of disturbing thoughts and the severity of internalizing symptoms (Mazza & Overstreet, 2000). Positive parental practices, such as displaying warmth toward children, close monitoring, and expressing clear expectations for obedience further promote positive functioning in children who may be exposed to risk factors outside of the home (e.g., community violence).

## Suicidal Ideation



Regardless of the type or nature of the individual's need for rehabilitation, it is essential that the rehabilitation team be conscious of and aware of the potential for suicidal ideation. Individuals in rehabilitation are often forced to confront problems that are interminable, which may be viewed as intolerable and inescapable. As a result, the individuals may be viewed at an increased risk for suicide (Chiles & Strosahl, 2005). The ability of the rehabilitation team to increase the patient's perceptions that the condition requiring rehabilitation is tolerable, escapable, and time limited improves the ability of the individual to consider other alternatives to suicide.

An effective rehabilitation team that is working with someone at risk for suicide has several important factors to consider. First, communication among team members regarding comments, thoughts, or perceptions that an individual may be at increased risk for suicide should be shared on a regular basis (Rudd, Joiner, & Rajab, 1999). Second, teams often have problems with the dispersion of responsibility regarding suicidal thoughts in both mental health treatment and educational settings. As a result, it is recommended that although it is everyone's responsibility to be vigilant and supportive, one individual member of the team with expertise in the assessment and intervention of suicide be given the responsibility of assessing those suspected of being at increased risk (Poland & Lieberman, 2002).

Individuals with a history of TBI may have deficits in flexible thinking, social skills, and general problem-solving abilities, among other concerns (Lezak et al., 2004). As a result, these individuals may be at greater risk when experiencing suicidal ideation because of the difficulty in generating new solutions to problems and recognizing that old strategies for problem solving may not fit the current situation. An example might be someone asking for a coworker or boss to help him with a task that a family member normally would do at home.

These individuals may also have difficulty generalizing successful problem-solving techniques from one situation to another. As a result, the individual may be quite competent in employing relaxation techniques at home or with using a problem-solving card system such as that suggested by Joiner, Rajab, and Rudd (2001) in the work environment.

## Educational Interventions

The literature surrounding suicide prevention and intervention programs for children and youth in schools is extensive and beyond the scope of this chapter

(e.g., [Brock, 2007](#); [Poland & McCormick, 1999](#); [Poland & Lieberman, 2002](#)). Currently several different approaches to universal screening for school-age individuals at risk of suicide are available, including Question, Persuade, and Refer (QPR). This is a program designed to help improve suicide prevention and awareness and encourage members of the community to respond when those thinking of suicide make themselves known. Additionally, there are several large efforts funded by private foundations such as the Yellow Ribbon Project that can be contacted to work with families and communities after the death of a student to suicide.

The federal government also has placed a priority on improving suicide prevention and intervention efforts in schools. The Garrett Lee Smith grant helps to fund the research and implementation of effective suicide prevention programs for youth and adolescence in schools.

### Occupational Interventions

Individuals who are at risk of suicide and who are involved with a rehabilitation program may present to coworkers in a variety of ways. The most common expressions seen prior to an attempt include depression, withdrawal, and apathy. Some individuals, particularly those who have been depressed for an extended period of time, appear to take comfort in the act of planning, preparing, and dying by suicide. Most often, the experience of these emotions is not joy for the event itself as much as a recognition that the suffering and pain (physical or mental) will soon end. For these individuals, who may have long been contemplating suicide, the act of having made a decision may come as a relief ([Wainrib & Bloch, 1998](#)).

The occupational setting can improve its prevention efforts for those in rehabilitation by promoting positive psychological and health behaviors for all of the staff and rehabilitation patients. A universal approach that advocates for global improvement will benefit both patients and staff. In particular, staff and patients should be encouraged to engage in regular exercise, devote time to personal hobbies or interests, and invest in social support systems, including meaningful relationships with peers.

### Family Support

Family discord and decreased familial support are among the many risk factors for suicidal behavior. Specifically, decreased levels of adaptability and cohesion within the family have indicated higher rates of suicide ([Compton, Thompson, &](#)

[Kaslow, 2005](#)). It is important to recognize that family problems serve as risk factors for suicide, but also recognize that strong family support alternatively serves as a protective intervention. One framework from which social support has been understood has been the social cognitive theory (SCT), in which an individual's behavior is influenced by personal and environmental factors ([Bandura, 1986](#); as cited in [Compton et al., 2005](#)). Family relationships are among the environmental factors influencing one's behaviors, thus suggesting an emphasis on family support in preventing and reducing suicidal behaviors. It may be useful to inform family members to become better educated regarding additional risk factors for suicide, such as reported thoughts of suicide, plans for suicide, access to weapons or medications, as well as other behavioral and affective symptoms such as depressive symptoms. Rehabilitation teams should have at hand local and national crisis numbers as well as information of the National Alliance on Mental Illness (NAMI). NAMI often provides local families with group support and peer mentoring.

## SUMMARY

Today's families are provided rehabilitation services from medical, mental health, and school systems with ever improving technologies and tools. These ever advancing tools and methods continue to require psychologists to be able to connect the family with the medical system. Although technology and tools may facilitate this ability at its core, it still remains an activity that requires a broad knowledge base in family dynamics, mental health services, and intervention design.

Similar to the problems faced by psychologists working in areas of special education and school psychology, rehabilitation psychologists work with unique or small samples. As a result, rehabilitation psychologists and professionals working in these settings may find it most beneficial to consider the use of small-N design in intervention and research approaches when conducting empirical research.

This chapter has provided an overview of the types of methods and interventions that are available to those psychologists working with patients with mental health disorders, TBI, or seizures. In each of these areas, we have discussed strategies applicable to the educational, work, and family setting. It is our belief that rehabilitation as an activity for psychologists must involve an interaction with each of these multiple systems. Further, we remain committed to the vision that the psychologist can act as a bridge between the health services

community, the family, the school, and the individual, to better shape and meld the goals to achieve better functioning.

## REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Anderson, V., Northam, E., Hendy, J., & Wrennall, J. (2001). *Developmental neuropsychology: A clinical approach*. Philadelphia, PA: Psychology Press Ltd.
- Angeleri, F., Angeleri, V. A., Foschi, N., Giaquinto, S., & Nolfe, G. (1993). The influence of depression, social activity, and family stress on functional outcome after stroke. *Stroke*, *24*(10), 1478–1483.
- Bandura, A. V., & Evarestov, R. A. (2012). First-principles calculations on thermodynamic properties of BaTiO<sub>3</sub> rhombohedral phase. *Journal of Computational Chemistry*, *33*(18), 1554–1563.
- Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. New Jersey: Prentice-Hall.
- Bardick, A. D., & Bernes, K. B. (2005). Closer examination of bipolar disorder in school-age children. *Professional School Counseling*, *9*(1), 72–77.
- Braga, L. W., Da Paz, A. C., & Ylvisaker, M. (2005). Direct clinician-delivered versus indirect family-supported rehabilitation of children with traumatic brain injury: A randomized controlled trial. *Brain Injury*, *19*(10), 819–831.
- Braga, L. W., Ylvisaker, M., Rossi, L., & Souza, L. N. (2006). Cognitive development and neuropsychological disorders. In L. W. Braga & A. C. de Paz (Eds.), *The child with traumatic brain injury or cerebral palsy: A context-sensitive, family-based approach to development* (pp. 55–102). New York: Taylor & Francis.
- Brink, J. D., Imbus, C., & Woo-Sam, J. (1980). Physical recovery after severe closed head trauma in children and adolescents. *Journal of Pediatrics*, *97*(5), 721–727.
- Brock, S. E. (2007). Assessment and intervention for bipolar disorder: Best practices for school psychologists. In *Workshop presented at the National Association of School Psychologists & American Healthcare Institute's Fourth Annual Critical Issues in School Psychology Summer Conference, Cleveland, OH*.
- Chiles, J., & Strosahl, K. (2005). *Clinical manual for assessment and treatment of suicidal patients*. Washington, DC: American Psychiatric Publishing.
- Codding, R. S., Hilt-Panahon, A., Panahon, C. J., & Benson, J. L. (2009). Addressing mathematics computation problems: A review of simple and moderate intensity interventions. *Education and Treatment of Children*, *32*(2), 279–312.
- Compton, M. T., Thompson, N. J., & Kaslow, N. J. (2005). Social environment factors associated with suicide attempt among low-income African Americans: The protective role of family relationships and social support. *Social Psychiatry and Psychiatric Epidemiology*, *40*(3), 175–185.
- Craft, S., Schatz, J., Glauser, T. A., Lee, B., & DeBaun, M. R. (1993). Neuropsychological effects of stroke in children with sick cell anemia. *Journal of Pediatrics*, *123*(5):712–717.
- de Boer, H. M. (2005). Overview and perspectives of employment in people with epilepsy. *Epilepsia*, *46*(Suppl. 1), 52–54.
- Dickerson, F. B., Boronow, J. J., Stallings, C. R., Origoni, A. E., Cole, S., & Yolken, R. H. (2004). Association between cognitive functioning and employment status of persons with bipolar disorder. *Psychiatric Services*, *55*(1), 54–58.
- Donders, J. (1992). Premorbid behavioral and psychosocial adjustment of children with traumatic brain injury. *Journal of Abnormal Child Psychology*, *20*(3), 233–246.
- Erchul, W. P., & Martens, B. K. (2010). *School consultation: Conceptual and empirical bases of practice* (3rd edn). New York, NY: Springer Publishing Company.

- Evans, R. L., Matlock, A. L., Bishop, D. S., Stranahan, S., & Pederson, C. (1988). Family intervention after stroke: Does counseling or education help? *Stroke*, *19*(10), 1243–1249.
- Gabis, L. V., Yangala, R., & Lenn, N. J. (2002). Time lag to diagnosis of stroke in children. *Pediatrics*, *110*(5), 924–928.
- Gaillard, W. D., Weinstein, S., Conry, J., Pearl, P. L., Fazilat, S., Fazilat, S.,... Theodore, W. H. (2007). Prognosis of children with partial epilepsy: MRI and serial 18FDG-PET. *Neurology*, *68*(9), 655–659.
- Gamble, D., & Moore, C. L. (2003). Supported employment: Disparities in vocational rehabilitation outcomes expenditures and service time for persons with traumatic brain injury. *Journal of Vocational Rehabilitation*, *19*, 47–57.
- Ginsburg, G. S., & Schlossberg, M. C. (2002). Family-based treatment of childhood anxiety disorders. *International Review of Psychiatry*, *14*, 143–154.
- Joiner, T. E., Jr., Rudd, M. D., & Rajab, M. H. (1999). Agreement between self-and clinician-rated suicidal symptoms in a clinical sample of young adults: Explaining discrepancies. *Journal of Consulting and Clinical Psychology*, *67*(2), 171–176.
- Kempler, P. (2005). Learning from large cardiovascular clinical trials: Classical cardiovascular risk factors. *Diabetes Research and Clinical Practice*, *68*(Supp 1), S43–S47.
- Kendall, E. (2003). Predicting vocational adjustment following traumatic brain injury: A test of psychosocial theory. *Journal of Vocational Rehabilitation*, *19*, 31–45.
- Klonoff, P. S., Watt, L. M., Dawson, L. K., Henderson, S. W., Gehrels, J. A., & Wethe, J. V. (2006). Psychosocial outcomes 1–7 years after comprehensive milieu-oriented neurorehabilitation: The role of pre-injury status. *Brain Injury*, *20*(6), 601–612.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th edn). New York: Oxford University Press.
- Lofthouse, N., & Fristad, M. A. (2004). Psychosocial interventions for children with early-onset bipolar spectrum disorder. *Clinical Child and Family Psychology Review*, *7*(2), 71–88.
- MacDonald, H. A., Colotla, V., Flamer, S., & Karlinsky, H. (2003). Posttraumatic stress disorder (PTSD) in the workplace: A descriptive study of workers experiencing PTSD resulting from work injury. *Journal of Occupational Rehabilitation*, *13*(2), 63–77.
- Mant, J., Carter, J., Wade, D. T., & Winner, S. (2000). Family support for stroke: A randomised controlled trial. *Lancet*, *356*(9232), 808–813.
- Marcantuono, J. T., & Prigatano, G. P. (2008). A holistic brain injury rehabilitation program for school-age children. *Neuro Rehabilitation*, *23*(6), 457–466.
- Mazza, J. J., & Overstreet, S. (2000). Children and adolescents exposed to community violence: A mental health perspective for school psychologists. *School Psychology Review*, *29*(1), 86–1010.
- McCusker, C. G., Kennedy, P. J., Anderson, J., Hicks, E. M., & Hanrahan, D. (2002). Adjustment in children with intractable epilepsy: Importance of seizure duration and family factors. *Developmental Medicine and Child Neurology*, *44*(10), 681–687.
- Mayer, M. J., Van Acker, R., Lochman, J. E., & Gresham, F. M. (Eds.). (2009). *Cognitive-behavioral interventions for emotional and behavioral disorders: School-based practice*. New York, NY: Guilford Press.
- Miklowitz, D. J., & Goldstein, M. J. (1990). Behavioral family treatment for patients with bipolar affective disorder. *Behavior Modification*, *14*(4), 457–489.
- Miklowitz, D. J., & Goldstein, M. J. (1997). *Bipolar disorder. A family-focused treatment approach*. New York: Guilford Press.
- Miklowitz, D. J., & Otto, M. W. (2006). New psychosocial interventions for bipolar disorder: A review of literature and introduction of the systematic treatment enhancement program. *Journal of Cognitive Psychotherapy*, *20*(2), 215–230.
- Mitrushina, M. N., Boone, K. B., & D'Elia, L. F. (1999). *Handbook of normative data for neuropsychological assessment*. New York, NY: Oxford University Press.
- Murray, H. M., Maslany, G. W., & Jeffery, B. (2006). Assessment of family needs following acquired brain

- injury in Saskatchewan. *Brain Injury*, 20(6), 575–585.
- Parfene, C., Stewart, T. L., & King, T. Z. (2009). Epilepsy stigma and stigma by association in the workplace. *Epilepsy & Behavior*, 15(4), 461–466.
- Poland, S., & Lieberman, R. (2002). Best practices in suicide intervention. *Best Practices in School Psychology IV*, 2, 1151–1165.
- Poland, S., & McCormick, J. S. (1999). *Coping with crisis: Lessons learned: A resource for schools, parents, and communities*. Dallas, TX: Sopris West.
- Rathvon, N. (2008). *Effective school interventions: Evidence-based strategies for improving student outcomes* (2nd edn). New York, NY: Guilford Press.
- Richardson, K. M., & Rothstein, H. R. (2008). Effects of occupational stress management intervention programs: A meta-analysis. *Journal of Occupational Health Psychology*, 13(1), 69–93.
- Rudd, M. D., Joiner, T. E., Jr., & Rajab, M. H. (2001). *Treating suicidal behavior: A time-limited approach*. New York: Guilford Press.
- Sattler, J. M., & Hoge, R. D. (2006). *Assessment of children: Behavioral, social, and clinical foundations* (5th edn). La Mesa, CA: Jerome M. Sattler.
- Shigaki, C. L. (2001). National and community resources for neuropsychological disorders. In B. Johnstone & H. H. Stonnington (Eds.), *Rehabilitation of neuropsychological disorders: A practical guide for rehabilitation professionals* (pp. 195–203). Philadelphia, PA: Taylor & Francis.
- Stancin, T., Wade, S. L., Walz, N. C., Yeates, K. O., & Taylor, H. G. (2008). Traumatic brain injuries in early childhood: Initial impact on the family. *Journal of Developmental and Behavioral Pediatrics*, 29(4), 253–261.
- Tsoua-Hadjis, E., Vemmos, K. N., Zakopoulos, N., & Stamatelopoulos, S. (2000). First-stroke recovery process: The role of family social support. *Archives of Physical Medicine and Rehabilitation*, 81(7), 881–887.
- van Heugten, C. M., Hendriksen, J., Rasquin, S., Dijcks, B., Jaeken, D., & Vles, J. H. (2006). Long-term neuropsychological performance in a cohort of children and adolescents after severe paediatric traumatic brain injury. *Brain Injury*, 20(9), 895–903.
- Wade, S. L., Taylor, H. G., Drotar, D., Stancin, T., & Yeates, K. O. (1996). Childhood traumatic brain injury: Initial impact on the family. *Journal of Learning Disabilities*, 29(6), 652–661.
- Wainrib, B. R., & Bloch, E. L. (1998). *Crisis intervention and trauma response: Theory and practice*. New York, NY: Springer.
- Weissman, A., Antinoro, D., & Chu, B. C. (2008). Cognitive-behavioral therapy for anxious youth in school settings: Advances and challenges. In M. Mayer, R. Van Acker, J. E. Lochman, & F. M. Gresham (Eds.), *Cognitive behavioral interventions for students with emotional/behavioral disorders*. New York, NY: Guilford Press.
- Wodrich, D. L., & Cunningham, M. M. (2008). School-based tertiary and targeted interventions for students with chronic medical conditions: Examples from type 1 diabetes mellitus and epilepsy. *Psychology in the Schools*, 45(1), 52–62.
- Yeates, K. O. (2000). Closed-head injury. In K. O. Yeates, M. D. Ris, & H. G. Taylor (Eds.), *Pediatric neuropsychology: Research, theory, and practice* (pp. 92–116). New York, NY: Guilford Press.
- Yeates, K. O., Taylor, H. G., Barry, C. T., Drotar, D., Wade, S. L., & Stancin, T. (2001). Neurobehavioral symptoms in childhood closed-head injuries: Changes in prevalence and correlates during the first year postinjury. *Journal of Pediatric Psychology*, 26, 79–91.
- Yeates, K. O., Taylor, H. G., Drotar, D., Wade, S. L., Klein, S., Stancin, T., & Schatschneider, C. (1997). Preinjury family environment as a determinant of recovery from traumatic brain injuries in school-age children. *Journal of the International Neuropsychological Society*, 3(6), 617–630.
- Yeates, K. O., Taylor, H. G., Walz, N. C., Stancin, T., & Wade, S. L. (2010). The family environment as a moderator of psychosocial outcomes following traumatic brain injury in young children. *Neuropsychology*, 24(3), 345–356.
- Young, M. E., & Fristad, M. A. (2007). Evidence based treatments for bipolar disorder in children and

adolescents. *Journal of Contemporary Psychotherapy*, 37, 157–164.

# Rehabilitation in Traumatic Brain Injury

*Carrie-Ann H. Strong and Jacobus Donders*

Traumatic brain injury (TBI) is a common neurological condition in which there is an external blunt or penetrating force to the skull, causing at least temporary disruption of cerebral functioning. Particularly with more severe injuries, such as those associated with prolonged coma or diffuse intracranial lesions, there can be significant and lasting cognitive and behavioral sequelae that may affect participation in various psychosocial domains, including family, school, work, and broader community integration (Kothari, 2007; Selassie et al., 2008). Neuropsychologists can play an important role in rehabilitation after TBI through assessment, intervention, and consideration of comorbidities and complicating psychosocial factors that influence recovery and longterm outcome. Much of the research on efficacy of rehabilitation after TBI is based on small samples and single-group data, and although there are a growing number of randomized controlled clinical trials, selection bias often remains uncontrolled (Perdices et al., 2006; Teasell et al., 2007). With these reservations in mind, most reviews of TBI rehabilitation support the efficacy of acute inpatient rehabilitation and, to a slightly lesser extent, postacute outpatient rehabilitation with positive longterm benefits, including less need for assistance, more productive participation in society, and less emotional distress (Cullen, Chundamala, Bayley, & Jutai, 2007; Prvu Bettger & Stineman, 2007; Turner-Stokes, 2008; Zhu, Poon, Chan, & Chan, 2007). The goal of this chapter is to review the role of the neuropsychologist in this regard, with emphasis on evidence-based methods and life span developmental issues. Because neuropsychologists are rarely involved with critical care management, this



review will focus on rehabilitative efforts after medical stabilization and emergence from coma.

## EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Each year in the United States, 1.7 million people sustain a TBI. Of those people, 235,000 require hospitalization and 50,000 are fatally injured (Faul, Xu, Wald, & Coronado, 2010). The majority (>80%) of TBIs can be classified as “uncomplicated mild,” as defined by the combination of all of the following: duration of loss of consciousness <30 minutes, Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) score > 12, duration of posttraumatic amnesia <24 hours, and no intracranial lesion on acute neuroimaging. Research has shown that the vast majority (>90%) of both children and adults with uncomplicated mild TBI and with no premorbid or comorbid complicating factors have an essentially unremarkable longterm recovery (Babikian & Asarnow, 2009; Carroll et al., 2004). However, persons with otherwise mild injuries who do have intracranial lesions on neuroimaging typically have less favorable outcomes (Rao et al., 2010; Smits et al., 2008). Studies with both children and adults have been inconsistent with regard to the relative importance of frontal location versus subcortical volume of lesions, but there is no doubt that in pediatric cases, earlier age of injury is associated with worse longterm psychological outcome (Anderson et al., 2006).

Across the life span, the cerebral compromise in TBI is the result of both primary and secondary factors (Bauer & Fritz, 2004). Primary forces include linear displacement due to acceleration or deceleration that causes focal lesions such as cortical contusions and intracranial rotation, which may result in diffuse lesions such as axonal injury. Secondary injuries arise indirectly, may continue to develop over the course of several days, and are the result of disrupted cerebral circulation and an associated cascade of neurochemical events leading to hypoxic–ischemic injury, diffuse edema, and neuronal excitotoxicity. Computerized tomography (CT) scan is still the most appropriate neuroimaging method on day of injury because it can be done fairly quickly in medically unstable patients while still visualizing sequelae of TBI that require immediate intervention (e.g., epidural hematoma). Follow-up MRI over more extended periods of time is necessary for a more accurate description of the degenerative changes after TBI (for a more detailed review of neuroimaging methods as applied to TBI, see Kurth & Bigler, 2008).

Major risk factors for TBI include very young age and old age, male

gender, and low socio-economic status (Corrigan, Selassie, & Orman, 2010). People incurring TBI frequently have preinjury comorbidities, which may range from learning disabilities in children to substance abuse in young adults to hypertension and diabetes mellitus in the elderly. Motor vehicle accidents are the most common cause of TBI from adolescence through young adulthood. Most cases of fatal TBI for this age group are firearm related, especially in African American males (Corrigan et al., 2010). Child abuse is the leading cause of severe TBI in infants and toddlers (Carty & Pierce, 2002), whereas older adults are at increased risk for TBI due to falls and associated subdural hematoma (Flanagan, Hibbard, Riordan, & Gordon, 2006; Langlois, Rutland-Brown, & Thomas, 2006). Recent military conflicts have also resulted in an increase in blast-related TBI, which is associated with specific risk factors that range from comorbid posttraumatic stress disorder (PTSD) in mild TBI to hypotensive/hypoxemic effects on the brain in more severe cases (DeWitt & Prough, 2009; Ling, Bandak, Armonda, Grant, & Ecklund, 2009).

### INITIAL ADMISSION TO REHABILITATION

As patients with TBI emerge from coma, problem behaviors such as confusion, agitation, and emotional lability are prominent. Formal neuropsychological tests are often not appropriate or possible, though brief but standardized measures aimed at tracking the patient's level of confusion and/or amnesia can be administered (Levin, O'Donnell, & Grossman, 1979). Because of ongoing posttraumatic amnesia, cognitive or insight-oriented treatments are also likely to be ineffective. However, there is evidence supporting the use of behavioral approaches at this stage to reduce agitation and to promote learning of motor and self-care skills through techniques like contingency management and positive behavior supports (Ylvisaker et al., 2007). There is a fairly rich amount of literature supporting the principles of applied behavioral analysis, particularly with regard to pediatric samples, although not specific to TBI (Slifer & Amari, 2009). To assuage more overt aggression and emotional lability, environmental modifications, such as reducing the number of visitors (Mysiw, Fugate, & Clinchot, 2007) and pharmacological approaches, such as use of beta-blockers (Warden et al., 2006) are also often indicated. Careful coordination with other rehabilitation team members is essential to ensure the consistency and specificity that are necessary for successful behavioral management after TBI (Beaulieu et al., 2008).

Another important role that can be undertaken by neuropsychologists at

this stage of rehabilitation is provision of evidence-based education to patients and families regarding natural recovery and prognosis. Research has also shown that a single, educational, outpatient therapy session can be very effective in the prevention of persistent symptomatology after mild TBI ([Paniak, Toller-Lobe, Reynolds, Melnyk, & Nagy, 2000](#)).

## **COGNITIVE SEQUELAE AND THEIR REHABILITATION**

There is no invariant “signature” cognitive profile after moderate to severe TBI; however, in general terms, impairments in the areas of attention, executive functioning, memory, and processing speed are common in both children and adults during the first year of recovery ([Goldstein & Levin, 2010](#); [Kirkwood, Yeates, & Bernstein, 2010](#); [Roebuck-Spencer, Baños, Sherer, & Novack, 2010](#)). Neuropsychological assessment can assist, across the life span, in identifying cognitive strengths and weaknesses as well as coping and adjustment styles, to provide suggestions for direction and mode of the most appropriate rehabilitation, to assist with differential diagnosis, and to track recovery in the context of community reintegration. There is convincing evidence that such assessment can provide incremental value to the prediction of outcome after TBI with measures of novel learning being the most robust in pediatrics ([Miller & Donders, 2003](#)), and measures of cognitive reserve, as well as processing speed and executive control having the greatest utility with adults ([Hanks et al., 2008](#)). Addition of formal measures of symptom validity is considered the standard of care in clinical neuropsychological assessment and should be included in the evaluation of persons with TBI ([Bush et al., 2005](#); [Heilbronner, Sweet, Morgan, Larrabee, & Millis, 2009](#)).

Rehabilitation of cognitive deficits should be undertaken in the context of evidence-based practice. Although cognitive rehabilitation represents a considerable proportion of intervention efforts after TBI, there is only moderate to limited evidence of its efficacy. Most studies that have been conducted in this arena are single-group pre–post test designs and few include detailed and useable information regarding efficiency and treatment strategies ([Rees, Marshall, Hartridge, Mackie, & Weiser, 2007](#)). Cognitive rehabilitation treatment should be undertaken early with targeted and pragmatic interventions as opposed to broad-based approaches with the futile hope of spontaneous generalization. There is sufficient empirical support for the rehabilitation of deficits in attention through specific skills training after TBI in both pediatric ([Galbiati et al., 2009](#); [Laatsch et al., 2007](#)) and adult samples ([Cicerone, 2007](#);

Rohling, Faust, Beverly, & Demakis, 2009). Specific guidelines have been developed in this regard (Sohlberg et al., 2003). In other cognitive domains, the use of environmental modifications and compensatory devices may be more appropriate for adults (Wilson et al., 2005), whereas children are often in need of special education services that include direct instruction and cognitive strategy interventions (Glang, Ylvisaker, et al., 2008). Constructive collaboration with other rehabilitation team members who address cognitive deficits, such as speech pathologists and classroom teachers, is highly desirable during cognitive rehabilitation (Wertheimer et al., 2008). Furthermore, such efforts are most successful when offered in a context in which the emotional difficulties of patients and families are addressed simultaneously in a holistic manner (Mateer, 2005, Wilson, 2008).

Although there is less robust evidence for the treatment of memory deficits after TBI, the literature does offer some guidance in this area. For example, teaching strategies that make memorization more effortful (e.g., techniques like rehearsal and visualization) may be effective, but mainly for those with mild deficits (Kaschel et al., 2002). For severe deficits, techniques such as errorless learning may be more effective (Clare & Jones, 2008; Ehlhardt et al., 2008; Sohlberg, 2005). Commonly, people with TBI also experience prospective memory problems. Remediation of such deficits typically involves utilization of compensatory strategies such as planners or electronic assistive devices. Unfortunately, only those with good insight and motivation will likely follow through with using such tools (Sohlberg et al., 2007). In general, although patients may report subjective improvement, there is no evidence to suggest that this type of therapy improves patients' quality of life (Bourgeois, Lenius, Turkstra, & Camp, 2007).

Another area commonly targeted in cognitive rehabilitation is executive functioning, particularly problem solving. Interventions in this domain include internalization and metacognitive strategies. Internalization techniques include teaching patients to "self-verbalize" as they problem solve. However, the associated research base is quite weak with uncontrolled research studies that lack detail in how to carry out these interventions (Cicerone, 2005). In metacognitive approaches to problem solving, therapists teach patients to set goals and subgoals and then evaluate and monitor them, with adjustment of the goal or approach as needed. Such methods are commonly used, but the research supporting their efficacy is weak with regard to application in young children or older adults. Randomized controlled studies of these techniques have shown that

both older pediatric and young adult patients with TBI can improve their problem solving and planning; however, processing takes longer and the strategies are not always maintained over time or generalized outside of familiar or other controlled settings (Kennedy et al., 2008; Mahone & Slomine, 2007). In this context, one needs to keep in mind that fatigability is common after TBI, and this may need supplemental interventions. These can include behavioral strategies such as teaching of pacing and sleep hygiene to pharmacological interventions that either promote wakefulness during the day (e.g., Provigil) or that target either initiation (e.g., Desyrel) or maintenance (e.g., Elavil) of sleep at night (Fellus & Elovic, 2007; Thaxton & Patel, 2007).

Insufficient self-awareness of cognitive deficits is a commonplace problem after moderate to severe TBI in both children and adults, even in those with acute sensitivity to physical losses, and who show only partial improvement over the first year postinjury (Hart, Seignourel, & Sherer, 2009). This can lead to poor compliance with treatment recommendations, longer lengths of stay in postacute rehabilitation, heightened caregiver distress, and worse educational and vocational outcomes (Flashman & McAllister, 2002). Neuropsychologists can assist in increasing awareness of deficits through the provision of education, direct feedback as patients complete tasks, or inviting self-appraisal by patients while using video-taped task prediction paradigms. The efficacy of these types of interventions is not well established. Results from research in this area are primarily from poorly controlled studies with small samples and insufficiently standardized therapeutic techniques (Fleming, Lucas, & Lightbody, 2006; Port, Willmott, & Charlton, 2002). Promise may be gleaned from one small, randomized controlled study in which self-awareness training using pretask prediction and posttask appraisal resulted in improved instrumental activities of daily living, even though there was no impact on general self-awareness or community integration (Goverover, Johnston, Togli, & DeLuca, 2007).

Whenever possible, cognitive rehabilitation should be coordinated with referral for special education services in the case of pediatrics (Hibbard, Martin, Cantor, & Moran, 2006) and vocational rehabilitation in the case of adults (Malec, 2005). In the former case, it is incumbent on the neuropsychologist to be aware of local and federal eligibility criteria as well as due process procedures and to facilitate transition between hospital and school (Glang et al., 2006). With regard to vocational rehabilitation, risk factors for failure to obtain or maintain competitive employment after adult TBI include older age, lower educational level, more severe cognitive impairment, physical debility, social skills deficits,

preinjury unemployment, and alcohol abuse (Tsaosides, Ashman, & Seter, 2008). At this time, there are really no evidence-based data to support one “best practice” standard for vocational rehabilitation (Fadyl & McPherson, 2009; Hart et al., 2006). However, neuropsychologists may assist with this process through the identification of cognitive profiles that can be mapped onto specific job responsibilities and need for any reasonable accommodations as well as identifying and treating interpersonal and other psychosocial risk factors, which could potentially impede a successful return to work. Collaboration with other rehabilitation professionals who can form liaisons with employers and possibly engage in on-site visits and job coaching is highly desirable. For the elderly who are no longer employed, some issues that may be more prevalent include the assessment of the patient’s ability to make informed decisions and/or to live independently (Goldstein, 2005). In addition, there is some evidence that older individuals with TBI may be at especially increased risk for driving-related problems (Brenner, Homaifar, & Schultheis, 2008). However, even with younger adults and adolescents, supplementing clinic-based assessment with formal, on-the-road driving evaluation is often advisable prior to making decisions about return to driving following moderate to severe TBI (Schanke & Sundet, 2000). At the same time, there are promising developments in the area of the potential application of driving simulators to assist with driver rehabilitation (Lew, Rosen, Thomander, & Poole, 2009).

## **EMOTIONAL AND PSYCHOSOCIAL SEQUELAE AND THEIR REHABILITATION**

TBI in both children and adults is associated with a strong risk for developing new adjustment and mood disorders as well as personality change in the postacute phase with affective lability, depression, and insufficient interpersonal pragmatics being relatively most common (Bloom et al., 2001; Max et al., 2006; Nicholl & LaFrance, 2009; Silver, Kramer, Greenwald, & Weissman, 2001). These various problems stem from a combination of direct organic (especially prefrontal) effects of TBI, adjustment reactions related to grief and loss issues, and moderating preinjury personality variables and coping style (Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008; Patterson & Staton, 2009).

There is controversy about the possibility of the co-occurrence of PTSD and TBI because of the paradox between amnesia for the event and later distress due to subjective reexperiences, but with milder injuries, the two do not appear to be completely mutually exclusive in either pediatric (Hajek et al., 2010) or

adult (Bryant et al., 2009) patients. When there appear to be overlapping symptoms without evidence for symptom magnification or other confounding issues, it is often advisable to treat the PTSD issues first, in light of recent findings that much of the subjective “postconcussion” symptoms in mild TBI as compared to moderate-severe TBI may be due to emotional distress (Belanger, Kretzmer, Vanderploeg, & French, 2010). There is empirical support for the efficacy of both cognitive behavioral therapy (Nemeroff et al., 2006) and pharmacotherapy (Davis, Frazier, Williford, & Newell, 2006) in the treatment of PTSD.

There is a much larger literature on depression following TBI. Primary and secondary depression after TBI are behaviorally similar syndromes with overlapping pathophysiological findings, but distinction between the two (which typically requires a thorough history and interview with family members) can allow the neuropsychologist to advise more appropriately regarding the propriety and timing of specific interventions (Moldover, Goldberg, & Prout, 2004). This is important because early identification and treatment of depression in TBI is associated with better longterm outcomes (Rao & Lyketsos, 2002). Ironically, although reduced deficit awareness can be a problem in terms of motivation for treatment, increased deficit awareness can be associated with a greater risk for reactive depression in both children (Barker-Collo, 2007) and adults (McBrinn et al., 2008), which suggests that neuropsychologists should include assessment of both of these domains in their evaluations of patients with TBI. Depression after TBI is linked with higher rates of disability, impaired psychosocial functioning, and decreased life satisfaction (Fleminger, Oliver, Williams, & Evans, 2003). In general, affective disturbance after TBI is at least as common as cognitive impairment and tends to persist for more prolonged periods of time; thus, improved emotional health should be just as much of a focus of rehabilitation (Prigatano, 2005).

Various studies on depression after TBI report a wide range of rates, from 26% to 46% in adult samples (Moldover et al., 2004; Seel, Macciocchi, & Kreutzer, 2010) but the rates may be even higher in elderly with TBI (Menzel, 2008). The timing of assessment may be relevant in this regard because many patients may not manifest the full spectrum of symptoms until well into the postacute recovery phase (Fleminger et al., 2003). In this context, it is also important to realize that some children who appear to adjust relatively well early on in their recovery may become much more distressed in later years and into young adulthood when they encounter more difficulties in school, greater

degrees of social isolation, and less autonomy with regard to issues such as driving (Anderson, Brown, Newitt, & Hoile, 2009; Beauchamp, Dooley, & Anderson, 2010). Therefore, longterm follow-up is necessary. Some authors also report that suicide rates are relatively higher in older adolescents and young adults with TBI (Perna, 2005). Neuropsychologists can play an important role in education of rehabilitation staff who have the most day-to-day contact with such patients to facilitate early identification of persons who might be at increased risk for self-harm (Wasserman et al., 2008). Active involvement of family members as equal partners in treatment planning is also desirable (Naar-King & Donders, 2006).

The diagnosis of depression following TBI may be seen as somewhat challenging, given the overlap among many depressive symptoms and common TBI sequelae; for example, psychomotor slowing, cognitive dysfunction, sleep problems, and fatigue. However, issues that can help to distinguish depression from other correlates of TBI include the fact that an egodystonic increase in irritability and frustration is relatively more common as the direct result of TBI on executive control, whereas a report of loss of enjoyment in previously enjoyed activities is relatively more suggestive of depression (Fleminger et al., 2003). Although it may be tempting to “correct” standard self-report inventories for the effect of such purported somatic or neurological symptoms, research has shown that this is ill-advised in persons who actually do have depression or other psychiatric comorbidities (Glassmire et al., 2003). Furthermore, particularly with adults in a medicolegal context, empirically established and well-validated symptom validity checks should be used routinely in the evaluation of persistent subjective self-reports of emotional dysfunction after TBI (Greiffenstein, Fox, & Lees-Haley, 2010; Nelson, Hoelzle, Sweet, Arbisi, & Demakis, 2010).

Typical treatments for depression after TBI include pharmacotherapy, psychotherapy, behavioral modification, and psychoeducation (Prigatano, Bargaro, & Caples, 2003). A recent literature review indicated that there is a paucity of randomized controlled trials for depression treatment following TBI, but that serotonergic antidepressants and cognitive behavioral interventions appear to have the relatively best evidence for efficacy in this condition (Fann, Hart, & Schomer, 2009). In selecting one or more of these approaches, optimal outcome is most likely when matching patient preference with an intervention (Fann, Jones, et al., 2009). Research also reveals strong patient acceptance and efficacy in reduction of mood problems for nontraditional approaches like telephone follow-up (Bell et al., 2005; Bombardier et al., 2009) and physical



exercise interventions ([Driver & Ede, 2009](#)).

If psychopharmacological approaches are selected as a treatment option for depression, typical choices of agents tend to mirror those used in the general population. Antidepressants, including selective serotonin reuptake inhibitors (SSRIs) and selective norepinephrine reuptake inhibitors (SNRIs), are commonly utilized, particularly in light of the fact that first-generation tricyclics have anticholinergic side effects that negatively affect cognition. It should be noted that Prozac is currently the only antidepressant that is specifically approved by the United States Food and Drug Administration for treatment of depression in children. Although there is some concern about the potential risk for increased suicidal thoughts, the general consensus in the field is that with regular follow up, the benefits of pharmacological treatment of depression in children and adolescents outweigh that risk ([Bridge et al., 2007](#); [Kratovich et al., 2006](#); [Rihmer & Akiskal, 2006](#)). At the other end of the age spectrum, SSRIs have been shown to be effective in the elderly but carry with them more side effects and drug–drug interactions that warrant special attention when such agents are prescribed to these patients ([Chemali, Chahine, & Fricchione, 2009](#)).

Wellbutrin may be particularly useful in cases of depression with apathy ([Corcoran, Wong, & O’Keane, 2004](#)). For patients with TBI who display persistent aggression or bipolar mood swings, pharmacotherapy may be more imperative than optional. Typical medications used in this population include atypical antipsychotics (save for Clozaril, which can lower seizure threshold), anticonvulsants such as Depakote and Lamictal, and lithium; the latter particularly in cases of people with premorbid manic symptoms ([Nicholl & LaFrance, 2009](#); [McAllister, 2007](#)).

Psychotherapy is another approach commonly selected for patients with TBI, particularly in the application of cognitive behavioral approaches, which are best offered as part of a more comprehensive or holistic program ([Cattelani, Zettin, & Zoccolotti, 2010](#)). Necessary ingredients for successful treatment include a strong therapeutic bond and some level of self-awareness ([Coetzer, 2007](#)). In addition, the patient and family’s cultural background needs to be taken into account ([Saltapidas & Ponsford, 2007](#)).

Psychotherapy in patients with TBI usually includes providing education, assisting the patient in building awareness of deficits, and helping the patient develop coping skills and compensatory strategies that will help her deal with not only mood but also cognitive issues. This is important because a person’s perceived self-efficacy for the management of cognitive symptoms has been

shown to be of great influence on global life satisfaction in patients with TBI (Cicerone & Azulay, 2007). Specific therapeutic techniques may include behavioral activation and cognitive restructuring focused on setting and working toward small and attainable goals. Depending on the degree of cognitive impairment, behavioral techniques may be more effective than cognitive methods when patients demonstrate considerable inflexibility of thinking. In addition, there can be a lack of carryover of therapeutic discussion; for that reason when working with people with TBI, clinicians should use memory aids such as written summaries of techniques or have a significant other involved in treatment to assist in carryover. In some cases, having shortened, focused sessions may be more effective (Judd & Wilson, 2005). There is also evidence that discrepancies between family and clinician perceptions of patient functioning tend to result in poorer therapeutic alliance and poorer patient effort in therapies; therefore, neuropsychologists should address family perceptions and functioning to facilitate patient investment in therapy (Sherer et al., 2007). Group therapy may be more helpful in the later stages of recovery when increased awareness has developed (Anson & Ponsford, 2006). Combining individual and group interventions may be associated with greater maintenance of gains over time (Ownsworth, Fleming, Shum, Kuipers, & Strong, 2008). Another issue that may need to be addressed with more insightful patients is the issue of attribution of blame for the injury because this can also contribute to emotional distress (Hart, Hanks, Bogner, Millis, & Esselman, 2007).

Social isolation and impairment is a prominent problem with moderate to severe TBI in both children and adults, once the level of progress has leveled off and individuals have still not returned to the “old self” (Hawthorne, Gruen, & Kaye, 2009; McCabe et al., 2007). This pattern may persist for years after injury after adult TBI (Sloan, Winkler, & Anson, 2007) and also remains problematic into adulthood after moderate to severe TBI that was sustained in early childhood (Donders & Warschausky, 2007). Issues such as reduction in social network size and loss of friendships likely arise from the disruption in behavior that is common in TBI. In addition to obviously inappropriate behavior like disinhibition and lability, specific and more subtle deficits that may contribute to social interaction include problems with social communication and interpersonal cue perception (Knox & Douglas, 2009; Struchen et al., 2008). Typically, the rehabilitation of social skills includes working with patients on developing pragmatic behaviors like social reciprocity, initiation, staying “on topic,” turn taking, active listening, and reading social cues. These skills can be developed

through behavioral interventions like shaping and cueing, or in group or individual therapy contexts through coaching and role playing (O'Reilly, Lancioni, & O'Kane, 2000).

Empirical evidence for the treatment of social skills deficit after TBI is limited, and it is unclear whether the treatment effects found in this domain can be generalized (Ylvisaker, Turkstra, & Coelho, 2005). There have been some small, randomized controlled trials that bode well. There is some initial evidence that social skills training can improve both skills and overall life satisfaction of adults with TBI with some maintenance of gains on follow-up (Dahlberg et al., 2007). One recent, small, randomized, controlled clinical trial also provided some modest evidence for the feasibility of remediation of emotional perception deficits after TBI (Bornhofen & McDonald, 2008). Research on social skills groups seems to suggest that improvement in a specific skill may be seen though this does not necessarily translate to improvements in mood or general social behavior (McDonald et al., 2008). A recent encouraging development is the implementation of web-based technology to serve adolescents with TBI who otherwise might not be likely to attend office-based treatment (Wade, Walz, Carey, & Williams, 2009).

It is important for the neuropsychologist to realize that families of both pediatric and adult patients with TBI often experience emotional distress and burden, and that this may persist for many years postinjury (Josie et al., 2008; Kreutzer, Marwitz, Godwin, & Arango-Lasprilla, 2010). Consequently, fostering community support and family cohesion is important, with special attention to disruptions in role distribution and communication (Noggle & Pierson, 2010; Sander, 2005). In this context, consideration needs to be given not only to the level of disability of the patient but also to the possible contribution of premorbid family dysfunction, as well as level of current support resources (Ergh, Rapport, Coleman, & Hanks, 2002; Wade, Taylor, et al., 2006). Caregivers' preinjury functioning may influence their global distress, whereas the functioning of the person with injury may be more related to subjective injury-related burden (Davis et al., 2009). It may be helpful to teach the family members pragmatic strategies to cope with adult or pediatric patient neurobehavioral problems, in addition to providing emotional support and helping the family identify community resources. Self-care and respite should also be encouraged (Kreutzer et al., 2010). However, although the area of family burden and distress is well researched in the TBI literature, studies looking at the efficacy of treatments to address these issues are limited in methodological rigor,

and there is no strong evidence at this time supporting any specific intervention method for family caregivers of adult individuals with TBI (Boschen, Gargaro, Gan, Gerber, & Brandys, 2007). Still, there is some encouraging evidence from randomized clinical trials for the feasibility of web-based family problem-solving therapy after pediatric TBI (Wade, Carey, & Wolfe, 2006a, 2006b).

Substance abuse is also an important issue to address in rehabilitation, especially with adolescents and young adults. Premorbid alcohol abuse tends to be associated with worse outcomes after TBI in the areas of employment, independent living, neurobehavioral functioning, and satisfaction with life (Corrigan, 2005). Risk factors for alcohol abuse after TBI include relatively young age and high frequency of prior use. Problems associated with overconsumption of alcohol after TBI include decreased tolerance for its effects, reduction of seizure threshold, and interactions with prescription medications. The most common treatment strategies include motivational interviewing, provision of education regarding the ill effects of alcohol after TBI, and case management. There has been one randomized control trial in the area of alcohol abuse after TBI. Corrigan and Bogner (2007) found that providing monetary incentive in a substance-abuse program improved attendance and premature termination.

## **SUMMARY AND SUGGESTIONS FOR THE FUTURE**

TBI is one of the most common causes of acquired cognitive and emotional-behavioral morbidity across the developmental continuum. Neuropsychologists can play an important role in the assessment and treatment of neurobehavioral sequelae of TBI. There is a dire need, however, for more randomized, controlled, clinical trials to demonstrate the most effective and value-added interventions, and how these differ across the life span. In addition, a longterm perspective is needed in the care of persons with severe TBI, who often have persistent difficulties with community integration and participation for many years postinjury. The greatest gaps in the knowledge base currently appear to be at the extremes of the age range, with uncertainty about things such as how to define injury severity in infants and toddlers, or how to determine the possible impact of an apparently mild TBI in an elderly person with a preexisting history of mild cognitive impairment or early-stage dementia. A comprehensive, developmental, biopsychosocial model that includes all possible variables—ranging from biomarkers to community supports that directly affect recovery from TBI and that also explores the factors that moderate and mediate these influences—is a

goal for scientist–practitioners in this field.

## REFERENCES

- Anderson, V., Brown, S., Newitt, H., & Hoile, H. (2009). Educational, vocational, psychosocial, and quality-of-life outcomes for adult survivors of childhood traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 24*(5), 303–312.
- Anderson, V. A., Catroppa, C., Dudgeon, P., Morse, S. A., Haritou, F., & Rosenfeld, J. V. (2006). Understanding predictors of functional recovery and outcome 30 months following early childhood head injury. *Neuropsychology, 20*(1), 42–57.
- Anson, K., & Ponsford, J. (2006). Evaluation of a coping skills group following traumatic brain injury. *Brain Injury, 20*(2), 167–178.
- Babikian, T., & Asarnow, R. (2009). Neurocognitive outcomes and recovery after pediatric TBI: Meta-analytic review of the literature. *Neuropsychology, 23*(3), 283–296.
- Barker-Collo, S. L. (2007). Behavioral profiles and injury severity following childhood traumatic brain injury. *Brain Impairment, 8*, 22–30.
- Bauer, R., & Fritz, H. (2004). Pathophysiology of traumatic brain injury in the developing brain: An introduction and short update. *Experimental and Toxicologic Pathology, 56*, 65–73.
- Beauchamp, M., Dooley, J., & Anderson, V. (2010). Adult outcomes of pediatric traumatic brain injury. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 315–328). New York, NY: Cambridge University Press.
- Beaulieu, C., Wertheimer, J. C., Pickett, L., Spierre, L., Schnorbus, T., Healy, W., . . . Jones, A. (2008). Behavior management on an acute brain injury unit: Evaluating the effectiveness of an interdisciplinary training program. *Journal of Head Trauma Rehabilitation, 23*(5), 304–311.
- Belanger, H. G., Kretzmer, T., Vanderploeg, R. D., & French, L. M. (2010). Symptom complaints following combat-related traumatic brain injury: Relationship to traumatic brain injury severity and posttraumatic stress disorder. *Journal of the International Neuropsychological Society, 16*(1), 194–199.
- Bell, K. R., Temkin, N. R., Esselman, P. C., Doctor, J. N., Bombardier, C. H., Fraser, R. T., . . . Dikmen, S. (2005). The effect of a scheduled telephone intervention on outcome after moderate to severe traumatic brain injury: A randomized trial. *Archives of Physical Medicine and Rehabilitation, 86*(5), 851–856.
- Bloom, D. R., Levin, H. S., Ewing-Cobbs, L., Saunders, A. E., Song, J., Fletcher, J. M., & Kowatch, R. A. (2001). Lifetime and novel psychiatric disorders after pediatric traumatic brain injury. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(5), 572–579.
- Bombardier, C. H., Bell, K. R., Temkin, N. R., Fann, J. R., Hoffman, J., & Dikmen, S. (2009). The efficacy of a scheduled telephone intervention for ameliorating depressive symptoms during the first year after traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24*(4), 230–238.
- Bornhofen, C., & McDonald, S. (2008). Comparing strategies for treating emotion perception deficits in traumatic brain injury. *Journal of Head Trauma Rehabilitation, 23*(2), 103–115.
- Boschen, K., Gargaro, J., Gan, C., Gerber, G., & Brandys, C. (2007). Family interventions after acquired brain injury and other chronic conditions: a critical appraisal of the quality of the evidence. *Neuro Rehabilitation, 22*(1), 19–41.
- Bourgeois, M. S., Lenius, K., Turkstra, L., & Camp, C. (2007). The effects of cognitive teletherapy on reported everyday memory behaviours of persons with chronic traumatic brain injury. *Brain Injury, 21*(12), 1245–1257.
- Brenner, L. A., Homaifar, B. Y., & Schultheis, M. T. (2008). Driving, aging, and traumatic brain injury: Integrating findings from the literature. *Rehabilitation Psychology, 53*, 18–27.
- Bridge, J. A., Iyengar, S., Salary, C. B., Barbe, R. P., Birmaher, B., Pincus, H. A., . . . Brent, D. A. (2007). Clinical response and risk for reported suicidal ideation and suicide attempts in pediatric antidepressant treatment: A meta-analysis of randomized controlled trials. *Journal of the American Medical*

- Association*, 297(15), 1683–1696.
- Bryant, R. A., Creamer, M., O'Donnell, M., Silove, D., Clark, C. R., & McFarlane, A. C. (2009). Posttraumatic amnesia and the nature of posttraumatic stress disorder after mild traumatic brain injury. *Journal of the International Society*, 15(6), 862–867.
- Bush, S. S., Ruff, R. M., Tröster, A. I., Barth, J. T., Koffler, S. P., Pliskin, N. H., . . . Silver, C. H. (2005). Symptom validity assessment: Practice issues and medical necessity: NAN Policy & Planning Committee. *Archives of Clinical Neuropsychology*, 20(4), 419–426.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., . . . WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. (2004). Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, 43, 84–105.
- Carty, H., & Pierce, A. (2002). Non-accidental injury: A retrospective analysis of a large cohort. *European Radiology*, 12(12), 2919–2925.
- Catroppa, C., Anderson, V. A., Morse, S. A., Haritou, F., & Rosenfeld, J. V. (2008). Outcome and predictors of functional recovery 5 years following pediatric traumatic brain injury (TBI). *Journal of Pediatric Psychology*, 33(7), 707–718.
- Cattalani, R., Zettin, M., & Zoccolotti, P. (2010). Rehabilitation treatments for adults with behavioral and psychosocial disorders following acquired brain injury: A systematic review. *Neuropsychology Review*, 20(1), 52–85.
- Chemali, Z., Chahine, L. M., & Fricchione, G. (2009). The use of selective serotonin reuptake inhibitors in elderly patients. *Harvard Review of Psychiatry*, 17(4), 242–253.
- Cicerone, K. D. (2005). Rehabilitation of executive function impairments. In W. M. High, A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 71–87). New York, NY: Oxford.
- Cicerone, K. D. (2007). Cognitive rehabilitation. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 765–777). New York, NY: Demos.
- Cicerone, K. D., & Azulay, J. (2007). Perceived self-efficacy and life satisfaction after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 22(5), 257–266.
- Clare, L., & Jones, R. S. (2008). Errorless learning in the rehabilitation of memory impairment: A critical review. *Neuropsychology Review*, 18(1), 1–23.
- Coetzer, R. (2007). Psychotherapy following traumatic brain injury: Integrating theory and practice. *Journal of Head Trauma Rehabilitation*, 22(1), 39–47.
- Corcoran, C., Wong, M. L., & O'Keane, V. (2004). Bupropion in the management of apathy. *Journal of Psychopharmacology*, 18(1), 133–135.
- Corrigan, J. D. (2005). Substance abuse. In W. M. High, A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 133–155). New York, NY: Oxford.
- Corrigan, J. D., & Bogner, J. (2007). Interventions to promote retention in substance abuse treatment. *Brain Injury*, 21(4), 343–356.
- Corrigan, J. D., Selassie, A. W., & Orman, J. A. (2010). The epidemiology of traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 25(2), 72–80.
- Cullen, N., Chundamala, J., Bayley, M., & Jutai, J.; Erabi Group. (2007). The efficacy of acquired brain injury rehabilitation. *Brain Injury*, 21(2), 113–132.
- Dahlberg, C. A., Cusick, C. P., Hawley, L. A., Newman, J. K., Morey, C. E., Harrison-Felix, C. L., & Whiteneck, G. G. (2007). Treatment efficacy of social communication skills training after traumatic brain injury: A randomized treatment and deferred treatment controlled trial. *Archives of Physical Medicine and Rehabilitation*, 88(12), 1561–1573.
- Davis, L. C., Sander, A. M., Struchen, M. A., Sherer, M., Nakase-Richardson, R., & Malec, J. F. (2009). Medical and psychosocial predictors of caregiver distress and perceived burden following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 24(3), 145–154.
- Davis, L. L., Frazier, E. C., Williford, R. B., & Newell, J. M. (2006). Longterm pharmacotherapy for

- posttraumatic stress disorder. *CNS Drugs*, 20(6), 465–476.
- DeWitt, D. S., & Prough, D. S. (2009). Blast-induced brain injury and posttraumatic hypotension and hypoxemia. *Journal of Neurotrauma*, 26(6), 877–887.
- Donders, J., & Warschawsky, S. (2007). Neurobehavioral outcomes after early versus late childhood traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 22(5), 296–302.
- Driver, S., & Ede, A. (2009). Impact of physical activity on mood after TBI. *Brain Injury*, 23(3), 203–212.
- Ehlhardt, L. A., Sohlberg, M. M., Kennedy, M., Coelho, C., Ylvisaker, M., Turkstra, L., & Yorkston, K. (2008). Evidence-based practice guidelines for instructing individuals with neurogenic memory impairments: What have we learned in the past 20 years? *Neuropsychological Rehabilitation*, 18(3), 300–342.
- Ergh, T. C., Rapport, L. J., Coleman, R. D., & Hanks, R. A. (2002). Predictors of caregiver and family functioning following traumatic brain injury: social support moderates caregiver distress. *Journal of Head Trauma Rehabilitation*, 17(2), 155–174.
- Fadyl, J. K., & McPherson, K. M. (2009). Approaches to vocational rehabilitation after traumatic brain injury: A review of the evidence. *Journal of Head Trauma Rehabilitation*, 24(3), 195–212.
- Fann, J. R., Hart, T., & Schomer, K. G. (2009). Treatment for depression after traumatic brain injury: A systematic review. *Journal of Neurotrauma*, 26(12), 2383–2402.
- Fann, J. R., Jones, A. L., Dikmen, S. S., Temkin, N. R., Esselman, P. C., & Bombardier, C. H. (2009). Depression treatment preferences after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 24(4), 272–278.
- Faul, M., Xu, L., Wald, M. M., & Coronado, V. G. (2010). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths 2002–2006*. Atlanta, GA: Centers for Disease Control and Prevention.
- Fellus, J. L., & Elovic, E. P. (2007). Fatigue: Assessment and treatment. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 545–555). New York, NY: Demos.
- Flanagan, S. R., Hibbard, M. R., Riordan, B., & Gordon, W. A. (2006). Traumatic brain injury in the elderly: Diagnostic and treatment challenges. *Clinics in Geriatric Medicine*, 22(2), 449–68; x.
- Flashman, L. A., & McAllister, T. W. (2002). Lack of awareness and its impact in traumatic brain injury. *Neuro Rehabilitation*, 17(4), 285–296.
- Fleming, J. M., Lucas, S. E., & Lightbody, S. (2006). Using occupation to facilitate self-awareness in people who have acquired brain injury: A pilot study. *Canadian Journal of Occupational Therapy*, 73(1), 44–55.
- Fleminger, S., Oliver, D. L., Williams, W. H., & Evans, J. (2003). The neuropsychiatry of depression after brain injury. *Neuropsychological Rehabilitation*, 13(1–2), 65–87.
- Galbiati, S., Recla, M., Pastore, V., Liscio, M., Bardoni, A., Castelli, E., & Strazzer, S. (2009). Attention remediation following traumatic brain injury in childhood and adolescence. *Neuropsychology*, 23(1), 40–49.
- Glang, A., Todis, B., Thomas, C. W., Hood, D., Bedell, G., & Cockrell, J. (2008). Return to school following childhood TBI: Who gets services? *Neuro Rehabilitation*, 23(6), 477–486.
- Glang, A., Ylvisaker, M., Stein, M., Ehlhardt, L., Todis, B., & Tyler, J. (2008). Validated instructional practices: Application to students with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 23(4), 243–251.
- Glassmire, D. M., Kinney, D. I., Greene, R. L., Stolberg, R. A., Berry, D. T., & Cripe, L. (2003). Sensitivity and specificity of MMPI–2 neurologic correction factors: Receiver operating characteristic analysis. *Assessment*, 10(3), 299–309.
- Goldstein, F. C. (2005). Older adults. In W. M. High, Jr., A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 235–246). New York: Oxford.
- Goldstein, F. C., & Levin, H. S. (2010). *Traumatic brain injury in older adults*. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 345–355). New York, NY: Cambridge.

- Goverover, Y., Johnston, M. V., Toglia, J., & Deluca, J. (2007). Treatment to improve self-awareness in persons with acquired brain injury. *Brain Injury, 21*(9), 913–923.
- Greiffenstein, M. F., Fox, D., & Lees-Haley, P. R. (2010). The MMPI-2 Fake Bad Scale in detection of noncredible brain injury claims. In K. B. Boone (Ed.), *Assessment of feigned cognitive impairment: A neuropsychological perspective* (pp. 210–235). New York, NY: Guilford Press.
- Hajek, C. A., Yeates, K. O., Gerry Taylor, H., Bangert, B., Dietrich, A., Nuss, K. E., . . . Wright, M. (2010). Relationships among postconcussive symptoms and symptoms of PTSD in children following mild traumatic brain injury. *Brain Injury, 24*(2), 100–109.
- Hanks, R. A., Millis, S. R., Ricker, J. H., Giacino, J. T., Nakese-Richardson, R., Frol, A. B., . . . Gordon, W. A. (2008). The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89*(5), 950–957.
- Hart, T., Dijkers, M., Fraser, R., Cicerone, K., Bogner, J. A., Whyte, J., . . . Waldron, B. (2006). Vocational services for traumatic brain injury: Treatment definition and diversity within model systems of care. *Journal of Head Trauma Rehabilitation, 21*(6), 467–482.
- Hart, T., Hanks, R., Bogner, J. A., Millis, S., & Esselman, P. (2007). Blame attribution in intentional and unintentional traumatic brain injury: Longitudinal changes and impact on subjective well-being. *Rehabilitation Psychology, 52*, 152–161.
- Hart, T., Seignourel, P. J., & Sherer, M. (2009). A longitudinal study of awareness of deficit after moderate to severe traumatic brain injury. *Neuropsychological Rehabilitation, 19*(2), 161–176.
- Hawthorne, G., Gruen, R. L., & Kaye, A. H. (2009). Traumatic brain injury and longterm quality of life: Findings from an Australian study. *Journal of Neurotrauma, 26*(10), 1623–1633.
- Heilbronner, R. L., Sweet, J. J., Morgan, J. E., Larrabee, G. J., & Millis, S. R.; Conference Participants. (2009). American Academy of Clinical Neuropsychology Consensus Conference Statement on the neuropsychological assessment of effort, response bias, and malingering. *Clinical Neuropsychologist, 23*(7), 1093–1129.
- Hibbard, M. R., Martin, T., Cantor, J., & Moran, A. I. (2006). Students with acquired brain injury: Identification, accommodations, and transitions in the schools. In J. E. Farmer, J. Donders, & S. Warschawsky (Eds.), *Treating neurodevelopmental disabilities: Clinical research and practice* (pp. 208–233). New York, NY: Guilford Press.
- Josie, K. L., Peterson, C. C., Burant, C., Drotar, D., Stancin, T., Wade, S. L., . . . Taylor, H. G. (2008). Predicting family burden following childhood traumatic brain injury: A cumulative risk approach. *Journal of Head Trauma Rehabilitation, 23*(6), 357–368.
- Judd, D., & Wilson, S. L. (2005). Psychotherapy with brain injury survivors: An investigation of the challenges encountered by clinicians and their modifications to therapeutic practice. *Brain Injury, 19*(6), 437–449.
- Kaschel, R., Della-Sala, S., Cantagallo, A., Fahbock, A., Laaksonen, R., & Kazen, M. (2002). Imagery mnemonics for the rehabilitation of memory: A randomized control trial. *Neuropsychological Rehabilitation, 12*, 127–153.
- Kennedy, M. R., Coelho, C., Turkstra, L., Ylvisaker, M., Moore, S. M., Yorkston, K., . . . Kan, P. F. (2008). Intervention for executive functions after traumatic brain injury: A systematic review, meta-analysis and clinical recommendations. *Neuropsychological Rehabilitation, 18*(3), 257–299.
- Kirkwood, M. W., Yeates, K. O., & Bernstein, J. H. (2010). Traumatic brain injury in childhood. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 299–313). New York, NY: Cambridge.
- Knox, L., & Douglas, J. (2009). Longterm ability to interpret facial expression after traumatic brain injury and its relation to social integration. *Brain and Cognition, 69*(2), 442–449.
- Kothari, S. (2007). Prognosis after severe TBI: A practical, evidence-based approach. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 169–199). New York, NY: Demos.
- Kratochvil, C. J., Vitiello, B., Walkup, J., Emslie, G., Waslick, B. D., Weller, E. B., . . . March, J. S. (2006).



- Selective serotonin reuptake inhibitors in pediatric depression: Is the balance between benefits and risks favorable? *Journal of Child and Adolescent Psychopharmacology*, 16(1–2), 11–24.
- Kreutzer, J. S., Marwitz, J. H., Godwin, E. E., & Arango-Lasprilla, J. C. (2010). Practical approaches to effective family intervention after brain injury. *Journal of Head Trauma Rehabilitation*, 25(2), 113–120.
- Kurth, S., & Bigler, E. D. (2008). Structural neuroimaging in clinical neuropsychology. In J. E. Morgan & J. H. Ricker (Eds.), *Textbook of clinical neuropsychology* (pp. 783–839). New York, NY: Taylor & Francis.
- Laatsch, L., Harrington, D., Hotz, G., Marcantuono, J., Mozzoni, M. P., Walsh, V., & Hersey, K. P. (2007). An evidence-based review of cognitive and behavioral rehabilitation treatment studies in children with acquired brain injury. *Journal of Head Trauma Rehabilitation*, 22(4), 248–256.
- Langlois, J. A., Rutland-Brown, W., & Thomas, K. E. (2006). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths*. Atlanta, GA: Centers for Disease Control and Prevention.
- Levin, H. S., O'Donnell, V. M., & Grossman, R. G. (1979). The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *Journal of Nervous and Mental Disease*, 167(11), 675–684.
- Lew, H. L., Rosen, P. N., Thomander, D., & Poole, J. H. (2009). The potential utility of driving simulators in the cognitive rehabilitation of combat-returnees with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 24(1), 51–56.
- Ling, G., Bandak, F., Armonda, R., Grant, G., & Ecklund, J. (2009). Explosive blast neurotrauma. *Journal of Neurotrauma*, 26(6), 815–825.
- Mahone, E., & Slomine, B. S. (2007). Managing dysexecutive disorders. In S. J. Hunter & J. Donders (Eds.), *Pediatric neuropsychological intervention* (pp. 287–313). New York, NY: Cambridge.
- Malec, J. F. (2005). Vocational rehabilitation. In W. M. High, A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 176–201). New York, NY: Oxford.
- Mateer, C. A. (2005). Fundamentals of cognitive rehabilitation. In P. W. Haligan & D. T. Wade (Eds.), *Effectiveness of rehabilitation for cognitive deficits* (pp. 21–29). New York, NY: Oxford.
- Max, J. E., Levin, H. S., Schachar, R. J., Landis, J., Saunders, A. E., Ewing-Cobbs, L., . . . Dennis, M. (2006). Predictors of personality change due to traumatic brain injury in children and adolescents six to twenty-four months after injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 18(1), 21–32.
- McAllister, T. W. (2007). Neuropsychiatric aspects of TBI. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 835–861). New York, NY: Demos.
- McBrinn, J., Colin Wilson, F., Caldwell, S., Carton, S., Delargy, M., McCann, J., . . . McGuire, B. (2008). Emotional distress and awareness following acquired brain injury: An exploratory analysis. *Brain Injury*, 22(10), 765–772.
- McCabe, P., Lippert, C., Weiser, M., Hilditch, M., Hartridge, C., & Villamere, J.; Erabi Group. (2007). Community reintegration following acquired brain injury. *Brain Injury*, 21(2), 231–257.
- McDonald, S., Tate, R., Togher, L., Bornhofen, C., Long, E., Gertler, P., & Bowen, R. (2008). Social skills treatment for people with severe, chronic acquired brain injuries: A multicenter trial. *Archives of Physical Medicine and Rehabilitation*, 89(9), 1648–1659.
- Menzel, J. C. (2008). Depression in the elderly after traumatic brain injury: A systematic review. *Brain Injury*, 22(5), 375–380.
- Miller, L. J., & Donders, J. (2003). Prediction of educational outcome after pediatric traumatic brain injury. *Rehabilitation Psychology*, 48, 237–241.
- Moldover, J. E., Goldberg, K. B., & Prout, M. F. (2004). Depression after traumatic brain injury: A review of evidence for clinical heterogeneity. *Neuropsychology Review*, 14(3), 143–154.
- Mysiw, W. J., Fugate, L. P., & Clinchot, D. M. (2007). Assessment, early rehabilitation intervention, and tertiary prevention. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 283–301). New York, NY: Demos.

- Naar-King, S., & Donders, J. (2006). Pediatric family-centered rehabilitation. In J. E. Farmer, J. Donders, & S. Warschausky (Eds.), *Treating neurodevelopmental disabilities: Clinical research and practice* (pp. 149–169). New York, NY: Guilford Press.
- Nelson, N. W., Hoelzle, J. B., Sweet, J. J., Arbisi, P. A., & Demakis, G. J. (2010). Updated meta-analysis of the MMPI-2 symptom validity scale (FBS): Verified utility in forensic practice. *Clinical Neuropsychologist*, *24*(4), 701–724.
- Nemeroff, C. B., Bremner, J. D., Foa, E. B., Mayberg, H. S., North, C. S., & Stein, M. B. (2006). Posttraumatic stress disorder: A state-of-the-science review. *Journal of Psychiatric Research*, *40*(1), 1–21.
- Nicholl, J., & LaFrance, W. C. (2009). Neuropsychiatric sequelae of traumatic brain injury. *Seminars in Neurology*, *29*(3), 247–255.
- Noggle, C. A., & Pierson, E. E. (2010). Psychosocial and behavioral functioning following pediatric TBI: Presentation, assessment, and intervention. *Applied Neuropsychology*, *17*(2), 110–115.
- O'Reilly, M. F., Lancioni, G. E., & O'Kane, N. (2000). Using a problem-solving approach to teach social skills to workers with brain injuries in supported employment settings. *Journal of Vocational Rehabilitation*, *14*, 187–193.
- Owensworth, T., Fleming, J., Shum, D., Kuipers, P., & Strong, J. (2008). Comparison of individual, group and combined intervention formats in a randomized controlled trial for facilitating goal attainment and improving psychosocial function following acquired brain injury. *Journal of Rehabilitation medicine*, *40*(2), 81–88.
- Paniak, C., Toller-Lobe, G., Reynolds, S., Melnyk, A., & Nagy, J. (2000). A randomized trial of two treatments for mild traumatic brain injury: 1 year follow-up. *Brain Injury*, *14*(3), 219–226.
- Patterson, F. L., & Staton, A. R. (2009). Adult acquired traumatic brain injury: Existential implications and clinical considerations. *Journal of Mental Health Counseling*, *31*, 149–163.
- Perdices, M., Schultz, R., Tate, R. L., McDonald, S., Togher, L., Savage, S., . . . Smith, S. (2006). The evidence base of neuropsychological rehabilitation in acquired brain impairment: How good is the research? *Brain Impairment*, *7*, 119–132.
- Perna, R. (2005). Major depression and suicidality following brain injury. *Journal of Cognitive Rehabilitation*, *23*, 5–7.
- Port, A., Willmott, C., & Charlton, J. (2002). Self-awareness following traumatic brain injury and implications for rehabilitation. *Brain Injury*, *16*(4), 277–289.
- Prigatano, G. P. (2005). Therapy for emotional and motivational disorders. In W. M. High, A.M. Sander, M.A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 118–130). New York, NY: Oxford.
- Prigatano, G. P., Bargaro, S., & Caples, H. (2003). Non-pharmacological management of psychiatric disturbances after traumatic brain injury. *International Review of Psychiatry*, *15*, 371–379.
- Prvu Bettger, J. A., & Stineman, M. G. (2007). Effectiveness of multidisciplinary rehabilitation services in postacute care: State-of-the-science. A review. *Archives of Physical Medicine and Rehabilitation*, *88*(11), 1526–1534.
- Rao, V., & Lyketsos, C. G. (2002). Psychiatric aspects of traumatic brain injury. *Psychiatric Clinics of North America*, *25*(1), 43–69.
- Rao, V., Bertrand, M., Rosenberg, P., Makley, M., Schretlen, D. J., Brandt, J., & Mielke, M. M. (2010). Predictors of new-onset depression after mild traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, *22*(1), 100–104.
- Rees, L., Marshall, S., Hartridge, C., Mackie, D., & Weiser, M.; Erabi Group. (2007). Cognitive interventions post acquired brain injury. *Brain Injury*, *21*(2), 161–200.
- Rihmer, Z., & Akiskal, H. (2006). Do antidepressants t(h)reat(en) depressives? Toward a clinically judicious formulation of the antidepressant-suicidality FDA advisory in light of declining national suicide statistics from many countries. *Journal of Affective Disorders*, *94*(1–3), 3–13.
- Roebuck-Spencer, T., Baños, J., Sherer, M., & Novack, T. (2010). Neurobehavioral aspects of traumatic

- brain injury sustained in adulthood. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 329–343). New York, NY: Cambridge.
- Rohling, M. L., Faust, M. E., Beverly, B., & Demakis, G. (2009). Effectiveness of cognitive rehabilitation following acquired brain injury: A meta-analytic re-examination of Cicerone *et al.*'s (2000, 2005) systematic reviews. *Neuropsychology*, *23*(1), 20–39.
- Saltapidas, H., & Ponsford, J. (2007). The influence of cultural background on motivation for and participation in rehabilitation and outcome following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *22*(2), 132–139.
- Sander, A. M. (2005). Interventions for caregivers. In W. M. High, A. M., Sander, M. A., Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 156–175). New York, NY: Oxford.
- Schanke, A. K., & Sundet, K. (2000). Comprehensive driving assessment: neuropsychological testing and on-road evaluation of brain injured patients. *Scandinavian Journal of Psychology*, *41*(2), 113–121.
- Seel, R. T., Macciocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major depression after moderate to severe TBI. *Journal of Head Trauma Rehabilitation*, *25*(2), 99–112.
- Sherer, M., Evans, C. C., Leverenz, J., Stouter, J., Irby, J. W., Lee, J. E., & Yablon, S. A. (2007). Therapeutic alliance in postacute brain injury rehabilitation: predictors of strength of alliance and impact of alliance on outcome. *Brain Injury*, *21*(7), 663–672.
- Selassie, A. W., Zaloshnja, E., Langlois, J. A., Miller, T., Jones, P., & Steiner, C. (2008). Incidence of longterm disability following traumatic brain injury hospitalization, United States, 2003. *Journal of Head Trauma Rehabilitation*, *23*(2), 123–131.
- Silver, J. M., Kramer, R., Greenwald, S., & Weissman, M. (2001). The association between head injuries and psychiatric disorders: findings from the New Haven NIMH Epidemiologic Catchment Area Study. *Brain Injury*, *15*(11), 935–945.
- Slifer, K. J., & Amari, A. (2009). Behavior management for children and adolescents with acquired brain injury. *Developmental Disabilities Research Reviews*, *15*(2), 144–151.
- Sloan, S., Winkler, D., & Anson, K. (2007). Longterm outcome following traumatic brain injury. *Brain Impairment*, *8*, 251–261.
- Smits, M., Hunink, M. G., van Rijssel, D. A., Dekker, H. M., Vos, P. E., Kool, D. R., . . . Dippel, D. W. (2008). Outcome after complicated minor head injury. *American Journal of Neuroradiology*, *29*(3), 506–513.
- Sohlberg, M. M. (2005). External aids for management of memory impairment. In W. M. High, A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation for traumatic brain injury* (pp. 47–70). New York, NY: Oxford.
- Sohlberg, M. M., Avery, J., Kennedy, M., Ylvisaker, M., Coelho, C., Turkstra, L., & Yorkston, K. (2003). Practice guidelines for direct attention training. *Journal of Medical Speech Language Pathology*, *11*, 19–39.
- Sohlberg, M. M., Kennedy, M. R. T., Avery, J., Coelho, C., Turkstra, L., Ylvisaker, M., & Yorkston, K. (2007). Evidence based practice for the use of external aids as a memory rehabilitation technique. *Journal of Medical Speech Pathology*, *15*, xv-li.
- Struchen, M. A., Clark, A. N., Sander, A. M., Mills, M. R., Evans, G., & Kurtz, D. (2008). Relation of executive functioning and social communication measures to functional outcomes following traumatic brain injury. *NeuroRehabilitation*, *23*, 185–198.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, *2*(7872), 81–84.
- Teasell, R., Bayona, N., Marshall, S., Cullen, N., Bayley, M., Chundamala, J., . . . Tu, L. (2007). A systematic review of the rehabilitation of moderate to severe acquired brain injuries. *Brain Injury*, *21*(2), 107–112.
- Thaxton, L. L., & Patel, A. R. (2007). Sleep disturbances: Epidemiology, assessment, and treatment. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 557–575). New York, NY: Demos.

- Tsauosides, T., Ashman, T., & Seter, C. (2008). The psychological effects of employment after traumatic brain injury: Objective and subjective indicators. *Rehabilitation Psychology, 53*, 456–463.
- Turner-Stokes, L. (2008). Evidence for the effectiveness of multidisciplinary rehabilitation following acquired brain injury: A synthesis of two systematic approaches. *Journal of Rehabilitation Medicine, 40*(9), 691–701.
- Wade, S. L., Carey, J., & Wolfe, C. R. (2006a). An online family intervention to reduce parental distress following pediatric brain injury. *Journal of Consulting and Clinical Psychology, 74*(3), 445–454.
- Wade, S. L., Carey, J., & Wolfe, C. R. (2006b). The efficacy of an online cognitive-behavioral family intervention in improving child behavior and social competence following pediatric brain injury. *Rehabilitation Psychology, 51*, 179–189.
- Wade, S. L., Taylor, G. H., Yeates, K. O., Drotar, D., Stancin, T., Minich, N. M., & Schluchter, M. (2006). Longterm parental and family adaptation following pediatric brain injury. *Journal of Pediatric Psychology, 31*(10), 1072–1083.
- Wade, S. L., Walz, N. C., Carey, J. C., & Williams, K. M. (2009). Brief report: Description of feasibility and satisfaction findings from an innovative online family problem-solving intervention for adolescents following traumatic brain injury. *Journal of Pediatric Psychology, 34*(5), 517–522.
- Warden, D. L., Gordon, B., McAllister, T. W., Silver, J. M., Barth, J. T., Bruns, J., . . . Neurobehavioral Guidelines Working Group. (2006). Guidelines for the pharmacologic treatment of neurobehavioral sequelae of traumatic brain injury. *Journal of Neurotrauma, 23*(10), 1468–1501.
- Wasserman, L., Shaw, T., Vu, M., Ko, C., Bollegala, D., & Bhalerao, S. (2008). An overview of traumatic brain injury and suicide. *Brain Injury, 22*(11), 811–819.
- Wertheimer, J. C., Roebuck-Spencer, T. M., Constantinidou, F., Turkstra, L., Pavol, M., & Paul, D. (2008). Collaboration between neuropsychologists and speech-language pathologists in rehabilitation settings. *Journal of Head Trauma Rehabilitation, 23*(5), 273–285.
- Wilson, B. A. (2008). Neuropsychological rehabilitation. *Annual Review of Clinical Psychology, 4*, 141–162.
- Wilson, B. A., Emslie, H., Quirk, K., Evans, J., & Watson, P. (2005). A randomized control trial to evaluate a paging system for people with traumatic brain injury. *Brain Injury, 19*(11), 891–894.
- Ylvisaker, M., Turkstra, L. S., & Coelho, C. (2005). Behavioral and social interventions for individuals with traumatic brain injury: A summary of the research with clinical implications. *Seminars in Speech and Language, 26*(4), 256–267.
- Ylvisaker, M., Turkstra, L., Coehlo, C., Yorkston, K., Kennedy, M., Sohlberg, M. M., & Avery, J. (2007). Behavioural interventions for children and adults with behaviour disorders after TBI: A systematic review of the evidence. *Brain Injury, 21*(8), 769–805.
- Zhu, X. L., Poon, W. S., Chan, C. C., & Chan, S. S. (2007). Does intensive rehabilitation improve the functional outcome of patients with traumatic brain injury (TBI)? A randomized controlled trial. *Brain Injury, 21*(7), 681–690.

# Rehabilitation in Stroke

*Rene Hernandez-Cardenache and Douglas Johnson-Greene*

## EPIDEMIOLOGY AND CLINICAL CHARACTERISTICS OF STROKE

Stroke is a major cause of morbidity and mortality in the United States, particularly among the elderly. Stroke remains the third leading cause of death in the United States and the leading cause of serious, long-term disability, necessitating expenditure of considerable resources for rehabilitation of its victims ([Centers for Disease Control and Prevention, 2001](#)). Stroke is largely a disease of developed nations, which speaks to putative role and importance of life style factors. Although the incidence of stroke has declined over the past several decades, the total number of stroke occurrence has actually increased because of the growing number of elderly baby boomers.

Persons with stroke represent the largest category of referrals to rehabilitation hospitals, which is why it is important that health care professionals be familiar with the treatment and rehabilitation of this population. Despite its widespread prevalence, potential for rendering large segments of our population disabled, and its enormous burden on rehabilitation and financial resources, stroke remains one of the least researched and least funded medical disorders. In this chapter, we will review the epidemiological and clinical characteristics of stroke, including associated risk factors and types of strokes.

## INCIDENCE AND PREVALENCE OF STROKE

Estimates of stroke incidence have been very difficult to determine because there

are no universal stroke registries in existence in the United States. However, two relatively small stroke registries have been established that can provide an estimate of the incidence of stroke. The Manhattan Stroke Registry ([Sacco et al., 2004](#)) samples persons living in Manhattan and the surrounding areas and was originally intended to examine ethnic and racial differences in persons with stroke. The Paul Coverdell Stroke Registry has been funded by the Centers for Disease Control since 2004 to establish state wide stroke registries in the state health departments of Georgia, Illinois, Massachusetts, and North Carolina ([Wattigney et al., 2003](#)) with a primary purpose of developing and implementing systems for collecting data on acute stroke characteristics and care.

Based on these registries and other available data, it is estimated that there are approximately 795,000 strokes in the United States annually ([American Heart Association, 2010](#)), including 610,000 first-ever strokes and 185,000 recurrent strokes. In terms of prevalence, there are about 6,400,000 stroke survivors alive today in the United States burdened with significant physical, functional, cognitive, psychological, and social impairments ([American Heart Association, 2010](#)).

There is clearly a relationship between age and stroke with incidence climbing in advanced years. It is estimated the incidence of stroke among Americans aged 20 and older is 11/1,000 persons ([Rosamond et al., 2007](#)). In contrast, the prevalence of stroke among Americans aged 65 and older is 40/1,000 persons, and 1 in 10 Americans over 75 has experienced a stroke ([Rosamond et al., 2007](#)). In terms of gender differences, men have a higher incidence of stroke compared to women ([American Heart Association, 2010](#); [Broderick et al., 1998](#)). There are also differences in stroke incidence by ethnicity. Specifically, African Americans have approximately twice the risk of stroke as White Americans ([Broderick et al., 1998](#)), with Black males having a higher incidence than White males. Gender differences across ethnic groups appear to be maintained with Black females experiencing more stroke than White females.

## Stroke Mortality

Advances in critical care and emergency medicine over the past 40 years have greatly increased survivability of patients with stroke, which has resulted in a decline of the death rates in the United States for several decades. In fact, from 1996 to 2006 the annual stroke death rate decreased by 33.5% ([American Heart Association, 2010](#)). This decreasing trend in stroke-related mortality has been

seen in all race and gender groups. Although stroke mortality rates have declined, the number of total stroke deaths has actually increased. This is because the rate of decline slowed and the number of elderly in the United States increased dramatically. Thus, stroke remains the third most common cause of death in the United States after heart disease and cancer and is responsible for approximately 150,000 deaths annually ([American Heart Association, 2010](#)). Strokes can occur at any age but are much more common in the elderly, with the death rate doubling every 10 years between 55 and 85. Thus, approximately three quarters of stroke deaths occur in individuals over 65 ([American Heart Association, 2010](#)). Stroke mortality varies considerably by race and gender. In general, males have higher incidence of stroke-related mortality than females. Blacks have a very high stroke mortality rate compared to other ethnic groups. For example, the age-adjusted mortality rate for Black males in 2006 was 67.1/100,000 compared to 41.7/100,000 for Whites. There are also geographic differences in stroke mortality with a “stroke belt” of high mortality being identified many years ago, which encompassed many of the southeastern states. Stroke-related mortality is also greatly affected by stroke type, with approximately 8% of ischemic stroke and 38% of hemorrhagic stroke patients dying within 30 days.

### **Economic Impact**

The direct and indirect annual expenditures for stroke are staggering and vary by the type of stroke one sustains ([Taylor et al., 1996](#)). In 2010, the total economic burden for stroke was estimated to be approximately 73.7 billion ([American Heart Association, 2010](#)). The average inpatient rehabilitation stay costs \$7,449, but the mean lifetime cost following ischemic stroke is \$140,048 ([Taylor et al., 1996](#)). Not surprisingly, severe strokes cost about twice as much as mild strokes.

In spite of the enormous costs attributable to stroke and its consequences, the large numbers of patients who are discharged with deficits subsequent to stroke, and the relatively low fraction of patients who receive rehabilitation services, only a minuscule level of funding is provided in this country for research and training in stroke rehabilitation. Such findings suggest an increased need to identify early risk factors of stroke in an effort to provide better predict, control, and modify epidemiological trends in stroke.

### **RISK FACTORS FOR STROKE**

Strokes or cerebral vascular accidents (CVAs) commonly produce permanent

physical and neuropsychological deficits. Given the importance of preventing and even reducing the prevalence of strokes, a striking body of literature has emerged in the last 20 years investigating the many risk factors of stroke. Age, heredity, race, ethnicity, and gender have been clearly identified as important risk factors in the understanding, evaluation and treatment of stroke.

As with many other vascular-related conditions, age is the most powerful risk factor contributing to stroke (Sacco et al., 1997). After the age of 55, the rate of stroke more than doubles in both men and women for each successive decade. In fact, after the age of 65 years, the incidence of stroke rises almost exponentially (Kaufman, 2007). The incidence of stroke in families has also been noteworthy and has been associated with elevated risks in the development of cerebral vascular disease. Probable explanations for such findings appear to be related to a genetic tendency for stroke or its many contributing risk factors along with the possibility of a common familial exposure to environmental risks (Kiely, Wolf, Cupples, Beiser, & Myers, 1993). Interestingly, the incidence and mortality rate among victims of stroke has varied widely across racial groups. Recent studies have documented that Blacks appear to be overrepresented, with an approximate 1.5-to 2.0-fold risk in mortality rate (Howard et al., 1994). However, proportionally more strokes occur at older ages in Whites than in either U.S. Blacks or Hispanic Whites. Similarly, gender differences in stroke incidence appear to place women at increased susceptibility to suffer from stroke across all ages, as studies have frequently shown a higher lifetime risk (Petrea et al., 2009). Hence, it is not surprising that afflicted women tend to be more disabled than men before and during the acute stages of recovery from stroke and therefore, are more likely (i.e., 3.5 times) to require institutionalization (Petrea et al., 2009). One factor many times neglected in recent studies involves the relative contributions of “lifestyle” on the development of stroke. That is, cultural and/or a person’s unique behavioral patterns involving diet, weight, exercise, utilization of health care, and management of stress may partially account for the vulnerability individuals have to the onset of stroke. Identifying these factors has alerted neuropsychological practitioners to these high-risk groups, thereby enabling greater research and clinical attention to even more important, potentially modifiable risk markers.

## **MODIFIABLE RISK FACTORS OF STROKE**

It is safe to say that a fairly large fraction of patients will be at increased risk for vascular disease. When a clinical history is being taken, it is important for



clinicians to ask patients whether they have the following common vascular risk factors: hypertension, cardiac conditions, diabetes mellitus, hypercholesterolemia, cigarette smoking, or history of transient ischemic attack (TIA).

Hypertension is the single most important frequently occurring modifiable risk factor for most strokes (Sacco et al., 1997). Elevated levels of blood pressure, both systolic and diastolic, are associated with both thrombotic and hemorrhagic strokes. Moreover, hypertension has been strongly implicated in most cases of stroke-induced dementia. However, the efficacy of antihypertensive treatment for controlling these risks has been well established (Rashid, Leonardi-Bee, & Bath, 2003). In fact, the widespread treatment of hypertension has led to a steady decline of stroke-related deaths in the last century. An epidemiological summary examining 17 trials of hypertensive treatments throughout the world found a 38% reduction in all stroke events and approximately 40% decline in fatal stroke, clearly supporting systematic treatment of hypertension (MacMahon & Rodgers, 1994). Effective management of hypertension can also be of benefit in reducing stroke risks associated with various cardiac conditions. In particular, atrial fibrillation, valvular disease, prosthetic valves, and acute myocardial infarcts all comprise formidable factors suggesting health susceptibilities to this population of patients. Of the group, atrial fibrillation is the most powerful and manageable cardiac precursor to stroke (Sacco et al., 1997). This is followed by cardiac valve abnormalities such as mitral stenosis, calcification, and valvular strands. Because these conditions have the capacity to alter the endocardial surfaces of the heart, they tend to produce thromboses that can travel and embolize in other areas of the body, including the brain. Albeit with its own associated risks, the use of anticoagulant medications have demonstrated promising results in at risk patients of embolic stroke.

Diabetes mellitus along with elevated levels of total cholesterol are both powerful risk factors for myocardial infarction, but less so for stroke (Kaufman, 2007). In the classic Framingham study, persons with glucose intolerance had twice the risk of brain infarction when compared to those who were nondiabetic (Sacco et al., 1997). In particular, individuals with diabetes have an increased susceptibility to atherosclerotic disease and prevalence of atherogenic risk factors, most notably in the form of hypertension, obesity, and abnormal lipids. For younger persons under the age of 45, elevated cholesterol has little or no association with stroke; however, for all individuals, only cholesterol levels

greater than 300 mg/ml are a risk factor for stroke (Kaufman, 2007). The incidence of stroke and TIA has been reduced in individuals with hypercholesterolemia by the widespread use of statins, along with dietary restrictions and important life style changes.

Maladaptive patterns in life style such as cigarette smoking convey a notable risk for the onset of stroke. In a meta-analysis study involving 32 separate studies examining the relationship between smoking and stroke, Shinton and Bever demonstrated a notable dose response between the number of cigarettes smoked and the relative risk of stroke (Shinton & Beevers, 1989). Stroke risk decreases significantly by 2 years and tends to be at the level of nonsmokers by 5 years after cessation of cigarette smoking (Wolf, D'Agostino, Kannel, Bonita, & Belanger, 1988). In spite of this, compared to nonsmokers, former cigarette smokers retain an almost two fold increased risk of stroke, and active smokers have a four fold greater risk (Kaufman, 2007). Such reductions have been observed to occur across the age and life span, applying to both moderate and heavy tobacco users.

In addition to cigarette smoking, various other maladaptive life style factors have been associated with increased stroke risk. Most recently, research interests have focused on the effects of controlling diet, weight, physical inactivity, and acute stress. For instance, there is some evidence that consumption of dairy products may be associated with reduced risk of stroke (Massey, 2001); however, these findings have not been replicated. In spite of this, when combined with a balanced diet, weight management, and physical activity, reductions of stroke may occur (Abbott et al., 1996). Interestingly, the pattern of obesity may be important, as central obesity as manifested by a high abdominal deposition of fat, rather than obesity involving the hips and thighs, has been more closely related to the occurrence of atherosclerotic disease (Sacco et al., 1997). Indeed, the presence of obesity has been associated with elevations in blood pressure and blood glucose, both of which have been recognized as independent risk factors for CVA (Sacco et al., 1997). In a large prospective study examining the relationship between the levels of physical activity and stroke among 7,530 men, researchers found that older men who were inactive or only partially active experience a three to four excess incidence of hemorrhagic stroke as compared to active men (Abbott, Rodriguez, Burchfiel, & Curb, 1994).

Collectively, it appears that physical activity exerts a beneficial influence on risk factors for atherosclerotic disease by reducing blood pressure, weight, raising high-density lipoprotein (HDL) cholesterol and lowering low-density

lipoprotein (LDL) cholesterol, and improving glucose tolerance. With these factors in mind, individuals could optimize their chances of avoiding stroke simply by first identifying, then controlling, and ultimately attempting to alter these modifiable risk factors. Knowing the underlying malleability of these modifiable and associated features of stroke, psychologists working in rehabilitation settings can empower their stroke patients and encourage them to take a proactive role in their health and future.

## GENERAL MECHANISMS AND TYPES OF STROKE

The term “stroke,” rather than representing a single neurological condition, is a broad rubric representing a range of cerebrovascular disorders. In its most reduced terms, stroke occurs when a cerebral blood vessel is either blocked by a clot or ruptures. When that happens, the part of the brain involved in the stroke cannot receive the vital nutrients and oxygenated blood it requires, thereby leading to neuronal death and neuropsychological dysfunction. Strokes can be classified as either ischemic (i.e., clots) or hemorrhagic (i.e., bleeds) in origin. The distinction between the two categories carries clinical importance for both evaluation and treatment purposes as the conditions are in rigid contrast to each other. Ischemic stroke occurs as a result of an “obstruction” within a blood vessel supplying blood to the brain resulting in reduced irrigation to the affected cerebral area. Although a TIA is often labeled “mini-stroke,” it is more accurately characterized as a “warning stroke” that is to be taken very seriously. TIA is often caused by a clot, the difference is that in TIA the blockage is more commonly transient, occurs rapidly, and lasts a relatively short period (i.e., mostly last between 30–60 minutes). Unlike a stroke, when a TIA is completed there is generally no permanent injury to the brain. Hemorrhagic strokes, however, result when a weakened blood vessel ruptures and bleeds into the surrounding cerebral tissue. Given the nature of these vascular changes in the brain, early and accurate identification of the underlying mechanisms of stroke are important to initiate the most suitable medical treatment. Most important, prompt medical attention allows therapeutic interventions that improve functional outcome.

### Ischemic Stroke

The most frequent causes of strokes consist of abnormalities stemming from the heart, blood vessels, and blood. The overwhelming majority of strokes are caused by *thrombosis*, a clot that propagates within an extracranial or

intracerebral artery and occludes blood flow to a regional area. The obstruction of blood flow can be due to buildup of *atherosclerotic plaques*, which are lipid deposits within the artery walls. Such obstruction of blood flow to the brain typically results from the accumulation of layers of coagulated blood and plaques that narrow the vessel openings, thereby restricting the passage of blood flow. Stroke resulting from cerebral atherosclerosis occurs when the narrowing of the vessels results in the thickening and hardening of the cerebral arteries. When this condition interrupts the blood supply to the area, it deprives the brain of essential amounts of oxygen and nutrients, leading to *ischemic stroke*. Ischemic stroke is generally an evolving process that often transpires over time, most commonly over hours to days. The nature of the illness tends to have more of a stuttering course over time; however, the distinction between it and other forms of cerebral vascular events is not often easy to make upon clinical examination alone.

Another well-known cause of ischemic stroke is an *embolus* from the heart, extracranial artery, or intracranial artery that wedges itself into a cerebral artery. The constituents of the embolus are usually comprised of a buildup of thrombotic material that has *traveled* away from a blood vessel wall and has lodged into a smaller one, thereby restricting required circulation and causing the sudden onset of symptoms. The formation of emboli can be due to the presence of a blood clot, lipid buildup, air bubbles, or even the commuting of a small mass of cells blocking cerebral arterial circulation. In embolic strokes, the clinical objective is to determine the source of the embolus so that future strokes may be prevented. In cardioembolic infarcts, the embolus originates in the heart and may develop as a result of atrial fibrillation. In this condition, the clot tends to form in the fibrillating left-atrial appendage and travels away from the heart into a narrow vessel. Common cardiac conditions such as myocardial infarction and valvular disease similarly harvest the growth of thrombi, which carry the potential of effecting brain functioning through similar mechanisms. Other causes of cerebral thrombosis and embolism can include cerebral vasculitis, causing the inflammation or spasmodic constriction of the vessels, drug abuse, and other blood-related disorders.

### **Hemorrhagic Stroke**

Unlike ischemic strokes, hemorrhagic strokes typically occur abruptly and because of elevations in intracranial pressure commonly produce severe headaches, nausea, and vomiting. Hemorrhagic strokes result from massive

bleeding into the brain tissue (i.e., so-called parenchymal tissue) that tends to be caused by hypertension, toxins, blood-related conditions, or congenital cerebral artery abnormalities. Many victims of hemorrhagic stroke suffer a loss of consciousness and may suffer severe neurological deficits that can be determined by the origin of the hemorrhage. A poor prognosis and negative outcome are often seen when the duration of loss of consciousness is longer than 2 days. Hemorrhagic strokes are oftentimes classified according to the anatomic locus of the bleeding (i.e., extradural, subdural, subarachnoid, inter/intra cerebral, or cerebellar).

Following the thrombotic and embolic stroke, *primary intracerebral hemorrhagic infarcts* are the third most common cause of stroke (Ropper & Brown, 2005). Such infarcts are most often the result of hypertension and tend to involve a rupture of a penetrating cerebral artery. Such deep and penetrating arteries typically irrigate regional structures of the brain such as the basal ganglia, thalamus, pons, and cerebellum. Although this type of stroke frequently involves these important subcortical structures, the blood rarely reaches the cortical surface. In spite of this, some studies have demonstrated that blood elements from such infarcts can be seen in cerebrospinal fluid in more than 90% of all cases (Ropper & Brown, 2005). Intracerebral hemorrhages originating from the spontaneous ruptures of cerebral aneurysms also fall under the hemorrhagic stroke classification. Aneurysms are balloon-like expansions from blood vessels often caused by congenital anatomical abnormalities, hypertension, arteriosclerosis, embolisms, or infections. The most common locations of aneurysms are the origin of the anterior communicating artery, posterior communicating artery, or the bifurcating points of the middle cerebral artery. In approximately 80% of cases, spontaneous hemorrhage is caused by rupture of an arterial aneurysm in the subarachnoid space, making localization of the regional areas involved fairly predictable (Blumenfeld, 2002).

The fourth most common cause of stroke is *subarachnoid hemorrhage* (SAH) involving bleeding under the arachnoid layer, causing damage to underlying brain tissue by exerting intracranial pressure and irritation. The course of SAH can be either acute or have a stuttered quality depending on the size of the affected blood vessel and of the rupture itself. However, from a clinical viewpoint, it is important to recognize its classic presentation. Such hemorrhages are notorious for producing relentless and severe headaches along with notable nuchal rigidity. Strenuous, albeit benign physical activities in the form of straining during stool release, overexertion in sexual activities, and

lifting can rupture an existing aneurysm and even precipitate the onset of a SAH. To a greater degree than MRI, CT of the brain may reliably reveal the presence of blood at the base of the brain or within the ventricular system. Moreover, a lumbar puncture will often show blood products and yield a xanthochromic cerebrospinal fluid (Blumenfeld, 2002). As previously mentioned, significant elevations in intracranial pressure may result in life-threatening medical emergencies requiring neurosurgical intervention as a result of herniation.

## **COGNITIVE AND EMOTIONAL ASSESSMENT OF STROKE**

Cognitive disorders during the acute stages of stroke are common and are important independent predictors of adverse outcome in victims of stroke (Billoski, 1992; Nys, Van Zandvoort et al., 2005b; West, Hill, Hewison, Knapp, & House, 2010). The importance of clinical assessment and treatment after stroke has been fairly well established (Snyder & Nussbaum, 1998; West et al., 2010). Neuropsychologists and rehabilitation psychologists delineate cognitive and emotional functioning, provide psychological treatment recommendations, and assist in treating acute neurobehavioral sequelae stemming from stroke. By repeated monitoring of the often emerging symptoms in psychological functioning, they are able to provide suitable recommendations to the rehabilitation efforts, and in many ways guide community reentry. Working closely with the interdisciplinary team, psychologists inform team members about relative strengths and weaknesses of the stroke survivor, educate family and relatives, and offer suitable recommendations for discharge planning.

### **Acute and Postacute Assessment**

During the acute phase of stroke, cognitive impairment is related not only to direct local effects of the stroke, but also hypoperfusion (Hillis, 2004) and *diaschisis* (i.e., functional deactivation) in nearby or remote areas of the brain (Ferro, 2001). As is commonly the case following acute stroke, the initial exposure to the patient may be at bedside. Most often, this will involve assessing the stroke survivor in a neurology intensive care unit (NICU) where the patient's mobility, cognition, and many times their capacity to communicate may be compromised. Physical symptoms that may directly affect the initial consultation include numbness, hemiparesis, decreased vision, dysarthria, and loss of balance or coordination (Snyder & Nussbaum, 1998). During these initial stages, one of the roles of the clinician is to obtain preliminary information regarding premorbid functioning (e.g., gainful employment, educational level, age,

activities of daily living [ADL]) along with recording a baseline of current cognitive and emotional assessment data. Periodic visits and consultation with the stroke survivor can provide the medical staff with both qualitative and quantifiable information reflecting cognitive changes across time and contribute to decisions regarding transfers to less acute levels of care. Meeting with immediate family at this stage can assist caregivers in having a better understanding of the nature of the evolving neurological condition, enable them to provide appropriate levels of stimulation and care, coordinate visits, and discuss the prospects of having to make important health care decisions, if the patient is incapacitated.

The responsible administration and valid interpretation of standardized neuropsychological tests is often quite difficult in this population. Because of the direct effects of stroke, it is often difficult for patients to be fully aware and responsive (e.g., fluctuating consciousness, aphasia, hemiparesis, visual-field cuts, medications effects) during the acute stages of inpatient hospitalization. However, clinicians are urged to attempt to assess the individual's neurobehavioral state, even if the determination is that the patient is not "formally testable" at the time. Attempting to intervene and "size up" the individual during the early stages of stroke permits clinicians the opportunity to see them during the most "mentally compromised moments" and can help establish "a point of reference" on which much of the psychological work that follows will rest. Currently, a range of brief cognitive measures has been used to assess cognitive impairments in patients with acute stroke. Even so, most of the measures are designed to offer preliminary information and not recommended to be used to arrive at absolute diagnostic conclusions. Instead, the purpose of using them during the acute stage is to assist clinicians at arriving at *tentative diagnostic impressions* and to drive the development of underlying hypotheses. Information obtained from initial screenings can also provide the clinical basis for more comprehensive assessment and treatment during the postacute recovery periods.

Although there is a need for short, practical instruments measuring global cognition in stroke, there remains little consensus about which tests are most suitable for this population (Pendlebury, Cuthbertson, Welch, Mehta, & Rothwell, 2010). Originally designed to render a practical "clinical assessment of change" in the cognitive status of geriatric inpatients, the *Mini-Mental Status Examination (MMSE)* has become the most widely used screening measure of gross cognitive functioning (Folstein, Folstein, & McHugh, 1975). In spite of its

popularity, it has been demonstrated to lack sensitivity to *mild cognitive impairment* and may be inadequate at detecting cognitive abnormalities associated with cerebral vascular disease (Tombaugh & McIntyre, 1992). The instrument is heavily weighted by language-based tasks, and tends to underestimate cognition in the well-educated patients. In addition, the *MMSE* was not intended to measure executive and/or higher-order functions, and is in effect, devoid of assessing this vital area. Hence, its applicability is limited with aphasic patients and those suspected to suffer from executive dysfunction. Given that impairments in executive functioning have been observed in more than 50% of victims of stroke in some populations, proper assessment of this important area remains essential (Barker-Collo & Feigin, 2006; Zinn, Bosworth, Hoenig, & Swartzwelder, 2007). In spite of the notable limitations of the *MMSE* for victims of stroke, the recently developed Montreal Cognitive Assessment (MoCA) was designed to be sensitive to mild cognitive impairment and, unlike the *MMSE*, includes brief attention and executive functioning tasks (Nasreddine et al., 2005). Such an instrument is a welcomed addition to the field, as it has been demonstrated that it is more sensitive to cognitive abnormalities in patients with cerebrovascular disease (Pendlebury et al., 2010). Moreover, clinicians can more reliably identify early symptoms of executive dysfunction, inattention, and memory impairment after TIA and stroke.

A slightly more extensive and recently developed instrument is the repeatable battery of neuropsychological status (RBANS). This brief and practical screening battery comprises five standardized index scores, similar in content to popular neuropsychological measures. The instrument has been demonstrated to have adequate discriminant and convergent validity in language, visuospatial/constructional, immediate and delayed memory indices (Larson, Kirschner, Bode, Heinemann, & Goodman, 2005). Wilde in 2006 found that left-hemisphere stroke patients performed more poorly in language/verbal memory indices than did right-hemisphere stroke patients, whereas right-hemisphere stroke victims showed reductions on the visuospatial/visual memory domains (Wilde, 2006). Moreover, in a sample of inpatient victims of stroke, the RBANS indices predicted cognitive disability 6 months later as recorded in follow-up telephone interviews (Larson et al., 2003). During a follow-up study 12 months later, the individual indices of the RBANS were found to predict ADL and the total score predicted cognitive disabilities as measured by the functional independence measure (FIM) (Larson et al., 2005). Although assessment later in the course of recovery from stroke will have a greater predictive validity,



neuropsychological data obtained during the acute inpatient periods are related to postdischarge adjustment and may represent valuable prognostic information, which can be used for appropriate discharge planning. Given the host of deficits that often emerge during the acute stages of stroke (i.e., hemiparesis, visual field cuts, aphasia, anosognosia, depression), practitioners are often taxed to the point that customized neuropsychological assessment is more often the rule, rather than the exception. Such flexible assessment techniques enable psychologists to “test the limits” of a cognitive function, provide important *qualitative* information to the interdisciplinary team, and contribute to helpful rehabilitation strategies that might similarly aid long-term outcome. It is of critical importance to translate the identified quantitative and qualitative neuropsychological deficits into “what it means” in the individual’s day-to-day functional activities. Therefore, every effort is to be made to present data in a manner that is *functionally meaningful* so that impressions and recommendations can be used by supportive staff and family during the road toward recovery.

The concept of “*vascular cognitive impairment*” is used in a variety of neurological disorders sharing the fact that the cerebral vascular system is compromised, resulting in cognitive reductions. It has been proposed as an umbrella term to recognize the spectrum of cognitive and behavioral changes associated with neurovascular pathology (O’Brien, 2006). Currently, there remains persistent controversy over agreeing on a common terminology referring to cognitive deterioration resulting from diseases of the blood supply system of brain. Researchers investigating the cognitive sequelae associated with stroke have recently adopted a more reductionist approach by defining cognitive outcome in terms of “vascular dementia” or even milder variants of this condition, such as “vascular cognitive impairment.” In fact, over the past few years, a broad range of terms that have included milder forms of cognitive impairment have been introduced, such as vascular cognitive disorder, mild cognitive impairment, and cognitive impairment-no dementia. Although these terms may be preferable and less diagnostically misleading than “vascular dementia,” they still attempt to capture diverse phenomena under a single heading, resulting in poor prognostic value, and confusion in the literature. Additionally, such concepts tell us little about the nature of the underlying cognitive disorder and the limitations in day-to-day functioning that might arise as a result of these deficits. de Haan, Nys, and van Zandvoort (2006) proposed that the term “vascular dementia” should be reserved for those conditions marked by progressive cognitive decline, which include diabetes mellitus,

leukoencephalopathy, and autosomal dominant arteriopathy (de Haan et al., 2006). In all other cases, a more clear and concise conceptualization of the cognitive consequences of stroke are needed.

In a 2005 longitudinal study examining the prognosis of acute cognitive disorders in 111 patients following stroke, the authors found that improvement as compared to baseline seemed to be the rule, rather than the exception in all cognitive domains (Nys, Van der Zandvoort et al., 2005b). Hence, demarcating the differences in the course of illness between “progressive” and “nonprogressive” in neurovascular pathologies appears to be central to this issue. This is most notably true in light of recent evidence suggesting that cognitive recovery rates following stroke have ranged from 41% to 83% of all cases in longitudinal studies, depending on the nature of the cognitive deficit (Nys, Van Zandvoort et al., 2005b). For this reason, evaluating the most commonly occurring cognitive impairments in this population appears to be an overriding objective of sound neuropsychological and medical care.

## **NEUROPSYCHOLOGICAL EVALUATION OF COGNITIVE IMPAIRMENTS**

### **Aphasia and Stroke**

Aphasia is an acquired impairment in language that results from neurological damage to the language areas of the brain; thus, it typically involves the left hemisphere. However, defining aphasia as solely a disorder of language is really an oversimplification of a complex clinical phenomenon. One of the most common causes of aphasia involves embolic or thrombotic (i.e., ischemic) stroke of the left middle cerebral artery, supplying the perisylvian cortical language areas. Other causes of sudden onset aphasia can involve cerebral hemorrhage secondary to a rupture of an aneurysm, arteriovenous malformation, traumatic brain injury, or hypertensive bleed. The variant forms and attributes of aphasia and its accompanying deficits indicate the site and often the extent of the neurological involvement.

In general, and from a clinical vantage point, the condition is most often characterized by deficits with language production, comprehension, and word selection. The incorrect selection of word choice and sound substitutions are among the most common features of acquired language impairment and are referred to as “paraphasic errors.” Aphasia tends to affect not only expressive or spoken language but also receptive language or comprehension along with the

efficient production of language in the form of reading and writing. For this reason, impairment in reading is referred to as *alexia*, whereas writing disorders have been coined *agraphia*. However, there is considerable variability within a given aphasia type regarding reading and writing, and for this reason a description of reading and writing abilities is not always included when discussing the “classic aphasia syndromes.” Well-known aphasia syndromes, however, allow clinicians to identify functional language constellations that provide useful algorithms for the diagnostic evaluation of aphasic patients.

Classification of aphasia syndromes is commonly achieved by a series of binary decisions regarding fluency of language, auditory comprehension, and verbal repetition. The eight classic syndromes have been used for many decades to easily communicate the basic language profiles of patients. For example, *fluent aphasias* include Wernicke’s, transcortical sensory, conduction, and anomic aphasias; whereas, *nonfluent aphasias* include global, mixed transcortical, Broca’s and transcortical motor. However, not all patients will neatly fit into a given classic syndrome and will more notably be distinguished by his or her divergence from these known profiles.

Wernicke’s aphasia is a *fluent aphasia* characterized by both poor auditory comprehension and repetition. Naming attempts are often paraphasic and the presence of severe *anomia* is more often the rule, rather than the exception. Similarly, transcortical sensory aphasia is also a fluent aphasia, and although comprehension remains impaired, repetition tends to be preserved. Conversely, in conduction aphasia there is good comprehension, but poor repetition. In fact, impairment in repetition is the hallmark of conduction aphasia, despite relatively good spontaneous speech. In anomic aphasia, acute lesions are generally located outside the perisylvian language region and involve the angular gyrus or inferior temporal region; therefore, such a fluent aphasia is characterized by good auditory comprehension and repetition, but with marked limitations in naming.

Although fluent aphasias are well recognized on the basis of expressive language facility, *nonfluent aphasias* are identified by a *lack* of verbal facility or expressive ease. The most commonly recognizable nonfluent aphasia is the global aphasia in which poor auditory comprehension and limited verbal repetition is present. Conversational speech is not only nonfluent but also characterized by a slow, halting production of speech. Auditory comprehension is usually limited to single-word comprehension, and repetition is compromised severely. In mixed transcortical aphasia, repetition is generally preserved although it typically occurs without good comprehension or naming ability.

Although the prognosis is variable in this aphasia, patients in whom the cause is cerebrovascular in nature have the greatest chance of recovery of the affected language functions. Regardless of the type of aphasia, data show that nonsevere language and motor dysfunction after stroke seem to show highly predictable functional recovery during the first 3 months, and that this is related to the initial severity of impairment (Lazar et al., 2010). In fact, similar predictability suggests the presence of underlying recovery mechanisms, which may be operating that are common to patients with mild to moderate stroke, regardless of the nature of aphasic dysfunction, particularly during the postacute periods.

A number of standardized tests are currently available for the assessment of aphasia. Such measures are not designed for bedside administration and are more appropriate for clinical administration during the postacute stages of stroke. Neuropsychologists along with speech and language pathologists commonly use such instruments to delineate language functions in an effort to aid recovery and treatment. The following is a brief list of instruments that can be used for this purpose:

1. **Aphasia screening and diagnostics:** Aphasia Diagnostic Profiles (Helm-Estabrooks, 1992).
2. **Auditory comprehension:** Token Test of the Multilingual Aphasia Examination (Benton, deHansher, & Siven, 1994).
3. **Comprehensive aphasia evaluation:** Boston Diagnostic Aphasia Examination (Goodglass & Edith Kaplan, 1983).
4. **Naming:** Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983).
5. **Reading and Writing:** Psycholinguistic Assessment of Language Processing in Aphasia (PALPA; Kay, Lesser, & Coltheart, 1992).

Instruments such as the Boston Diagnostic Examination (BDAE) (Goodglass & Kaplan, 1983) evaluate language skills based on perceptual modalities (auditory, visual, and gestural); processing functions (comprehension, analysis, and problem solving); and response modalities (writing, articulation, and manipulation). Such an instrument is designed as a comprehensive measure of acquired language disorder. Similarly, the Boston Naming Test as a subtest of the BDAE, has been widely used as a sensitive measure of confrontation naming following stroke. Short versions, using either 15 or 30 words have been found to be reliable and valid screening tests with age and education effects vary across

different studies. Verbal fluency tasks have also been widely used for many decades and have shown some discriminative value in differentiating vascular cognitive impairment from Alzheimer's dementia. For example, category cueing involving fruits or animals may more heavily rely on posterior left parietal-temporal functioning, whereas phonemically cued or letter fluency generation has recently been associated with left dorsolateral frontal lobe functioning. Although such measures often provide useful information regarding the neuroanatomical and the neurophysiological areas involved with cerebrovascular disease, assessment of aphasia can present notable challenges to the clinician, particularly when the language impairment itself limits information exchange regarding the impact of this and other deficits. Although treatment aimed at reducing cognitive dysfunction following stroke is not the focus of this chapter, rehabilitation is an important component of cognitive recovery from the acute to subacute (i.e., first 3 months) time period. In fact, even minimal speech rehabilitation in individuals suffering from aphasia after stroke were associated with recovery of language up to 70% of the individual's maximum possible level ([Lazar et al., 2010](#)).

### Memory and Stroke

Memory functioning involves the ability to encode, store, save, and retrieve information when needed. Such processes are particularly important in the rehabilitation of stroke, as they are essential for the learning of new skills and the recovery of old skills. Memory is one of the cognitive domains most frequently affected by stroke ([Brown & Eyler, 2001](#)). In a recent study 83% of subjects with notable deficits in verbal memory and 78% of subjects with visual memory and visuospatial deficits had shown significant recovery by 6 months ([Nys et al., 2005b](#)). Such findings are consistent with a body of literature supporting the fact that many cognitive deficits resulting from stroke improve in the first few weeks to months after stroke ([Gottesman & Hillis, 2010](#)). Some of this improvement can be attributed to natural spontaneous recovery; however, other recovery occurs secondary to cerebral plasticity as a result of adjacent or contralesional brain regions taking over cognitive tasks that were previously performed by ischemic regions ([Marshall et al., 2000](#)). An important concept surrounding this idea rests on the notion that cognitive functions cannot really be understood in terms of being the restricted responsibility of a specialized group of neurons located in a particular area of the brain. Instead, such functions may be seen as the end product of a complex functional system uniting several

different anatomical areas. Hence, damage to one area (i.e., subcortical), can lead to substantial neuropsychological impairments warranting clinical attention during stroke rehabilitation (Hochstenbach et al., 1998). From a cognitive perspective, it is important to note that damage to any perceptual or cognitive process can disrupt learning and memory dependent on that process. Clinicians are to be mindful that learning and recall require intact levels of alertness and attention. That is, impaired sensorium, even when mild and transient, in the form of delirium or confusional states or drowsiness, will weaken attentional resources important for encoding and memory. Active learning and recall, particularly of complex stimuli, require adequate effort and motivation, planning, strategies, monitoring, and working memory. Such “executive functions” are often impacted by strokes affecting the frontal lobes, which in turn can clinically present as specific types of learning and memory impairments. Such failures are dependent on the exact region involved in the frontal injury (Lim & Alexander, 2009).

Anatomically and regardless of the etiology, including stroke, damage to left limbic structures produces impaired verbal memory (i.e., list learning, story recall, etc.) and some degree of visual memory (i.e., figures, geometric designs, etc.) (Frisk & Milner, 1990). Damage secondary to stroke of the right limbic structures, however, produces impaired configurational visual memory and may impair spatial memory (Doyon & Milner, 1991). Perhaps, as humans are inherently socially driven and verbally equipped organisms, left-sided lesions to these limbic areas seem to produce more “functionally” important memory deficits, particularly as they relate to cognitive ADL. However, bilateral lesions to limbic structures are required for permanent, global episodic memory impairment (Lim & Alexander, 2009).

Research investigating the long-term deficits in episodic memory after stroke indicate that poor verbal memory (i.e., immediate, delayed, and recognition) could be predicted by lesion characteristics 1 year after stroke (Schouten et al., 2009). Not surprisingly, the researchers have found that patients with left-hemispheric lesions showed a poorer performance on the verbal memory subtests (i.e., *Rey Auditory Verbal Learning Test*) when compared to patients with right hemisphere lesions. Interestingly, when examining subcortical to cortical lesions following ischemic stroke, investigators found that patients with subcortical lesions performed less well on verbal memory subtests than those with cortical lesions. Indeed, there has been notable evidence that subcortical lesions, particularly of the basal ganglia following stroke, may lead

to substantial neuropsychological disorders, which deserve attention during the course of neurorehabilitation ([Hochstenbach et al., 1998](#)). Lastly, as expected, larger lesions produced more significant reductions in verbal memory performances when compared with patients observed to have smaller lesions following ischemic stroke. One frequently cited hypothesis for this postulates that large, acute lesions result in greater and more prolonged “neural shock” than multiple small lesions distributed over time as would be observed in progressive neurodegenerative disorders (i.e., vascular dementia). Such findings support the importance of studies examining lesion characteristics and the valuable role such knowledge can have in evaluating memory following stroke.

In a cross-sectional study examining verbal learning and memory functioning following pediatric stroke, subjects scored significantly lower than controls on the California Verbal Learning Test—Children, as well as on measures of intellectual functioning and auditory attention/working memory ([Lansing et al., 2004](#)). Furthermore, no significant lateralization differences were observed between right-and left-hemisphere lesion patients on indices of verbal learning and memory, intellectual ability, or attention/working memory functions. Such findings stand in stark contrast to the adult stroke literature suggesting lateralization differences discussed earlier in the chapter ([Schouten et al., 2009](#)). Stroke subjects were not as efficient as control subjects at creating context or semantic clusters to facilitate recall. Such a finding is further supported by increased vulnerability to *retroactive interference* observed among pediatric stroke victims. The study also concluded that children who suffered from lesions secondary to stroke occurring prenatally or up to 12 months (i.e., early), showed poor performance in verbal learning and memory when compared to youth with later stroke onset ([Lansing et al., 2004](#)). Consistent with other findings, a study suggests that focal brain damage in childhood results in relatively mild impairments in general intellectual functioning, auditory attention/working memory, and verbal learning and memory after pediatric cerebrovascular accidents ([Bates, 1994](#)).

The results of these findings are clinically important in the setting of neurorehabilitation, where therapists may try to teach new functional skills to victims of stroke. Depending on the nature of the impairment, memory disorders are variably amenable to a plethora of rehabilitation plans. Such strategies depend on the nature of the memory deficit (encoding versus retrieval); however, assessment of the deficits is essential to delineate the affected functions to promote community reentry. Below is a list of instruments commonly used to

assess memory deficits secondary to stroke:

1. *Revised Visual Retention Test* (Benton, 1992)
2. Buschke Selective Reminding Test (Buschke & Fuld, 1974)
3. California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 2000)
4. Hopkins Verbal Learning Test (Brandt, 1991)
5. Rey Auditory Verbal Learning Test (Rey, 1964)
6. Rey-Osterreith Complex Figure Test (Osterreith, 1944)
7. *Wechsler Memory Scale, Fourth Edition* (Wechsler, 2009)

## **ANOSOGNOSIA AND UNAWARENESS SYNDROMES AFTER STROKE**

In 1898, G. Anton described patients who were blind as a result of bilateral occipital lobe lesions, though they insisted that their vision was intact. Such a phenomenon has been referred to as Anton's syndrome or *cortical blindness* and has been reported by many investigators. The underlying cause may be damage to the posterior cerebral artery occlusions or traumatic occipital lobe injury (Kaufman, 2007). Alternatively, extensive brain injury stemming from anoxia, multiple cerebral infarcts, or multiple sclerotic lesions may cause cortical blindness along with other related impairments. In part as a result of Anton's observations, there has been growing interest into unawareness syndromes. The term "anosognosia" was subsequently coined by Babinski in 1914 to describe patients suffering from left hemiplegia following a right cerebral hemisphere stroke, who denied any difficulties with their left, upper, or lower extremities (Babinski, 1914). Although the term was initially used to characterize anosognosia for hemiplegia (AHP), it has more recently been used to describe a lack of subjective experience for a wide range of neurological and neuropsychological disturbances. Anosognosia today is more broadly defined as the lack of awareness or underestimation of a specific deficit in sensory, perceptual, motor, affective, or cognitive function owing to a brain lesion (Orfei et al., 2007).

The frequency of anosognosia is strongly influenced by the time of the assessment after the stroke. It is rarely observed beyond the 3-month, subacute stage following stroke (Cutting, 1978). In fact, anosognosia can be accurately described as a transient clinical phenomenon, rarely achieving a chronic nature (Starkstein, 2010). In a recent meta-analysis examining the prevalence of the condition, Pia et al. (2004) found the frequency of anosognosia to range from



20% to 44%, depending on the time since the stroke occurred. More important, in a study investigating AHP during a postacute rehabilitation phase, the researchers found that AHP presents a significant risk for negative functional outcome in stroke rehabilitation (Hartman-Maeir, Soroker et al., 2001). Although various instruments have been designed for research purposes to better assess its presence and severity, the condition is relatively simple to diagnose within the clinical arena. Moreover, it represents an aspect of hemiattention of notable importance to neurorehabilitation professionals.

Although a number of questionnaires and diagnostic methods have been developed to assess the presence and severity of anosognosia following stroke, they are often limited by inadequate discriminative ability or a narrow scope on specific deficits. In the clinical arena, although clinicians are often quick to identify a lack of awareness of *motor impairment* when asking the patients to use their paralyzed or weak limb, these observations only allow clinicians to detect the presence of anosognosia for the apparent motor deficits. In part, such a limitation in clinical assessment of this condition has led to the development of high-fidelity assessment instruments. Cutting, in 1978, developed the Cutting's Questionnaire that includes items that identify the patients' general awareness of the sensory-motor deficits, along with recording the presence of variants of unawareness syndromes such as *anosodiaphoria* (i.e., indifference to the presence of a deficit) or *misoplegia* (i.e., the morbid dislike or hatred of paralyzed limbs in patients with hemiplegia). Cutting's Questionnaire may be useful during the early diagnostic stages poststroke; however, its dichotomous classification (aware/unaware) limits its use in understanding the *injury severity* of the unawareness (Cutting, 1978).

Modern developments in the assessment of anosognosia include instruments that allow clinicians to evaluate both the presence and degree of impairment (i.e., mild, moderate, or severe) of anosognosia for sensory-motor deficits (Bisiach, Vallar et al., 1986). Even more recently, Starkstein and colleagues introduced the Anosognosia Questionnaire, a standardized measure, to gauge the presence of the denial of illness (Starkstein, Federoff et al., 1993). On this instrument, patients are asked to answer a series of items designed to elicit and quantify the *severity* of deficit unawareness along with performing requested tasks aided by the examiner (i.e., lift both arms). Identifying such a lack of "deficit awareness" carries important ethical and medical-legal implications germane to the appropriate care of patients. As treating physicians and psychologists, it is common practice to have patients identify treatment

goals and to assist in developing a treatment program around those goals. However, if the afflicted patient's perceptions of himself are unrealistic, then his goals will almost invariably be unrealistic. Therefore, it is the responsibility of treating clinicians to be mindful and assume an active role in structuring the care of these patients when they are unable to do this independently. Without such guidance, patients may not receive the necessary treatments to avoid rehabilitative complications that will impede community reentry and an optimal, successful outcome.

## VISUAL AND SPATIAL NEGLECT

Unilateral spatial neglect (USN) is an acquired disorder that affects an individual's ability to acknowledge or respond to stimulation presented to the side opposite a brain lesion. Such a disorder is a disabling feature of stroke with an estimated prevalence of 30% (Rossi et al., 1990). Many terms like unilateral neglect, hemi-inattention, hemispacial neglect, and visual neglect are frequently used interchangeably to describe USN. USN is most commonly observed in patients with focal disease most often resulting from neurovascular insufficiency (i.e., embolic or hemorrhagic stroke) but can also be seen with tumors as an ictal manifestation in individuals with seizures and in patients with colossal lesions (Snyder & Nussbaum, 1998). Clinically, and even from a practical perspective, the features of USN become apparent as patients show difficulties negotiating their surroundings with wheelchairs without bumping into objects, failing to eat or neglect food from one side of the plate, or attend to one side of the body (i.e., shaving). USN may affect the individual's capacity to attend to one side of the body (personal neglect), the space within reaching distance (near extrapersonal neglect), the space beyond reaching distance (far-extrapersonal neglect) or combination of all of these variants (Beschlin et al., 1997). The rate of recovery from neglect is greatest in the first month following stroke (Cassidy et al., 1998). Although the acute signs of USN may resolve suddenly, many individuals may continue to show impaired signs of chronic neglect, extending to months or even years after the onset. Such findings can have a significant impact on the individual's ability to carry out essential ADL and increase susceptibility to psychiatric dysfunction. Also, patients with USN have been found to have longer lengths of stay in neurorehabilitation centers and exhibit lower scores on the FIM, and thus require more direct assistance at discharge (Denes et al., 1982; Katz et al., 1999). Because of these limitations, the presence of neglect following stroke has been strongly associated with an increased risk for injury and with

poor functional outcome ([Jehkonen et al., 2000](#)).

## **Neuropathological Mechanisms of Neglect**

Neglect can occur following cerebral vascular lesions of either the left or right hemisphere; however, it more frequently occurs following right-than left-hemisphere lesions. Also, the severity of the USN is much greater and tends to lateralize toward the left hemisphere of the brain. In part due to its statistical rarity, when persistent right-sided neglect is present, it should raise the suspicion of lesions afflicting both hemispheres (i.e., bilateral lesions). Having said this, USN can occur as a result of lesions at different anatomical regions and varies in its presentation ([Mesulam et al., 1985](#)). Researchers have proposed many different theoretical explanations to explain neglect, yet there is no one unifying theory that can fully account for the unique presentations of the neglect.

A compelling theory posits that USN is due to hemispheric attentional differences of the brain. That is, in an individual with no known neurological disease or impairments, the right hemisphere of the brain attends to both the right and left hemispaces, whereas the left attends only to the right hemisphere ([Mesulam et al., 1985](#); [D'Esposito et al., 1997](#)). Following a right hemisphere cerebrovascular lesion, attention is directed to the right hemisphere resulting in a neglect of the left hemisphere. However, a lesion of the left hemisphere does not usually result in USN because the intact right hemisphere will continue to direct needed attentional resources to both hemispaces. Neglect can be caused by a variety of cortical and subcortical lesions, a finding in keeping with the well-established notion that there are multiple neural circuits mediating the distribution of attention in space. The most frequently observed region of damage is the inferior right parietal lobe, but lesions of frontal cortex, cingulate gyrus, basal ganglia, thalamus, and reticular formation may also cause neglect ([Heilman et al., 1993](#)). Hence, rehabilitation from USN requires a working diagnosis and the ability to link the disorder to the underlying processing deficits wherein attention is likely disrupted ([Snyder & Nussbaum, 1998](#)).

## **Assessment of Disorders of Neglect**

Early assessment of USN can lead to several important clinical objectives such as initiating treatment interventions involving cognitive rehabilitation during hospitalization, the need for outpatient perceptual neuropsychological assessment, employing measures to assure safety, and responsible discharge planning. In spite of this, there is some evidence that standardized assessment of

USN during the acute care phase poststroke occurs at a much lower rate than previously believed (Menon-Nair et al., 2006). Direct observation of behavior when a patient is interacting with the examiner, using a manual or electric wheelchair, eating, writing, or handling common objects in the environment may reveal the presence of USN. Basic bedside clinical screening may require clinicians to more closely delineate the nature of the contralateral orienting deficits. In more subtle cases of USN, formal neuropsychological testing may be warranted to detect the specific deficits. In accordance with the nature of USN, left neglect is generally more severe as measured by cognitive testing (Denes et al., 1982) than right neglect, and larger lesions increase the severity of neglect (Hier et al., 1983). It is important to note that individuals with neglect may exhibit adequate performance on some tasks, and poor performance on others because some tasks may be sensitive to different elements of neglect. In fact, there is some evidence that in the setting of acute stroke, performance on some measures of neglect syndromes tends to be inconsistent across time. However, the distribution of neglect subtype symptoms appears to become more discrete over time (Hamilton et al., 2008). Clinicians completing analyses of neuropsychological test performance in this population often examine and converge the pattern of scores across different tasks to arrive at a diagnosis of a particular subtype of neglect. However, patients also show high variability in performance on a single task, which can be due to fluctuations in general arousal state, fatigue, or attention-based distractibility secondary to stroke (Snyder & Nussbaum, 1998).

Most standardized measures of neglect are comprised of visual tasks that are carried out in the peripersonal space, and several can be adapted to tactile modalities or can be performed in far-extrapersonal space. In spite of the common analogs of these instruments, some investigators have suggested that these tasks tend to tap into several neuropsychological constructs, which may add variability to the test results. As such, general and broad assessment is needed. Below is a list of frequently used standardized instruments often used to identify and quantify the presence of USN:

1. *Cancellation Task* (Gauthier, Dehaut, & Jeanette, 1989)
2. *Clock-Drawing Test* (Freedman, Leach, Kaplan, Winocur, Shullman, & Delis, 1994)
3. *Line Bisection Test* (Schenkenberg, Bradford, & Ajax, 1980)
4. *National Institute of Health Stroke Scale* (Adams, Davis, Torner,

- Grimsman, & Vande Berg, 1998)
5. *Reading and Writing Subtests from the Behavioral Inattention Battery*  
(Wilson et al., 1987)

## EVALUATION OF POSTSTROKE DEPRESSION

Depression is by far the most common emotional response following stroke, with estimated prevalence rates ranging from a low of 19% (Robinson, 2003) to as much as 36% (Hackett, Yapa, Parag, & Anderson, 2005) of all stroke survivors. Poststroke depression (PSD) is associated with a wide range of negative outcomes, including decreased rehabilitation efficiency, (Gillen, Eberhardt, Tennen, Affleck, & Groszmann, 1999) functional limitations in ADL (Chemerinski, Robinson, & Kosier, 2001; Pohjasvaara, Vataja, Leepävuori, Kaste, & Erkinjuntti, 2001), and increased mortality (Chemerinski et al., 2001a; Gump, Matthews, Eberly, & Chang, 2005; House, Knapp, Bamford, & Vail, 2001).

Although the prevalence and comorbidities of PSD have been well documented, a consensus has not been reached concerning the definition, and valid methods of assessment have proven elusive. Discussion has focused on two issues, diagnostic criteria and construct validity of measures. With regard to diagnostic criteria, the presumed “gold standard” for clinical diagnosis of major depression, the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* (DSM-IV; American Psychiatric Association, 2000), includes vegetative and somatic symptoms such as weight loss/gain, decreased/increased appetite, and insomnia or hypersomnia. Many depression measures are validated using DSM-IV criteria and therefore include these symptoms. However, among persons with acute stroke, these symptoms may be related to their physical condition or the hospital environment, and may not be specific to depression. For example, the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) includes the items “I did not feel like eating,” “I had trouble keeping my mind on what I was doing,” and “I felt everything I did was an effort.” Similarly, in their study of depressive symptomatology in 189 stroke patients, Stein, Sliwinski, Gordon, and Hibbard (1996) found that the specificity of somatic symptoms in two commonly used measures was quite low, incorrectly classifying over 50% of nondepressed patients as depressed. Rybarczyk and his colleagues (Rybarczyk, Winemiller, Lazarus, & Haut, 1996) sought to remedy the problem of depression measures that include somatic items and have developed the only depression measure specifically validated for use

with patients with stroke.

In addition to a reliance on somatic symptoms, the content validity of measurement items may also be reduced when tests validated for use with the general population are used to assess depression in persons with stroke. For example, the Geriatric Depression Scale (GDS; Yesavage et al., 1983) asks, “have you dropped many of your activities and interests?” a question that likely has little predictive validity for recently hospitalized patients with stroke. Finally, many stroke survivors have significant cognitive limitations that affect their language functioning, and it may be difficult for them to comprehend test questions, particularly those utilizing reverse scaling or complex Likert-type responses.

Robinson (2003) has discussed the difficulty of accurate diagnosis of PSD in the absence of standardized methodology and criteria, but reported that the inclusion of somatic symptoms did not significantly impact diagnostic accuracy of PSD. In a study following 142 stroke survivors for 2 years, participants who had been identified as depressed during their initial evaluation continued to show a higher degree of both vegetative and psychological symptoms over the course of the study. Modifying *DSM-IV* criteria to exclude symptoms that were not significantly more frequent in the depressed patients (early awakening and weight loss) eliminated only three participants previously identified as depressed, and the unmodified *DSM-IV* criteria still retained positive predictive value (PPV) of 88% 2 years poststroke. However, other studies appear to contradict Robinson’s findings. Using discriminant analysis, de Coster, Leentjens, Lodder, and Verhey (2005) compared the sensitivity of somatic and nonsomatic symptoms of depression in 206 stroke survivors and found that depressed mood was the best discriminator between depressed and nondepressed patients, but somatic items had varying levels of predictive power. Reduced appetite, psychomotor retardation, and fatigue were sensitive predictors of depression, whereas early awakening, insomnia, and weight loss had relatively low discriminative power. Spalletta, Ripa, and Caltagirone (2005) applied *DSM-IV* criteria to 200 stroke survivors and found that 25% met criteria for major depressive disorder and 31% met criteria for minor depressive disorder. Frequencies of both vegetative and cognitive symptoms were different for all diagnostic groups. In particular, fatigue, insomnia, and psychomotor agitation/retardation differentiated those with minor depression from those without depression. A possible age-cohort effect has been shown in which older patients with stroke harbor more negative views of depression, which can

potentially impact their self-report (Roger & Johnson-Greene, 2008).

The assessment of PSD using self-report measures is also complicated by difficulties establishing accurate criterion validity. For example, Zimmerman, Posternak, and Chelminski (2005) found that although a consensus panel had initially recommended that remission of depression be defined as  $\leq 7$  on the 17-item Hamilton Rating Scale for Depression (HRSD), defining remission as the absence of clinically significant symptoms would require a cutoff of  $\leq 2$ . In a study of Dutch elders who had self-referred for depression treatment, a cutoff of  $\geq 25$  on the Center for Epidemiologic Studies Depression Scale offered optimal sensitivity and specificity, but if subthreshold depression or dysthymia was included, the optimal cutoff was  $\geq 22$  (Haringsma, Engels, Beekman, & Spinhoven, 2004). Further complications can be found in the self-report nature of depressive symptoms.

Roger and Johnson-Greene (2009) examined the sensitivity and specificity of four standardized measures for assessing depression in 67 adults with acute stroke admitted to an inpatient rehabilitation unit. Using *DSM-IV* criteria, major depression was present in 15% of the sample and minor depression in an additional 28%. The four depression measures yielded rates of depression ranging from 14% to 46% when their recommended clinical threshold scores were applied. CES-D had the greatest PPV at 28%. Cutoff scores were adjusted using receiver operating curve (ROC) analyses, and PPV ranged from 28% to 34% when using lower cutoff scores, representing an improvement for all measures. The authors concluded that common assessment measures for depression yield significantly different classification rates for depression in persons with acute stroke and that traditional threshold scores for commonly used objective depression measures may not be optimally sensitive for detection of depression in stroke populations.

In summary, there is no universally accepted definition or criteria for PSD. It is a construct that requires validation and is likely not synonymous with *DSM-IV* criteria for major depression. Thus, a major weakness for any research examining PSD is the lack of a “gold standard” definition for PSD. It is clear that the clinical thresholds of commonly used measures may under diagnose the presence of depression in this population. The clinical importance of “minor” or “subsyndromal” depression in a stroke survivor population has been discussed for over a decade, and there is debate as to whether it represents a separate construct from major depression. Even subthreshold depression appears to be correlated with function in persons with stroke. A double-blind randomized

treatment study (Chemerinski *et al.* 2001b) found that patients meeting criteria for either major or minor depression whose depression had remitted at follow-up showed significantly greater recovery in ADL functions than those whose depression did not remit.

## **REHABILITATION WITH STROKE SURVIVORS**

The pattern and extent of cognitive and emotional impairment varies widely among persons with stroke, which complicates the rehabilitation of this population. Although many persons with stroke eventually return to independent functioning, most will experience significant limitations in basic ADLs, such as bathing, dressing, and grooming, and instrumental activities of daily living (IADL), such as cooking, grocery shopping, and functioning in the community. In some instances, ability to perform routines (ADLs and IADLs) may be intact, but learning of new tasks and planning and performance of nonroutine activities are affected. Persons with stroke may also experience problems in social functioning and recreational functioning due to such difficulties as depression, apathy, and loss of interest or drive for once pleasurable activities. Few persons who were employed prior to their stroke will return to work. Social isolation and family dysfunction often occur following stroke due largely to care taking demands on others and to the patient's residual emotional dyscontrol, poor self-regulation of behavior, and personality changes. Persons with severe residual behavioral disturbance may not regain the ability to reside and function in the community safely without highly structured and controlled environments and supportive interventions. For persons who remain severely disabled, part-time or full-time supervision may be necessary.

Acute phase rehabilitation treatment includes subacute in-hospital care and acute inpatient hospital rehabilitation. Recommendations for postacute treatment are based upon the person's cognitive status, cognitive and functional goals, and supervision needs and can include home-based rehabilitation, outpatient rehabilitation, community reentry programs, comprehensive day treatment programs, and residential programs. Although individuals may exhibit physical disabilities following stroke, they often parallel prominent and persistent cognitive and emotional impairments; consequently, an emphasis of treatment is decreasing the impact of these neuropsychological difficulties on ADL, mobility, community integration, and vocation. Although interventions to reduce cognitive impairment or to teach compensatory strategies for cognition are a standard component of stroke rehabilitation programs at each stage along the continuum



of care, the effectiveness of cognitive rehabilitation has been controversial largely due to lack of research demonstrating efficacy.

Because stroke affects all areas of functioning, treatment involves an interdisciplinary team of experienced professionals, including, but not limited to, physiatry (physical medicine and rehabilitation physician), neuropsychology, occupational therapy, physical therapy, rehabilitation nursing, speech–language pathology, psychology, therapeutic recreation, vocational rehabilitation, case management, and social work. The neuropsychologist generally becomes involved in care at the inpatient rehabilitation phase of treatment and remains a core discipline through postacute rehabilitation. At each stage along the continuum of care, the neuropsychologist may play several key roles: (a) assessment and ongoing monitoring of neuropsychological functioning; (b) based on neuropsychological evaluation, providing treatment recommendations and planning for cognitive, emotional, and behavioral impairments; (c) providing education to the treatment team, patient, and family regarding neuropsychological functioning; (d) providing interventions for behavioral management and emotional functioning; and (e) providing interventions for cognition.

### **Acute Comprehensive Inpatient Rehabilitation**

Comprehensive inpatient rehabilitation (CIR) is a program of coordinated and integrated medical and rehabilitation services that comprises a comprehensive interdisciplinary treatment team. The inpatient program beds are located in a designated area and programs are generally provided within a hospital or hospital-affiliated facility. Program requirements typically include 3 or more hours of rehabilitation therapies each day and need for two or more rehabilitation therapy disciplines (e.g., physical therapy, occupational therapy, speech and language pathology, neuropsychology). General CIR goals are to assess and identify areas of cognitive and functional impairment resulting from the TBI, identify rehabilitation treatment goals with the patient and family, and provide rehabilitative interventions (e.g., retraining, compensation, and education) to increase functional independence and ability to return to a home and community setting.

### **Subacute Rehabilitation**

For persons who are not able to participate in the intensity of therapy provided in CIR programs or for persons who do not need intensive multiple therapies but do

require medical supervision of a physiatrist due to medical comorbidities, subacute rehabilitation provides an option. Subacute rehabilitation is recommended immediately following acute care and medical stabilization in cases in which the person does not meet criteria for the intensity of the CIR program. Alternately, subacute rehabilitation may be recommended immediately following CIR in cases in which patients require a reduced pace or more time to meet inpatient rehabilitation goals. These programs provide physical therapy, occupational therapy, and speech and language pathology, but at reduced frequency and intensity. Some programs also provide neuropsychology or psychology as a consultation service. Subacute programs are generally located within a hospital, hospital-based skilled nursing facility, skilled nursing facility, non-hospital-based residential facility, or hospital with transitional rehabilitation beds.

### **Home-Based Therapies**

Following inpatient hospitalization or upon completion of an integrated inpatient rehabilitation program, persons who continue to have functional impairments and who have a discharge setting that is determined to be safe may receive home-based therapies. Therapy within the home benefits persons with barriers or contraindications to participation in outpatient rehabilitation programs such as medical comorbidities, frailty, transportation difficulties, or need for therapies specifically to address functioning with the home. These home-based therapies are selected according to continued areas of functional impairment and may include physical therapy, occupational therapy, and/or speech and language therapy.

### **Outpatient Rehabilitation Therapies**

In the outpatient rehabilitation setting, persons have access to multiple therapy and rehabilitation services, including physiatry, physical therapy for ambulation/mobility impairment, occupational therapy for impairment in ADL, speech and language pathology for communication and language impairment, neuropsychology for cognitive and neurobehavioral impairment and adjustment to disability, vocational rehabilitation for return to work, and social work and case management. In the traditional outpatient therapy model, interventions and goals are discipline-specific, and persons schedule and attend treatments within the discipline(s) needed. Persons, for example, may attend only speech and language therapy for a certain number of hours per week, or persons may have 1

to 3 hours each of physical therapy, occupational therapy, or speech and language therapy each week. There is generally little or no integration of the therapies.

### **Comprehensive Day Treatment Programs**

Persons typically attend day-treatment programs for approximately 4 to 6 hours per day, for 3 to 5 days per week, generally for 2 to 6 months. Day treatment is designed to be intensive and structured and it combines group and individual therapies. Group treatments may be program wide or may be discipline-specific, and group interventions address specific goals such as social skills, cognitive skills (e.g., orientation, memory, attention, problem-solving), language and communication skills, vocational issues, and independent living skills. In addition, education for families is provided. One specific model of day-treatment program is called comprehensive/holistic day treatment or holistic neuropsychological rehabilitation. This is an intensive program with the following distinctive features: (a) a neuropsychological orientation with a neuropsychologist as a core treatment team member rather than a consultant to address cognitive, neurobehavioral, intrapersonal, interpersonal and psychosocial, and vocational issues; (b) integrated goal setting and outcome monitoring by a staff with transdisciplinary responsibilities; (c) frequent, formal staff meetings attended by core treatment team meetings and a team manager for each patient; (d) group interventions; (e) dedicated resources, including dedicated space, a core treatment team, and a patient-to-staff ratio of 2 to 1; (f) systematic inclusion of family members/close others on a weekly basis for education and adjustment/coping interventions and support; (g) inclusion of a dedicated independent-living or vocational trial; and (h) focus on outcomes in productivity, independent living, psychosocial adjustment, and emotional adjustment.

### **SUMMARY AND FUTURE DIRECTIONS**

Stroke remains a major cause of morbidity and mortality in the United States, particularly among the elderly. Persons with stroke continue to represent the leading source of referrals to rehabilitation hospitals warranting the need for health care professionals to become skilled in the treatment and rehabilitation of this needy population. In spite of its well-known prevalence, the potential for rendering large segments of our population disabled, and because of its huge burden on rehabilitation and financial resources, stroke represents one of the

least researched and least funded medical disorders. Ironically, only a diminutive amount of annual funding is provided in the United States for research and training in stroke rehabilitation. Although advances in the technology of critical care and emergency medicine over the past 40 years have significantly increased survivability of patients with stroke, in 2010 the total economic burden for stroke remained staggering and estimated to be \$ 73.7 billion ([American Heart Association, 2010](#)). Nonmodifiable at risk factors, including age, heredity, race, ethnicity, and gender have been implicated and identified as factors to consider in the understanding, evaluation, and treatment of stroke. More important, it is essential for clinicians working with stroke populations to assess whether they have the following common modifiable vascular risk factors: hypertension, cardiac conditions, diabetes mellitus, hypercholesterolemia, cigarette smoking, a history of TIA, or practice a maladaptive life style. Knowing the underlying malleability of these key modifiable features of stroke, psychologists working in rehabilitation settings can empower their stroke patients in taking control of these important aspects of their health and encourage them to be proactive and mindful of their choices for the future.

Hypertension is the single most important frequently occurring modifiable risk factor for most strokes ([Sacco et al., 1997](#)). Strokes can be classified as either ischemic (i.e., clots) or hemorrhagic (i.e., bleeds) in origin. The distinction between the two categories carries clinical importance for both evaluation and treatment purposes. Cognitive disorders during the acute stages of stroke are common and are important independent predictors of adverse outcome in victims of stroke. Currently, a range of brief cognitive measures is being used to assess for neuropsychological impairments in patients with acute stroke. The most recent of such measures shows promising application with stroke populations. The effects of stroke can lead to a number of neuropsychological disorders, which can include fluent and nonfluent aphasias, visual and verbal memory deficits, anosognosia and unawareness syndromes, and USN. Numerous standardized instruments are currently being used to delineate the presence of these disabling disorders. Psychiatrically, PSD is by far the most common emotional response following stroke. PSD is associated with a wide range of negative outcomes, including decreased rehabilitation efficiency, functional limitations in ADL, and increased mortality. Given the lack of specificity with conventional measures of clinical depression, evaluation of PSD may require using measures of depression that are specifically designed and validated for this neurologically impaired population. Acute inpatient rehabilitation goals are to

include identification of areas of cognitive and functional impairment resulting from stroke, establishing rehabilitation treatment goals with the patient and family, and providing rehabilitative interventions (e.g., retraining, compensation, education) to increase functional independence upon returning home. For persons who are unable to participate in intense therapy provided in CIR programs, but require ongoing medical supervision by a physiatrist due to medical comorbidities, subacute rehabilitation provides a viable option. Some individuals who continue to suffer from functional impairments and have a discharge setting that is determined to be safe may alternatively receive home-based therapies. In the traditional outpatient therapy model, interventions and goals are discipline-specific, and participants schedule and attend weekly treatments within the discipline(s) as needed. Day treatment is designed to be intensive and structured, often combining group and individual therapies. Lastly, group treatments are often available to stroke populations and may be program wide or may be discipline specific. Group interventions will commonly address specific goals such as social skills training, management of residual cognitive sequelae, language and communication skills, vocational issues, along with advancing independent living skills.

## REFERENCES

- Abbott, R. D., Curb, J. D., Rodriguez, B. L., Sharp, D. S., Burchfiel, C. M., & Yano, K. (1996). Effect of dietary calcium and milk consumption on risk of thromboembolic stroke in older middle-aged men: The Honolulu heart program. *Stroke*, *27*(5), 813–818.
- Abbott, R. D., Rodriguez, B. L., Burchfiel, C. M., & Curb, J. D. (1994). Physical activity in older middle-aged men and reduced risk of stroke: The Honolulu Heart Program. *American Journal of Epidemiology*, *139*(9), 881–893.
- Adams, H., Davis, P., Torner, J., Grimsman, K., & Vande Berg, J. (1998). *The NIH Stroke Scale*. Iowa City, IA: University of Iowa Health Care.
- American Heart Association. (2010). Heart disease and stroke statistics–2010 update: A report from the American Heart Association. *Circulation*, *121*, e46–e215.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Babinski, J. (1914). Contribution to the study of mental disturbance in organic cerebral hemiplegia (anosognosia). *Revue Neurologique*, *1*, 845–848.
- Barker-Collo, S., & Feigin, V. (2006). The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychology Review*, *16*(2), 53–64.
- Bates, E. (1994). Modularity, domain specificity and the development of language. *Discussions in Neuroscience*, *10*, 136–149.
- Benton, A. (1992). *Benton Visual Retention Test* (5th edn). San Antonio, TX: Pearson.
- Benton, A. L. (1974). *Visual retention test*. The Psychological Corporation.
- Benton, A. L., Hamsher, K. de S., Rey, G. I., & Sivan, A. B. (1994). *Multilingual Aphasia Examination* (3rd edn). Iowa City, IA: AJA Associates.
- Beschin, N., Cubelli, R., Della Sala, S., & Spinazzola, L. (1997). Left of what? The role of egocentric

- coordinates in neglect. *Journal of Neurology, Neurosurgery, and Psychiatry*, 63(4), 483–489.
- Billoski, T. V. (1992). *Introduction to paleontology 2* (6th ed.). New York, NY: Institutional Press.
- Bisiach, E., Vallar, G., Perani, D., Papagno, C., & Berti, A. (1986). Unawareness of disease following lesions of the right hemisphere: Anosognosia for hemiplegia and anosognosia for hemianopia. *Neuropsychologia*, 24(4), 471–482.
- Blumenfeld, H. (2002). *Neuroanatomy through clinical cases* (Vol. 1). Sunderland: Sinauer Associates.
- Brandt, J. (1991). The Hopkins Verbal Learning Test: Development of a new memory test with six equivalent forms. *Clinical Neuropsychologist*, 5(2), 125–142.
- Broderick, J., Brott, T., Kothari, R., Miller, R., Khoury, J., Pancioli, A., . . . Shukla, R. (1998). The greater Cincinnati/Northern Kentucky Stroke Study: Preliminary first-ever and total incidence rates of stroke among blacks. *Stroke*, 29(2), 415–421.
- Brown, G. G., & Eyler Zorrilla, L. T. (2001). Neuropsychological aspects of stroke. *Neuropsychology of Cardiovascular Disease* (pp. 301–324). Mahwah, NJ: Lawrence Erlbaum Associates.
- Buschke, H., & Fuld, P. A. (1974). Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology*, 24(11), 1019–1025.
- Cassidy, T. P., Lewis, S., & Gray, C. S. (1998). Recovery from visuospatial neglect in stroke patients. *Journal of Neurology, Neurosurgery, and Psychiatry*, 64(4), 555–557.
- Centers for Disease Control and Prevention. (2001). Prevalence of disabilities associated with health conditions among adults: United States 1999. *Morbidity and Mortality Weekly Report*, 56, 504–507.
- Chemerinski, E., Robinson, R. G., & Kosier, J. T. (2001a). Improved recovery in activities of daily living associated with remission of poststroke depression. *Stroke*, 32(1), 113–117.
- Chemerinski, E., Robinson, R. G., Arndt, S., & Kosier, J. T. (2001b). The effect of remission of poststroke depression on activities of daily living in a double-blind randomized treatment study. *Journal of Nervous and Mental Disease*, 189(7), 421–425.
- Cutting, J. (1978). Study of anosognosia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 41(6), 548–555.
- de Coster, L., Leentjens, A. F., Lodder, J., & Verhey, F. R. (2005). The sensitivity of somatic symptoms in poststroke depression: A discriminant analytic approach. *International Journal of Geriatric Psychiatry*, 20(4), 358–362.
- de Haan, E. H., Nys, G. M., & Van Zandvoort, M. J. (2006). Cognitive function following stroke and vascular impairment. *Current Opinion Neurology*, 19(6), 559–564.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *CVLT-II*. New York: The Psychological Corporation.
- Denes, G., Semenza, C., Stoppa, E., & Lis, A. (1982). Unilateral spatial neglect and recovery from hemiplegia a follow-up study. *Brain*, 105(3), 543–552.
- D’Esposito, M., Grossman, M., Payer, F., Onishi, K., Morrison, D., Sadek, A., & Alavi, A. (1998). Language comprehension and regional cerebral defects in frontotemporal degeneration and Alzheimer’s disease. *Neurology*, 50(1), 157–163.
- Doyon, J., & Milner, B. (1991). Right temporal-lobe contribution to global visual processing. *Neuropsychologia*, 29(5), 343–360.
- Ferro, J. M. (2001). Hyperacute cognitive stroke syndromes. *Journal of Neurology*, 248(10), 841–849.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). “Mini-mental state.” A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12(3), 189–198.
- Freedman, M., Leach, L., Kaplan, E., Winocur, G., Shulman, K., & Delis, D. C. (1994). *Clock drawing: A neuropsychological analysis*. New York, NY: Oxford University Press.
- Frisk, V., & Milner, B. (1990). The relationship of working memory to the immediate recall of stories following unilateral temporal or frontal lobectomy. *Neuropsychologia*, 28(2), 121–135.
- Gauthier, L., Dehaut, F., & Joanette, Y. (1989). The bells test: A quantitative and qualitative test for visual neglect. *International Journal of Clinical Neuropsychology*, 11(2), 49–54.

- Gillen, R., Eberhardt, T. L., Tennen, H., Affleck, G., & Groszmann, Y. (1999). Screening for depression in stroke: Relationship to rehabilitation efficiency. *Journal of Stroke and Cerebrovascular Diseases*, 8(5), 300–306.
- Goodglass, H., & Kaplan, E. (1983). *Boston diagnostic examination for aphasia*. Philadelphia: Lea and Febiger.
- Gottesman, R. F., & Hillis, A. E. (2010). Predictors and assessment of cognitive dysfunction resulting from ischaemic stroke. *Lancet Neurology*, 9(9), 895–905.
- Gump, B. B., Matthews, K. A., Eberly, L. E., & Chang, Y. F. (2005). Depressive symptoms and mortality in men: Results from the Multiple Risk Factor Intervention Trial. *Stroke*, 36(1), 98–102.
- Hackett, M. L., Yapa, C., Parag, V., & Anderson, C. S. (2005). Frequency of depression after stroke: A systematic review of observational studies. *Stroke*, 36(6), 1330–1340.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23, 56–62.
- Hamilton, R. H., Coslett, H. B., Buxbaum, L. J., Whyte, J., & Ferraro, M. K. (2008). Inconsistency of performance on neglect subtype tests following acute right hemisphere stroke. *Journal of the International Neuropsychological Society*, 14, 23–32.
- Haringsma, R., Engels, G. I., Beekman, A. T., & Spinhoven, P. (2004). The criterion validity of the Center for Epidemiological Studies Depression Scale (CES-D) in a sample of self-referred elders with depressive symptomatology. *International Journal of Geriatric Psychiatry*, 19(6), 558–563.
- Hartman-Maeir, A., Soroker, N., & Katz, N. (2001). Anosognosia for hemiplegia in stroke rehabilitation. *Neurorehabilitation and Neural Repair*, 15(3), 213–222.
- Heilman, K. M., Watson, R. T., & Valenstein, E. (1993). Neglect and related disorders. *Clinical Neuropsychology*, 3, 279–336.
- Helm-Estabrooks, N. (1992). *ADP: Aphasia Diagnostic Profiles*. Chicago, IL: Riverside Publishing.
- Hier, D. B., Modlock J., & Caplan, L. R. (1983). Recovery of behavioral abnormalities after right hemisphere stroke. *Neurology*, 33, 345–350.
- Hillis, A. E. (2004). Progress in cognitive neuroscience research on dysgraphia: Introduction. *Neurocase*, 10(2), 89–90.
- Hochstenbach, J., Mulder, T., van Limbeek, J., Donders, R., & Schoonderwaldt, H. (1998). Cognitive decline following stroke: A comprehensive study of cognitive decline following stroke. *Journal of Clinical and Experimental Neuropsychology*, 20(4), 503–517.
- House, A., Knapp, P., Bamford, J., & Vail, A. (2001). Mortality at 12 and 24 months after stroke may be associated with depressive symptoms at 1 month. *Stroke*, 32(3), 696–701.
- Howard, G., Anderson, R., Sorlie, P., Andrews, V., Backlund, E., & Burke, G. L. (1994). Ethnic differences in stroke mortality between non-Hispanic whites, Hispanic whites, and blacks. The National Longitudinal Mortality Study. *Stroke*, 25(11), 2120–2125.
- Jehkonen, M., Dastidar, P., Laippala, P., & Vilkki, J. (2000). Unawareness of deficits after right hemisphere stroke: Double-dissociations of anosognosias. *Acta Neurological Scandinavica*, 102, 378–384.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test* (2nd edn). Philadelphia, PA: Lea & Febiger.
- Katz, N., Hartman-Maeir, A., Ring, H., & Soroker, N. (1999). Functional disability and rehabilitation outcome in right hemisphere damaged patients with and without unilateral spatial neglect. *Archives of Physical Medicine and Rehabilitation*, 80(4), 379–384.
- Kaufman, D. M. (Ed.). (2007). *Clinical neurology for psychiatrists* (6th ed.). Philadelphia, PA: Saunders.
- Kay, J., Lesser, R., & Coltheart, M. (1992). *PALPA: Psycholinguistic assessments of language processing in aphasia*. Hove, England: Lawrence Erlbaum Associates.
- Kiely, D. K., Wolf, P. A., Cupples, L. A., Beiser, A. S., & Myers, R. H. (1993). Familial aggregation of stroke. The Framingham Study. *Stroke*, 24(9), 1366–1371.
- Lansing, A. E., Max, J. E., Delis, D. C., Fox, P. T., Lancaster, J., Manes, F. F., & Schatz, A. (2004). Verbal learning and memory after childhood stroke. *Journal of the International Neuropsychological Society*,

- 10(5), 742–752.
- Larson, E. B., Kirschner, K., Bode, R. K., Heinemann, A. W., Clorfene, J., & Goodman, R. (2003). Brief cognitive assessment and prediction of functional outcome in stroke. *Topics in Stroke Rehabilitation*, 9(4), 10–21.
- Larson, E., Kirschner, K., Bode, R., Heinemann, A., & Goodman, R. (2005). Construct and predictive validity of the repeatable battery for the assessment of neuropsychological status in the evaluation of stroke patients. *Journal of Clinical and Experimental Neuropsychology*, 27(1), 16–32.
- Lazar, R. M., Minzer, B., Antoniello, D., Festa, J. R., Krakauer, J. W., & Marshall, R. S. (2010). Improvement in aphasia scores after stroke is well predicted by initial severity. *Stroke*, 41(7), 1485–1488.
- Lim, C., & Alexander, M. P. (2009). Stroke and episodic memory disorders. *Neuropsychologia*, 47(14), 3045–3058.
- MacMahon, S., & Rodgers, A. (1994). The epidemiological association between blood pressure and stroke: Implications for primary and secondary prevention. *Hypertension Research*, 17(Suppl. I), S23–S32.
- Massey, L. K. (2001). Dairy food consumption, blood pressure and stroke. *Journal of Nutrition*, 131(7), 1875–1878.
- Marshall, R. S., Perera, G. M., Lazar, R. M., Krakauer, J. W., Constantine, R. C., & DeLaPaz, R. L. (2000). Evolution of cortical activation during recovery from corticospinal tract infarction. *Stroke*, 31(3), 656–661.
- Menon-Nair, A., Korner-Bitensky, N., Wood-Dauphinee, S., & Robertson, E. (2006). Assessment of unilateral spatial neglect post stroke in Canadian acute care hospitals: Are we neglecting neglect? *Clinical Rehabilitation*, 20(7), 623–634.
- Mesulam, M. M. (1985). Attention, confusional states, and neglect. In M. M. Mesulam (Ed.), *Principles of behavioral neurology* (pp. 125–168). Philadelphia, PA: FA Davis.
- Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., . . . Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, 53(4), 695–699.
- Nys, G. M. S., Van Zandvoort, M. J. E., De Kort, P. L. M., Van der Worp, H. B., Jansen, B. P. W., Algra, A., . . . De Haan, A. (2005a). The prognostic value of domain-specific cognitive abilities in acute first-ever stroke. *Neurology*, 64, 821–827.
- Nys, G. M., Van Zandvoort, M. J., De Kort, P. L., Jansen, B. P., Van der Worp, H. B., Kappelle, L. J., & De Haan, E. H. (2005b). Domain-specific cognitive recovery after first-ever stroke: A follow-up study of 111 cases. *Journal of the International Neuropsychological Society*, 11(7), 795–806.
- O’Brien, J. T. (2006). Vascular cognitive impairment. *American Journal of Geriatric Psychiatry*, 14(9), 724–733.
- Orfei, M. D., Robinson, R. G., Prigatano, G. P., Starkstein, S., Rüsçh, N., Bria, P., Caltagirone, C., & Spalletta, G. (2007). Anosognosia for hemiplegia after stroke is a multifaceted phenomenon: A systematic review of the literature. *Brain*, 130(Pt 12), 3075–3090.
- Osterrieth, P. A. (1944). Filetest de copie d’une figure complex: Contribution a l’étude de la perception et de la memoire [The test of copying a complex figure: A contribution to the study of perception and memory]. *Archives de Psychologie*, 30, 286–356.
- Pendlebury, S. T., Cuthbertson, F. C., Welch, S. J., Mehta, Z., & Rothwell, P. M. (2010). Underestimation of cognitive impairment by Mini-Mental State Examination versus the Montreal Cognitive Assessment in patients with transient ischemic attack and stroke: A population-based study. *Stroke*, 41(6), 1290–1293.
- Petrea, R. E., Beiser, A. S., Seshadri, S., Kelly-Hayes, M., Kase, C. S., & Wolf, P. A. (2009). Gender differences in stroke incidence and poststroke disability in the Framingham heart study. *Stroke*, 40(4), 1032–1037.
- Pia, L., Neppi-Modona, M., Ricci, R., & Berti, A. (2004). The anatomy of anosognosia for hemiplegia: A meta-analysis. *Cortex*, 40(2), 367–377.



- Pohjasvaara, T., Vataja, R., Leppävuori, A., Kaste, M., & Erkinjuntti, T. (2001). Depression is an independent predictor of poor long-term functional outcome poststroke. *European Journal of Neurology*, *8*(4), 315–319.
- Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*, 385–401.
- Rashid, P., Leonardi-Bee, J., & Bath, P. (2003). Blood pressure reduction and secondary prevention of stroke and other vascular events: A systematic review. *Stroke*, *34*(11), 2741–2748.
- Rey, A. (1964). *L'examen clinique en psychologie*. Paris: Presses Universitaires de France.
- Robinson, R. G. (2003). Poststroke depression: Prevalence, diagnosis, treatment, and disease progression. *Biological Psychiatry*, *54*(3), 376–387.
- Roger, P. R., & Johnson-Greene, D. (2009). Comparison of assessment measures for poststroke depression. *Clinical Neuropsychologist*, *23*(5), 780–793.
- Roger, P. R., & Johnson-Greene, D. (2008). Attitudes towards depression among rehabilitation participants with acute stroke: Evidence of an age cohort effect. *Rehabilitation Psychology*, *53*(2), 210–214.
- Ropper, A. H., & Brown, R. H. (2005). *Adams and Victor's principles of neurology*. (8th edn). New York: McGraw Hill.
- Rossi, P. W., Kheifets, S., & Reding, M. J. (1990). Fresnel prisms improve visual perception in stroke patients with homonymous hemianopia or unilateral visual neglect. *Neurology*, *40*(10), 1597–1599.
- Rosamond, W., Flegal, K., Friday, G., Furie, K., Go, A., Greenlund, K., Haase, N., . . . Hong, Y. (2007). Heart disease and stroke statistics—2007 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, *115*(5), e69–e171.
- Rybarczyk, B., Winemiller, D. R., Lazarus, L. W., & Haut, A. (1996). Validation of a depression screening measure for stroke inpatients. *American Journal of Geriatric Psychiatry*, *4*(2), 131.
- Sacco, R. L., Anand, K., Lee, H. S., Boden-Albala, B., Stabler, S., Allen, R., & Paik, M. C. (2004). Homocysteine and the risk of ischemic stroke in a tri-ethnic cohort: The Northern Manhattan Study. *Stroke*, *35*, 2263–2269.
- Sacco, R. L., Benjamin, E. J., Broderick, J. P., Dyken, M., Easton, J. D., Feinberg, W., . . . Wolf, P. A. (1997). American Heart Association Prevention Conference IV: Prevention and rehabilitation of stroke: Risk factors. *Stroke*, *28*(7), 1507–1517.
- Schenkenberg, T., Bradford, D. C., & Ajax, E. T. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology*, *30*(5), 509–517.
- Schouten, E. A., Schiemanck, S. K., Brand, N., & Post, M. W. (2009). Long-term deficits in episodic memory after ischemic stroke: Evaluation and prediction of verbal and visual memory performance based on lesion characteristics. *Journal of Stroke and Cerebrovascular Diseases*, *18*(2), 128–138.
- Shinton, R., & Beevers, G. (1989). Meta-analysis of relation between cigarette smoking and stroke. *British Medical Journal*, *298*(6676), 789–794.
- Snyder, P. J., & Nussbaum, P. D. (Eds.). (1998). *Clinical neuropsychology* (1st ed.). Washington, DC: American Psychological Association.
- Spalletta, G., Ripa, A., & Caltagirone, C. (2005). Symptom profile of DSM-IV major and minor depressive disorders in first-ever stroke patients. *American Journal of Geriatric Psychiatry*, *13*(2), 108–115.
- Starkstein, S. (2010). E. 06.01 The stroke patient as a challenge for the psychiatrist. *European Neuropsychopharmacology*, *20*, S212.
- Starkstein, S. E., Fedoroff, J. P., Price, T. R., & Leiguarda, R. (1993). Neuropsychological deficits in patients with anosognosia. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*, *6*, 43–48.
- Stein, P. N., Sliwinski, M. J., Gordon, W. A., & Hibbard, M. R. (1996). Discriminative properties of somatic and nonsomatic symptoms for post stroke depression. *Clinical Neuropsychologist*, *10*(2), 141–148.
- Taylor, T. N., Davis, P. H., Torner, J. C., Holmes, J., Meyer, J. W., & Jacobson, M. F. (1996). Lifetime cost of stroke in the United States. *Stroke*, *27*(9), 1459–1466.
- Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review.

- Journal of the American Geriatrics Society*, 40(9), 922–935.
- Wattigney, W. A., Croft, J. B., Mensah, G. A., Alberts, M. J., Shephard, T. J., Gorelick, P. B., . . . Collins, J. L. (2003). Establishing data elements for the Paul Coverdell National Acute Stroke Registry: Part 1: Proceedings of an expert panel. *Stroke*, 34(1), 151–156.
- Wechsler, D. (2009). *Wechsler Memory Scale* (4th edn). San Antonio, TX: Pearson.
- West, R., Hill, K., Hewison, J., Knapp, P., & House, A. (2010). Psychological disorders after stroke are an important influence on functional outcomes: A prospective cohort study. *Stroke*, 41(8), 1723–1727.
- Wilde, M. C. (2006). The validity of the repeatable battery of neuropsychological status in acute stroke. *Clinical Neuropsychologist*, 20(4), 702–715.
- Wilson, B. A., Cockburn, J., & Halligan, P. W. (1987). *Behavioral Inattention Test*. Fareham, UK: Thames Valley Test Company.
- Wolf, P. A., D'Agostino, R. B., Kannel, W. B., Bonita, R., & Belanger, A. J. (1988). Cigarette smoking as a risk factor for stroke. The Framingham Study. *Journal of the American Medical Association*, 259(7), 1025–1029.
- Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V., Adey, M., & Leirer, V. O. (1982). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research*, 17(1), 37–49.
- Zimmerman, M., Posternak, M. A., & Chelminski, I. (2005). Is the cutoff to define remission on the Hamilton Rating Scale for Depression too high? *The Journal of Nervous and Mental Disease*, 193(3), 170–175.
- Zinn, S., Bosworth, H. B., Hoenig, H. M., & Swartzwelder, H. S. (2007). Executive function deficits in acute stroke. *Archives of Physical Medicine and Rehabilitation*, 88(2), 173–180.

# Cognitive Rehabilitation Following Neurosurgical Intervention

*F. Scott Winstanley and Sherry T. Thrasher*

Cognitive rehabilitation has made substantial progress over the years in improving cognitive functioning in individuals with various types of neurologic injury. Cognitive morbidity has been reduced and overall cognitive abilities have improved with the implementation of empirically validated cognitive remediation strategies in individuals with a variety of neurologic disorders. The majority of studies examining the efficacy of cognitive rehabilitation have focused primarily on individuals who have suffered stroke and traumatic brain injury (TBI). However, cognitive rehabilitation following neurosurgical intervention (e.g., epilepsy surgery) remains underrepresented in the literature. “Neurosurgical intervention” is a broad term and can include numerous different procedures, including tumor resections, arteriovenous malformation (AVM) resection, deep brain stimulation (DBS), and a variety of epilepsy surgeries, including corpus callosotomy, lesionectomy, multiple subpial transections (MST), selective amygdalohippocampectomy, frontal lobectomy, and anterior temporal lobectomy. Describing the vast amount of procedures and examining the cognitive morbidity associated with these procedures in such a heterogeneous population is beyond the scope of a single chapter. Thus, it is our goal to focus on one of the most heavily researched neurosurgical interventions and its subsequent cognitive morbidity, that is, patients undergoing temporal lobe resection for medically intractable epilepsy.

This chapter serves to educate neuropsychologists and other practitioners in the field of cognitive rehabilitation about individuals undergoing temporal

lobectomy focusing specifically on (a) the comprehensive preoperative assessment prior to which includes state-of-the-art neurodiagnostic testing as well as baseline neuropsychological assessment, (b) the neurosurgical intervention and cognitive morbidity associated with the intervention, and (c) preoperative and postoperative neural plasticity of the epileptic brain (functional/structural neural reorganization) and postoperative cognitive rehabilitation.

## **EPILEPSY SURGERY AND TEMPORAL LOBE RESECTION**

Surgical resection of epileptogenic tissue is an effective treatment for individuals with medically refractory seizures (Télléz-Zenteno, Dhar, & Wiebe, 2005; Wiebe, Blume, Girvin, & Eliasziw, 2001). The most common surgical resection site is in the anterior temporal lobes, and surgical resection in this area can have long-term seizure freedom success upwards of 60% to 80% (McIntosh, Wilson, & Berkovic, 2001; Télléz-Zenteno et al., 2005). However, resection of anterior medial temporal lobe structures is accompanied by cognitive morbidity, especially with resections in the language-dominant (traditionally left) anterior temporal lobe. Verbal memory decline is a common finding in left anterior temporal lobectomy (L-ATL) and has been shown to occur in upwards of 30% to 60% of L-ATL cases (Baxendale, Thompson, Harkness, & Duncan, 2006; Binder et al., 2008; Chelune, Naugle, Lüders, & Awad, 1991; Chiaravalloti & Glosser, 2001; Gleissner, Helmstaedter, Schramm, & Elger, 2004; Helmstaedter & Elger, 1996; Hermann et al., 1995; Kneebone, Lee, Wade, & Loring, 2007; Lee, Yip, & Jones-Gotman, 2002; Lineweaver et al., 2006; Loring et al., 1995; Martin et al., 1998; Sabsevitz, Swanson, Morris, Mueller, & Seidenberg, 2001; Stroup et al., 2003). Postoperative language deficits, particularly naming deficits, are also a common finding in L-ATL and occur in about 25% to 60% of cases (Bell, Davies, Hermann, & Walters, 2000; Hamberger, Seidel, McKhann, & Goodman, 2010; Hamberger, Seidel, McKhann, Perrine, & Goodman, 2005; Hermann, Wyler, Somes, & Clement, 1994; Langfitt & Rausch, 1996; Sabsevitz et al., 2003). The goal of epilepsy surgery is to significantly reduce, if not cure, epileptic seizures in the patient while taking extensive precautions to reduce cognitive morbidity. Identifying preoperative risk factors of postoperative morbidity is a critical part in the patients overall care. This is done through an extensive presurgical work-up, the stages of which, this chapter examines.

### **Presurgical Assessment**

The comprehensive preoperative assessment at most epilepsy centers includes baseline neuropsychological testing, functional and structural magnetic resonance imaging (MRI), electroencephalography (EEG) recordings, computed tomography (CT)/positron emission tomography (PET) scans, magnetoencephalography (MEG), and the intracarotid amobarbital procedure (IAP). In addition to identifying the seizure focus, results of these studies also inform the neurosurgeon, as well as the patient, of the potential risk factors for cognitive morbidity following the surgery. Thus, an informed decision can be reached by the patient, in collaboration with their neurosurgical team, regarding the feasibility of a neurosurgical intervention. This information can also assist clinicians in developing specific cognitive rehabilitation treatment plans that will cater to the potential deficits of each patient.

## Neuropsychological Assessment

Neuropsychological assessment is a critical part of the preoperative assessment and postoperative assessment of individuals undergoing ATL and is a standard level of care at the majority of major epilepsy surgery centers. The preoperative assessment establishes whether the candidate's baseline cognitive abilities can assist in lateralizing seizure focus and cerebral dysfunction (Busch, Frazier, Haggerty, & Kubu, 2005; Frazier, Iampietro, Chapin, & Kubu, 2009; Jones-Gotman et al., 2010; Loring et al., 2008) and can also be useful in identifying inconsistencies in functioning compared to results seen with EEG and structural MRI, most often observed in individuals with atypical language representation. Baseline neuropsychological testing includes in-depth assessment of a number of cognitive domains, including intellectual functioning, verbal and nonverbal memory, language (e.g., expressive speech, comprehension, naming, repetition, and reading), attention/concentration, executive functioning, visual spatial abilities, and sensorimotor abilities. A complete assessment with diagnostic clinical interview ranges from 5 to 8 hours. Preferably, the assessment is conducted in 1 full day, but can be divided into 2 half-days if needed. The following reviews a standard battery of tests used to measure each cognitive domain in a typical presurgical evaluation.

## Intellectual Functioning

Comprehensive preoperative neuropsychological assessments traditionally include some form of intelligence testing. Though early studies showed some promise in determining seizure lateralization with discrepancies between verbal

IQ (VIQ) and performance IQ (PIQ), a larger study of 215 patients showed that VIQ/PIQ asymmetry correctly lateralized only 17% of patients and incorrectly lateralized a total of 13% (Hermann et al., 1995). Though not of lateralizing value, these measures are highly reliable and, in conjunction with educational and occupational history, can help determine premorbid level of functioning, which offers a baseline of cognitive performance to which other cognitive tests can be compared. Thus, an individual with a superior IQ with low-average verbal memory scores, may indicate a decline in premorbid abilities, whereas IQ scores in the low-average range would be more commensurate. The most commonly used IQ measure among many epilepsy centers is the *Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV)*; (Wechsler, 2008).

## Attention

“Attention” is a broad and complex cognitive entity that refers to a number of cognitive processes. In its most basic form, attention includes the fundamental ability to receive and respond to sensory information. There are multiple aspects of attention, including simple attention, selected or focused attention (ability to attend to one stimuli selectively while blocking out others), sustained attention (ability to focus on a particular stimulus for an extended period of time), and divided attention (focusing primarily on one stimulus, while able to keep other information in the background). This can be further divided into auditory versus visual attention. This complex process incorporates a number of neuroanatomical regions, including the frontal lobes, anterior cingulate cortex, premotor and prefrontal regions, dorsolateral prefrontal cortex, posterior parietal systems, as well as extensive subcortical white matter networks. Auditory tests of attention evaluate the functioning of the dominant hemisphere, whereas visual measures evaluate the functioning of the nondominant hemisphere to assist with seizure lateralization. Digit span is an auditory measure of simple attention (Wechsler, 2008). Speech sounds and seashore rhythms (Lezak, Howieson, & Loring, 2004) are auditory measures of sustained attention or ability to maintain attention over a given period of time. Measures of visual, divided attention include the *Stroop Test* (Stroop, 1935), *Trail Making Tests* (Army Individual Test Battery, 1944), and *Symbol Digit Modality Tests (SDMT)*; (Smith, 1982). Additional measures of sustained attention include the “Go/No-Go” test and the *Continuous Performance Test-Second Edition (CPT-2)* (Conners, 2001).

## Memory

Memory abilities, particularly declarative memory, can be viewed as having three fundamental stages: (a) encoding, (b) consolidation (storage), and (c) retrieval of information. *Encoding* is the stage at which the information begins to be registered or begins to enter memory. *Consolidation* is the maintenance and storage of this information. *Retrieval* is the ability to recall the information when necessary. The nature of memory deficits observed in ATL patients typically includes difficulty learning new information, that is, difficulties with encoding and consolidation. The neuroanatomic underpinnings of the encoding and consolidation process consist of the hippocampus, including dentate gyrus and subiculum, as well as entorhinal cortex, perirhinal cortex, and parahippocampal gyrus (Zola-Morgan & Squire, 1993). These structures undergo extensive resection in ATL, which in turn can cause postoperative memory problems in certain patients.

**Verbal Memory:** Memory assessment includes both verbal and nonverbal measures. A number of list-learning tests are used across various centers to assess verbal memory, but the *Rey Auditory Verbal Learning Test (RAVLT)* has recently been adopted by the NIH Common Data Elements Consortium as the preferred learning test for verbal memory assessment in patients undergoing work-up for epilepsy surgery, as this test has been demonstrated to be the most sensitive in identifying verbal memory impairment in left, language dominant temporal lobe epilepsy patients (Loring et al., 2008). The *California Verbal Learning Test-II (CVLT-II)* and the *Selective Reminding Test (SRT)* are other common list learning tests. (Buschke, 1973; Delis, Kramer, Kaplan, & Ober, 2000). Assessment of verbal memory abilities is one of the more important aspects of the comprehensive preoperative assessment in epilepsy surgery. Preoperative verbal memory performance on neuropsychological testing is one of the strongest predictors of postoperative verbal memory decline in left, language-dominant temporal lobe resections, with patients demonstrating higher, intact verbal memory abilities at greater risk for significant postoperative verbal memory decline (Baxendale, Thompson, Harkness, & Duncan, 2007; Baxendale et al., 2006; Binder et al., 2008; Bonelli et al., 2010; Chelune et al., 1991; Davies, Risse, & Gates, 1998; Gleissner et al., 2004; Helmstaedter & Elger, 1996; Hermann et al., 1995; Jokeit et al., 1997; Lineweaver et al., 2006; Stroup et al., 2003).

**Nonverbal Memory:** Nonverbal memory focuses on mesial temporal structures of the nondominant temporal lobe. Nonverbal memory tests have shown to be less sensitive to identifying nondominant seizure foci and do not

predict postoperative outcome as well in right temporal lobe (RTL) epilepsy patients as verbal memory tests do in left temporal lobe (LTL) epilepsy patients. Additionally, there is not a universally accepted nonverbal measure. A common nonverbal memory test is the *Rey-Osterrieth Complex Figure Test (ROCFT)* (Osterrieth, 1944). The *Brown Location Test (BLT)*, a dot location test, thought to be a “pure” nonverbal memory measure is being used by some epilepsy centers throughout the country (Brown et al., 2010). Memory for faces, a subtest on the *Wechsler Memory Scale-Third Edition (WMS-III)* (Wechsler, 1997), is also thought to be a “pure” measure of nonverbal memory. However, the current *Wechsler Memory Scales-Fourth Edition (WMS-IV)* does not include the faces subtest (Wechsler, 2008). Nonverbal memory deficits after right anterior temporal lobectomy have been observed, but are not as consistent or frequent as the material specific verbal memory decline in L-ATL patients (Lee et al., 2002).

## Language

A comprehensive assessment of language includes auditory naming (Hamberger et al., 2003) and visual confrontation naming (Kaplan, Goodglass, & Weintraub, 1983), as well as verbal fluency, reading, sentence repetition, and assessment of comprehension, which utilizes either a token test or a brief exam of complex ideational material (Kaplan et al., 1983). Some studies have demonstrated the efficacy of the *Boston Naming Test (BNT)*, a visual confrontation naming test, for lateralization of side of surgery (Busch et al., 2005, 2009; Keary, Frazier, Busch, Kubu, & Iampietro, 2007; Loring et al., 2008). In addition, auditory naming (Hamberger et al., 2003) has also been shown to lateralize left temporal lobe epilepsy (TLE) and also predict decline in left temporal lobectomy. More recently, a newer test of *Famous Faces/Famous Landmarks* (Tranel, 2006) has been shown to be sensitive to LTL dysfunction. Some studies have indicated the ability of high preoperative performance on language tests to predict postoperative outcome (Yucus & Tranel, 2007).

## Executive Functioning

Executive functioning is a broad category divided into multiple subcategories, including fluency, concept formation, planning, organization, and working memory. Like attention, executive functioning tasks can be either auditory or visual measures, which assist in lateralizing/localizing seizure focus. From a neuroanatomical perspective, tests of executive functioning are accessing the functions of the frontal lobes. Specifically, the anterior cingulate cortex and



inferior frontal cortex influence verbal fluency and verbal generation tasks, such as animal naming. The *Wisconsin Card Sorting Test (WCST)* is a measure of concept formation and set shifting. Neuroanatomically, the *WCST* is a measure of the dorsolateral prefrontal cortex. Planning abilities are accessed by tower tests and a figure-drawing test, such as the *ROCFT*. Arithmetic and letter-number sequencing are measures of working memory (Wechsler, 2008).

## Visual Spatial

Deficits in visual-spatial/visual perceptual abilities can be indicative of dysfunction in the nondominant hemisphere. Visual-spatial tests usually have some degree of mental manipulation. The *Judgment of Line Orientation (JLO)* and *Facial Recognition* (Benton, 1975, 1994) are two standard measures of visual-spatial abilities. The Hooper Visual Organization Test is a measure of visual organization. Other tests of visual-spatial abilities include block design, clock drawing, and the copy phase of the *ROCFT*.

## Motor

Motor tasks are used in neuropsychological assessment for the purpose of lateralization. The primary motor cortex is the primary anatomy structure that is assessed through motor testing. There are three motor tasks that are commonly used and each test accesses different motor abilities. Grip strength measures an individual's strength, finger tapping is a measure of speed, and the grooved pegboard is a measure of fine-motor dexterity (Lezak et al., 2004). It is expected that there will be some variation in performance between the dominant and nondominant hand; however, if there is a significant discrepancy, it can assist in lateralizing the profile. Another aspect of motor functioning is somatosensory assessment, which evaluates tactile, auditory, and visual senses.

## Personality

A personality assessment is included to determine whether the patient has any psychiatric problems that should be further evaluated or treated prior to surgery. Screening measures include the *Beck Depression Inventory (BDI)* and *Beck Anxiety Inventory (BAI)*; (Beck, Steer, & Brown, 1996). BDI and BAI are typically administered to patient's who do not have a psychiatric history and are not reporting current emotional distress. The *Minnesota Multiphasic Personality Inventory-Second Edition (MMPI-2)* is an example of a longer objective

personality measure and is the most common personality measure used (Piotrowski & Keller, 1989).

## Neurodiagnostic Tools

### Electroencephalography

EEG records electrical activity from the cerebral cortex and is measured in microvolts ( $\mu\text{V}$ ). EEG recordings are the single most important tool for diagnosing a patient with epilepsy and also play a critical role in identifying epileptogenic seizure foci. Initially, these recordings are conducted using scalp electrodes placed around standard areas of the head. Patients in the initial phase of a presurgical work-up (phase I) will often undergo long-term continuous audio visual (CAV) EEG monitoring (sometimes referred to as “cave” CAV-EEG). In addition to placement of scalp electrodes, the patient is also videotaped during the seizure to correlate semiology with electrical discharges seen on EEG. Often, the EEG, in conjunction with other neurodiagnostic tests, is sufficient to identify the seizure focus and enable the patient to proceed to surgery. However, when results remain inconclusive, the patient can undergo intracranial EEG with subdural electrode placement for more accurate recordings. This more invasive procedure is discussed further in a later section.

### Structural MRI

Comprehensive epilepsy centers have all patients undergo preoperative structural MRI (if not contraindicated) for identification of any organic lesion, malformation, or structural abnormality. Structural MRI assists with lateralization of seizure focus as well as in identifying risk of cognitive morbidity. Research has demonstrated that the degree of structural abnormality and neuronal cell loss of the left, language-dominant hippocampus is related to preoperative verbal memory performance (Rausch, 1987; Saling et al., 1993; Sass et al., 1992), and degree of neuronal loss/hippocampal sclerosis in the hippocampus undergoing resection has been shown to correlate with postoperative verbal memory decline (Hermann, Wyler, Somes, Berry, & Dohan, 1992; Martin et al., 2000; Rausch & Babb, 1993; Sass et al., 1992). Absence of hippocampal atrophy has also been related to better preoperative verbal memory performance and greater risk of postoperative verbal memory decline (Lencz et al., 1992; Trenerry et al., 1993). In addition to being correlated with verbal memory, research has also implicated the structural integrity of the

hippocampus as playing a role in visual confrontation naming abilities. Studies have shown that resection of a healthy, nonsclerotic hippocampus in the left, language-dominant hemisphere is related to greater decline in postoperative naming abilities and that these naming errors tend to be more common for words that are acquired later in life (Bell et al., 2000; Yucus & Tranel, 2007). Other studies have shown that removal of the hippocampus affects auditory but not visual naming abilities (Hamberger et al., 2010). Quantitative volumetrics of the LTL have also been shown to correlate with confrontation naming ability (Seidenberg, Geary, & Hermann, 2005). However, it is important to note that structural abnormalities in temporal lobe epilepsy patients extend beyond the mesial temporal lobes and impair a number of other cognitive abilities, which have been shown by more recent studies (Dabbs, Jones, Seidenberg, & Hermann, 2009).

### 2-[18F]-fluoro-2-deoxy-D-glucose–Positron Emission Tomography

Interictal 2-[18F]-fluoro-2-deoxy-D-glucose (FDG)–PET is useful in the preoperative assessment of individuals with temporal lobe epilepsy, as hypometabolism in temporal lobe areas has been shown to lateralize seizure focus. However, the utility of FDG–PET to predict cognitive outcome has been mixed. One study indicated a relationship suggesting a lack of PET hypometabolism being related to greater postoperative verbal memory decline (Griffith et al., 2000), whereas Leeman, Leveroni, and Johnson (2009) failed to show a consistent relationship to preoperative PET hypometabolism and postoperative verbal memory decline.

### Single-Photon Emission Computed Tomography

Ictal single-photon emission computed tomography (SPECT) has shown to be a highly effective tool in lateralizing seizure focus in individuals with mesial temporal lobe epilepsy. Ictal SPECT is performed during the EEG monitoring and entails a radioactive isotope, usually  $^{99m}\text{Tc}$ -hexamethylpropylamine, ( $^{99m}\text{Tc}$ -HMPAO) being injected within 5 to 20 seconds after seizure onset. Sensitivity rates have been found to be as high as 97% in mesial temporal lobe epilepsy patients.

### Proton Magnetic Resonance Spectroscopy

Proton magnetic resonance spectroscopy ( $^1\text{H}$ -MRS) measures metabolic changes based on signals from N-acetyl aspartate (NAA) and creatine + phosphocreatine (Cr) and choline (Cho)-containing compounds and is a sensitive tool for measuring neuronal cell loss and identifying seizure focus in patients with temporal lobe epilepsy. In addition to assisting in seizure lateralization, hippocampal  $^1\text{H}$ -MRS results have also been shown to be related to cognitive abilities such as visual confrontation naming ([Martin et al., 1999](#); [Sawrie et al., 2000](#)) verbal memory abilities ([Sawrie et al., 2000](#); [Hanoglu et al., 2004](#); [Mantoan et al., 2009](#)), as well as being able to predict verbal memory outcome ([Hanoglu et al., 2004](#)).

### Diffusion Tensor Imaging

Diffusion tensor imaging (DTI) tractography is used in measuring the health and integrity of the white matter tracts in the brain by measuring the degree and direction of water diffusion. Damage to white matter tracts evident on DTI has been shown to correlate with lateralized cortical dysfunction in patients with epilepsy. In addition, some studies have demonstrated an association between DTI measures and cognitive functioning in TLE patients ([Diehl et al., 2006](#); [Powell et al., 2007](#)), though its utility to predict postoperative outcome is still under investigation. Other DTI research has yielded promising results in predicting postoperative language deficits as well as potential neural reorganization of language functioning in temporal lobectomy patients ([Yogarajah et al., 2010](#)).

### Intracarotid Amobarbital Procedure

The IAP, or Wada test, was originally designed to determine language lateralization in epilepsy surgical candidates ([Wada, 1949](#)). Later, a memory paradigm was added in an effort to protect against postoperative amnesia ([Milner, Branch, & Rassmussen, 1962](#)). The procedure entails injecting a fast-acting barbiturate directly into the internal carotid artery, which fills the anterior cerebral artery (ACA), anterior choroidal artery, and middle cerebral artery (MCA) and consequently anesthetizes the anterior one third of the hippocampus and essential lateral perisylvian language areas.

It was held as “the gold standard” for language lateralization for over 50 years and was thought to be essential for predicting postoperative memory outcome in patients with epilepsy. However, recent literature over the past

several years has demonstrated that the Wada test is not essential in all ATL cases as was once thought. [Sabsevitz et al. \(2003\)](#) examined the ability of language laterality as determined by the IAP and a functional MRI (fMRI) semantic decision task to predict postoperative naming abilities following temporal lobe resection. The fMRI task proved to be a stronger, noninvasive predictor of postoperative naming decline, with fMRI laterality indices showing stronger correlations with BNT decline than Wada language laterality ([Sabsevitz et al., 2003](#)). Interestingly, this was the first study to ever examine language laterality as determined by the IAP and its ability to predict language outcome.

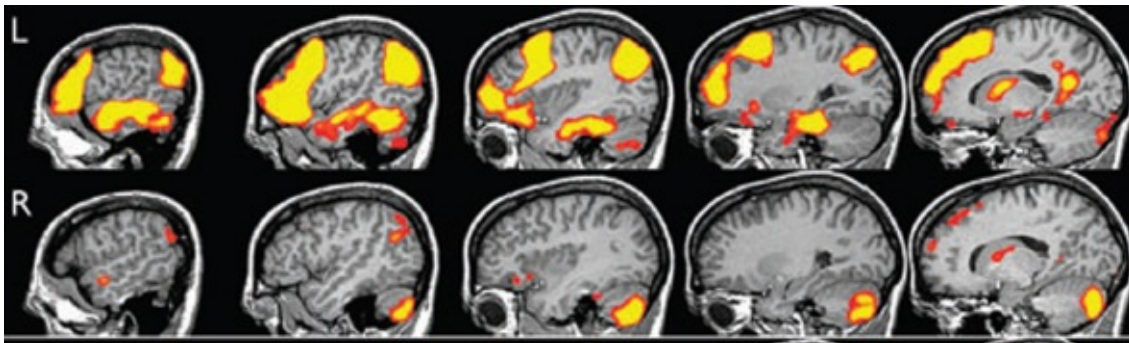
The utility of once “gold standard” IAP memory scores and IAP memory asymmetry to predict postoperative memory decline, specifically verbal memory decline, also has become a highly debated topic in epilepsy surgery. Some studies have demonstrated that IAP memory scores can be useful to predict postoperative verbal memory outcome ([Kneebone et al., 2007](#); [Loring et al., 1995](#); [Chiaravalloti & Glosser, 2001](#); [Sabsevitz et al., 2001](#)); whereas other studies have shown IAP memory is not a strong predictor of outcome, particularly in the context of other preoperative variables ([Binder et al., 2008](#); [Kirsch et al., 2005](#); [Stroup et al., 2003](#)). In light of these results and advances in functional neuroimaging, many centers are moving to fMRI to determine language lateralization, and there has also been research indicating fMRI as a predictor for verbal memory outcome as well ([Binder et al., 2008](#)).

### fMRI of Language and Memory

Multiple fMRI tasks to activate the hippocampus have been developed to assist with the lateralization of seizure focus, assess the functioning of the hippocampi, and predict postoperative memory outcome. In a relatively small sample of LTL epilepsy patients with left hippocampal sclerosis, [Richardson et al. \(2003\)](#) showed that greater functional activation in the left hippocampus elicited by a verbal encoding task, compared with activation in the right hippocampus, was the strongest independent predictor of verbal memory decline. In a much larger sample, [Bonelli et al. \(2010\)](#) showed that greater activation with a verbal list-learning task in patients with left TLE was related to higher preoperative verbal memory performance and predicted postoperative verbal memory decline. This study also examined a sample of right TLE surgical candidates and showed that using a face-encoding task activation of the right hippocampus was related to better nonverbal memory on a design memory task and also predicted postoperative nonverbal memory decline.

Though these results potentially show promise for using hippocampal activation paradigms for predicting memory outcome, other studies have shown that a semantic decision language paradigm (Binder et al., 1996) is the better predictor of memory outcome when compared to a hippocampal activation, scene-encoding paradigm (Binder et al., 2010). This same semantic-decision/tone decision contrast task (Binder et al., 1996, 1997) has shown high correlations with IAP language lateralization (Binder et al., 1996; Sabsevitz et al., 2003), has been able to predict postoperative language decline (Sabsevitz et al., 2003), and recently, this language paradigm has been shown to predict verbal memory decline over Wada memory results (Binder et al., 2008; see Figure 11.1). These research studies explain why fMRI has become an essential tool at more centers over the years to assist with evaluating preoperative cognitive abilities and predict postoperative cognitive outcome.

### Intracranial Subdural Electrode Recordings and Electrical Stimulation Mapping

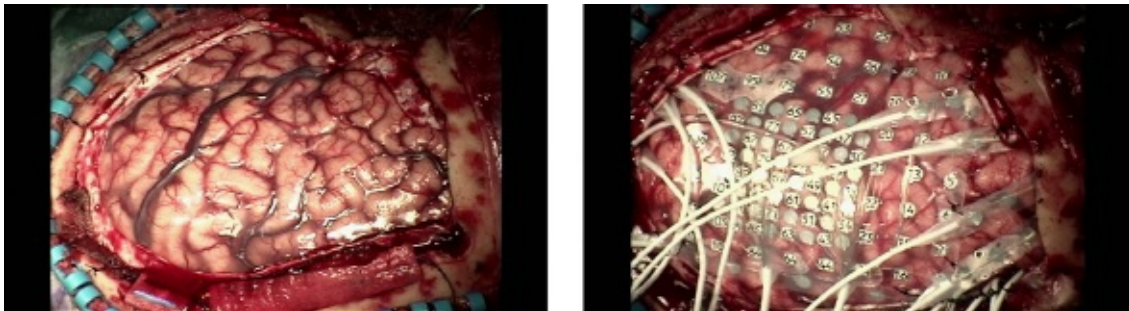


**FIGURE 11.1** Figures show average activation patterns for 30 healthy controls using the semantic-decision, tone-decision task.

*Source:* From Binder et al. (1997). Used with permission.

After an extensive preoperative work-up utilizing the majority of neurodiagnostic techniques mentioned in this chapter, if there is still uncertainty as to the localization of the epileptic focus, then more invasive EEG monitoring is conducted using intracranial subdural grids, strips, and depth electrodes. During this procedure, the neurosurgeon performs a craniotomy and places the array of grid and strip electrodes under the dura throughout the lateral surface of the brain in an effort to more precisely record the zone of seizure onset (Figure 11.2). Subdural strips and grids can also be placed interhemispherically as well

as along the basal temporal lobes. Depth electrodes may also be placed in areas of suspected focus, most commonly in the hippocampus. After placement of all electrodes, the patient is taken back to the monitoring unit for recording of seizure activity. After the localization of seizures is identified with the intracranial monitoring (usually within 4–7 days), the next step (depending on the location of the seizures and prospected resection site) is to map eloquent cortex through electrical stimulation mapping (ESM). Advanced neuroimaging techniques like the ones used at Yale School of Medicine Comprehensive Epilepsy Center enable clear coregistration of subdural grid placements on a 3D image of the brain for better visualization of grid contact location. This facilitates identifying localization of epileptogenic focus as well as more efficient cortical stimulation mapping (see [Figure 11.3](#)).



**FIGURE 11.2** These two figures show a left temporal craniotomy for the placement of subdural grid and strip electrodes. Used with permission.

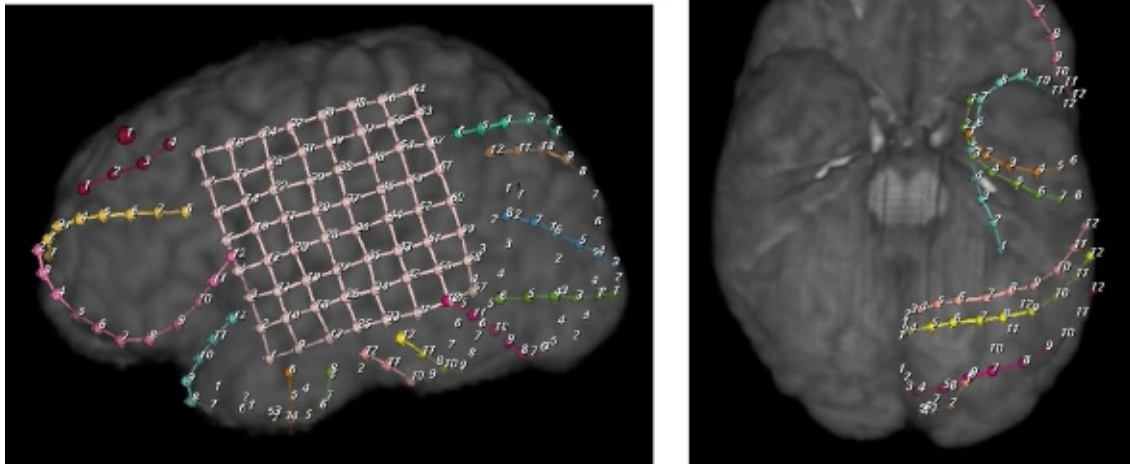
ESM for years has been the gold standard for identifying essential sensory and motor cortex as well as cortical language areas in patients undergoing dominant hemisphere resection ([Ojemann, 1983](#); [Penfield, 1959](#)). The following lists the stimulation parameters and language-mapping protocol at the Yale School of Medicine:

*ESM Stimulation Parameters.* Extraoperative ESM used at Yale entails using a GRASS Instruments stimulator, which delivers a biphasic square wave 60 Hz pulse train with a pulse width of 500  $\mu$ s. Intracranial EEG is continuously monitored to observe for after-discharges or seizures. Maximum stimulation duration for each language task trial is 5 seconds. Stimulation is delivered using pairs of adjacent electrodes. At each electrode pair tested, the stimulation intensity is raised in steps of 2 mA until the maximum current of 10 mA was attained, or after-discharges were noted on the EEG. Stimulation onset was

timed to start before presentation of the language cue. If no response was elicited in 5 seconds, the electrical stimulation was terminated. If language task disruption was noted during stimulation of a contact pair, this was confirmed by additional trials. A site is considered essential for language if a stimulus-related dysfunction was noted on two of three trials in the absence of local or remote after-discharges. Each site receives a minimum of two trials for each language condition.

*Standard ESM Language Tasks.* Our standard battery of language tests during ESM includes the following: For expressive speech, *Counting*: The patient is asked to count from one to ten to screen for disruption of speech production. *Prose Recitation*: The patient is asked to recite the “pledge of allegiance” or alternative prose to screen for disruption of language. The following tests are administered only at sites where speech arrest was not observed during prose recitation and counting. *Reading*: To identify cortical areas essential for reading aloud (orthographic to phonological translation), the patient is given simple paragraphs to read during stimulation. *Visual Naming*: The patient is shown line drawings from the Snodgrass and Vanderwart sample (1980) and asked to name these aloud to test visual common noun (VC) naming. *Auditory Naming*: Stimuli for auditory common noun (AC) naming were chosen from a published set for which normative data have been established ([Hammeke et al., 2005](#)). Finally, *Speech Comprehension* is tested by asking the patient either to follow several one-step commands or to repeat a phrase presented during stimulation. Comprehension is demonstrated if the patient is able to name the auditory definition. For sites in which auditory naming is impaired, these tests are implemented to distinguish between deficits involving name retrieval and impairments due to inability to comprehend the auditory definition cue.





**FIGURE 11.3** These two figures show the extensive coverage with subdural grid and strip electrodes, which not only record seizure activity but also are then used for extraoperative cortical stimulation mapping of eloquent language and sensorimotor cortex.

*Source:* Courtesy of Dennis Spencer, Ken Vives, and Biolmage Suite, Yale School of Medicine.

Early studies suggested that the proximity of surgical resection to sites identified by ESM was related to postoperative language morbidity (Haglund, Berger, Shamseldin, Lettich, & Ojemann, 1994; Ojemann, 1979; Ojemann & Dodrill, 1981). In centers that perform ESM-tailored resections, neurosurgeons typically use a 1-cm boundary between identified language sites and tissue to be resected (Haglund et al., 1994; Hermann et al., 1999). For years, epilepsy centers relied on a visual picture-naming test to identify essential language sites during mapping, as well as a behavioral neuropsychological test. However, several studies have demonstrated that an auditory definition naming task (e.g., “What a king wears on his head”) may be better suited to map language representation in the temporal lobe (Bell et al., 2003; Hamberger & Seidel, 2003; Hamberger & Tamny, 1999). Evidence suggests that this task is better correlated with self-reported word-finding difficulties (Hamberger & Seidel, 2003), identifies more anterior temporal language areas during ESM (Hamberger, Goodman, Perrine, & Tamny, 2001; Hamberger et al., 2005) and predicts naming outcome after surgery (Hamberger et al., 2005). Other preliminary evidence suggests the potential utility of using proper noun stimuli (e.g., auditory and visual stimuli of famous faces and famous landmarks) may identify additional language sites not elicited by traditional common noun auditory and visual naming stimuli as well

as predict postoperative language outcome ([Winstanley et al., 2008](#)). The nature of surgical resection itself, even with respecting certain positive language sites, can also play a role in whether or not a patient experiences decline. For example, whether or not postoperative naming decline is observed following removing basal temporal lobe language areas remains a debated topic in the field ([Lüders et al., 1986](#); [Krauss et al., 1996](#)). More recently, whether or not the hippocampus is removed during temporal lobectomy has shown different postoperative naming results ([Hamberger et al., 2010](#)).

## Surgical Approaches

Surgical approaches for temporal lobe resection vary from surgeon to surgeon and from center to center. Some centers use a traditional “en bloc” resection, in which the extent of tissue to be resected is predetermined prior to surgery. Other centers perform more tailored resections based on intraoperative electrocorticography (ECoG) and results of cortical stimulation mapping of eloquent cortex. The nature and extent of resection can also depend on other preoperative variables mentioned previously (e.g., structural integrity of hippocampus, preoperative neuropsychological testing, and Wada memory) ([Spencer & Ojemann, 1993](#)). A typical resection in the language-dominant hemisphere is about 3 to 5 cm from the temporal pole and includes the lateral middle and inferior gyrus of the temporal neocortex (anterior portion of superior temporal gyrus is sometimes resected but the majority of superior temporal gyrus is preserved). Afterward, mesial temporal lobe structures (e.g., amygdala, hippocampus, and parahippocampal gyrus) are resected. Nondominant temporal resections can extend 4 to 6 cm from the temporal pole.

Though anterior temporal lobectomy is the most common approach in temporal lobe epilepsy resections, a standard temporal lobectomy is not always possible. A standard ATL is not performed if there are preoperative variables that would predict potentially negative outcome from a cognitive perspective (e.g., positive language sites in the proposed surgical resection zone indicated from cortical stimulation mapping of language; high preoperative neuropsychological testing). In such cases, alternative procedures can be used. Such alternate procedures include MST, selective amygdalohippocampectomy, and lesionectomy. Cognitive morbidity has been associated with each of these procedures, though no procedure has received as much research as temporal lobe resections. One study compared left TLE patients who underwent either a standard ATL, selective amygdalohippocampectomy, or lesionectomy and their

pre-and postoperative performance on verbal memory measures (Helmstaedter, Hermann, Lassonde, Kahane, & Arzimanoglou, in press). The results showed that all three groups showed similarly impaired deficits on verbal memory measures compared to controls on preoperative testing. However, left ATL and amygdalohippocampectomy patients showed long-term memory deficits postoperatively, but only the left ATL group showed deficits in verbal learning abilities.

The left cortical lesionectomy patients showed relatively minimal pre-to postchange in verbal learning and memory abilities.

### Cognitive/Neurobehavioral Morbidity Following Standard Anterior Temporal Lobectomy

Many cognitive and neurobehavioral changes can occur after anterior temporal lobectomy. As mentioned, verbal memory decline is a common finding in L-ATL and has been shown to occur in upwards of 30% to 60% of L-ATL cases. Postoperative language deficits, specifically naming deficits, are also a common finding in L-ATL and occur in about 25% to 60% of cases. Another common residual artifact of an anterior temporal lobectomy is contralateral superior quadrantanopia (about 50% of ATL cases), resulting from encroachment on the Meyer's loop. However, these visual deficits do not tend to significantly affect the individual in everyday life. The deficits of most concern are poor daily memory and word-finding difficulties.

Not all patients decline to the same nature and extent as others, and as mentioned earlier, there are a significant amount of preoperative predictor variables that enable us to identify those at risk for postoperative cognitive morbidity. However, recent studies in structural and functional neuroimaging have begun to offer evidence into postoperative neural reorganization patterns, which may also offer insight into the nature and extent of postoperative cognitive morbidity and explain variance in postoperative cognitive morbidity in surgery patients.

### **Neural Plasticity, Postsurgical Structural/Functional Reorganization, and Cognitive Rehabilitation**

#### Neural Plasticity and Functional Reorganization (Preoperatively)

The majority of what we know about the brain's ability to reorganize cognitive functioning, more specifically language abilities, stems from research of patients

with epilepsy. Much of what has been learned about this population and their atypical language networks and the brain's ability to reorganize in general has come from invasive language-mapping techniques mentioned previously (e.g., IAP and ESM), as well as more highly advanced noninvasive techniques such as fMRI. It has been repeatedly demonstrated that patients with epileptogenic tissue arising from the left hemisphere, particularly those with early-onset left hemisphere epilepsy, have higher rates of atypical language representation. Studies have shown evidence of both intrahemispheric reorganization of language functions, as well as interhemispheric reorganization to the contralateral right hemisphere. This shift in language from the left to right hemisphere can result in the hindrance of developing nonverbal and visual spatial abilities, known as the "crowding effect." Other examples of neural reorganization can be found in research examining language sites identified with ESM in patients with or without hippocampal sclerosis. Recently it has been demonstrated that patients with more posterior language areas being involved in patients with the syndrome of mesial temporal sclerosis (MTS) ([Hamberger et al., 2007](#)).

Other studies have shown preoperative reorganization of verbal and nonverbal memory abilities due to unilateral MTS ([Powell et al., 2007](#)).

### Neural Plasticity and Functional Reorganization (Postoperatively)

Postoperative neural reorganization in the adult brain as a result of essential memory and language structures being removed remains a controversial area of research. However, there have been some reports that look potentially promising for postsurgical neural reorganization in the postsurgical epileptic brain. A number of studies over the years have purported to offer evidence of functional reorganization of the adult brain following temporal lobe resection. Though these studies offer hope for new gains in the field, they are not without their limitations and deserve review for the purposes of this chapter.

### Postoperative Language Reorganization

[Backes \(2005\)](#) looked at RTL and LTL resection patients, all of whom were left language dominant as determined by IAP, who underwent multiple postoperative fMRI language paradigms, and were compared to activation patterns of controls. The results showed discordance between IAP dominance and decreased activation in the LTL group. Their activation patterns were compared to controls. Their results showed only LTL patients showed a decrease in activation for a

word-generation task, compared to controls, as well as greater bilateral activation compared to the RTL group. However, no preoperative fMRI was examined, and therefore, a true postoperative “reorganization” cannot be implied. Indeed, left TLE patients show a higher degree of bilateral language patterns than individuals with right TLE. In another fMRI study, [Cheung et al. \(2009\)](#) claimed that postoperative memory performance was related to functional activation on the contralateral side of resection in patients who had unilateral resection. They looked at both left TLE and right TLE patients in this study. [Patariaia et al. \(2005\)](#) demonstrated reorganization of receptive language abilities with their MEG tasks. Interhemispheric, language specific, reorganization in patients who underwent L-ATL was shown to have taken place even when the resection did not directly encroach on Wernicke’s area.

### Postoperative Memory Reorganization

[Cheung et al. \(2009\)](#) examined a series of TLE patients (9 LTLE; 8 RTLE) who underwent unilateral temporal lobe resection. The results of their study showed that L-ATL patients showed postoperative verbal memory decline. Interestingly, both their postoperative verbal and nonverbal memory scores were positively correlated to fMRI activation patterns in the contralateral, right mesial temporal lobe. The same pattern was observed for the right ATL patients with memory scores being positively correlated with contralateral mesial temporal lobe structures. The study is not without its limitations (e.g., small sample size, no preoperative fMRI data) but at least provides preliminary evidence for the potential of reorganization of memory functioning postoperatively. Much further research in this area is required before any theoretical conclusions can be drawn regarding postoperative reorganization of memory abilities in anterior temporal lobectomy patients.

### Postoperative Structural Reorganization

There have also been multiple DTI studies that have examined functional reorganization of cognitive abilities following surgery. [Schoene-Bake et al. \(2009\)](#) showed that left temporal resection patients showed more widespread affections ipsilateral and also fractional anisotropy (FA) decrease in the contralateral inferior longitudinal fasciculus with their DTI study.

[Concha et al. \(2007\)](#) claimed to have shown that irreversibility of DTI white matter abnormalities in seizure-free patients suggested structural changes and not functional changes were occurring in the white matter. Another study

claiming to support postoperative structural reorganization in temporal lobectomy patients was by [Yogarajah et al. \(2010\)](#). In a study of 26 LTL and 20 RTL epilepsy patients, this study showed that there was an increase in postoperative fractional anisotropy in the ventro-medial LTL language networks, indicating structural reorganization following anterior temporal lobe surgery. They argued that the decrease in language scores was a result of this structural reorganization secondary to damaging the dorsolateral language pathway. They indicated that DTI was a useful tool to predict postoperative language outcome.

## Postsurgical Cognitive Rehabilitation

The utility of cognitive rehabilitation techniques to improve cognitive functioning in postneurosurgical patients, particularly ATL patients, is underrepresented in the literature. Cognitive remediation and rehabilitation in patients with epilepsy has met with some controversy and the efficacy of cognitive rehabilitation with this particular population has not been consistently demonstrated. At the present time, there is no specific empirically validated protocol for cognitive rehabilitation for individuals who have undergone neurosurgical intervention (including ATL patients), either in the acute, or long-term stages. Only one study, [Helmstaedter et al. \(2008\)](#), has examined a standard protocol on postoperative cognitive rehabilitation in temporal lobe epilepsy patients.

## Cognitive Rehabilitation and Outcome Through Behavioral Retraining

[Helmstaedter et al. \(2008\)](#) examined the efficacy of cognitive rehabilitation in rehabilitating memory functions in people who have undergone temporal lobe resection. The rehabilitation program performed on 55 patients versus 57 controls included the following:

*I. Metacognitive Neuropsychological Group Therapy: (Five, 1-hour sessions)* Included psychoeducation of the effects of brain functioning and cognitive deficits; learning compensatory strategies to cope with cognitive deficits; and cognitive exercises for attention, problem solving, and mnemonic techniques to facilitate memory.

*II. Cognitive Exercises (4–5 sessions per week):* Focused on the cognitive abilities of attention, memory, and executive functions. Six exercises (two for visually sustained attention, two for verbal memory, two for nonverbal memory) were used repetitively for each patient catering to each of her deficits. Once an individual reached 90% accuracy on a given task, she progressed to the next

level of difficulty. The authors note that the same list-learning tests that were used pre-and postoperatively were not used during the cognitive remediation training.

*III. Three to 4 sessions per week of occupational therapy* that also included exercises for memory functions, attention, and problem solving, which was aimed at enhancing these cognitive abilities as they apply in the workplace. Individual counseling and “sociotherapy” that included social activities was also offered during this time period.

Postoperatively, the authors note that approximately 78% of the patients were seizure free without any seizures or auras following the surgery. There were no significant differences in seizure outcome with the matched groups. Seizure outcome did not affect the changes in cognitive performance on tests of verbal memory, figural memory, or attention ([Helmstaedter et al., 2008](#)). Overall, the authors reported modest results in the cognitive domains that underwent rehabilitation. Attention improved modestly independent of the cognitive rehabilitation program. Limitations that the authors point out, which are significant, include lack of randomization and preoperative differences of baseline cognitive abilities before the surgery, as well as the short time period (3 months postsurgery) of the postoperative assessment. Patients in both groups (rehab and nonrehab) were likely to still improve in cognitive functioning as time passed, which is why the majority of epilepsy centers conduct the initial postoperative assessments at 6 and 12 months postsurgery. The authors reported that cognitive retraining enhanced verbal memory outcome; however, the verbal memory improvement was seen in LTL epilepsy patients with verbal memory problems. Patients with left TLE and left temporal resection, which has substantially higher proportions of verbal memory problems versus right TLE and RTL resection patients, did not appear to improve significantly after surgery.

### Cognitive Rehabilitation/Improvement Through Pharmacological Therapy

Research has demonstrated that pharmacological therapy has been beneficial to cognitive disorders in a variety of neurologic disorders, particularly Alzheimer’s disease (AD). However, in a well-controlled randomized cross-over study with epilepsy patients with subjective memory problems, a 3-month trial of donepezil did not yield any significant difference between the placebo group and the donepezil group on memory testing ([Hamberger et al., 2007](#)). Thus, it is unlikely that these medications would yield positive results in improving memory abilities in postoperative temporal lobectomy patients.

## SUMMARY

This chapter provided a thorough and informative examination of the comprehensive presurgical assessment for patients undergoing anterior temporal lobectomy, in addition to describing the anterior temporal lobectomy itself and its associated cognitive morbidity. This chapter also examined research on premorbid neural plasticity of the epileptic brain as well as potential postoperative functional/structural neural reorganization. Finally, postoperative cognitive rehabilitation and pharmacological treatment were examined.

This chapter served to educate the clinician mainly in preoperative predictors of postoperative cognitive abilities (specifically verbal memory and naming), which include side of surgical resection (nondominant better than dominant), age of seizure onset (early better than late), preoperative neuropsychological testing (lower better than higher), and presence or absence of a lesion or MTS (better to have positive imaging results versus no lesion). As mentioned, recently it has been shown that advanced functional imaging, particularly fMRI language paradigms, has been a strong predictor in postoperative outcome.

Individuals with who have undergone surgical resection of essential brain areas are a unique and challenging population in the field of cognitive rehabilitation. It is well documented that transient deficits may occur after surgery (e.g., language and memory), which then improve over the course of several weeks by the natural healing process of the brain itself. However, some of the cognitive morbidity may be permanent in nature. Overall, the present research has not indicated that extensive cognitive retraining and rehabilitation can assist a person in “regaining functioning” after temporal lobectomy. Pharmacological treatment does not appear to offer assistance either. Thus, compensatory mechanisms that cater to the surroundings of the individual may be the route to focus on for cognitive rehabilitation of the individual who has undergone neurosurgical resection, be it of epileptic tissue or secondary to the other types of neurosurgery mentioned. Such up-to-date empirically validated compensatory mechanisms can be found in chapters throughout this book.

## REFERENCES

- Army Individual Test Battery. (1944). *Manual of directions and scoring*. Washington, DC: War Department, Adjutant General's Office.
- Backes, W. H., Deblaere, K., Vonck, K., Kessels, A. G., Boon, P., Hofman, P.,... Aldenkamp, A. P. (2005). Language activation distributions revealed by fMRI in postoperative epilepsy patients: Differences between left-and right-sided resections. *Epilepsy Research*, 66(1-3), 1-12.



- Baxendale, S., Thompson, P., Harkness, W., & Duncan, J. (2006). Predicting memory decline following epilepsy surgery: A multivariate approach. *Epilepsia*, *47*(11), 1887–1894.
- Baxendale, S., Thompson, P., Harkness, W., & Duncan, J. (2007). The role of the intracarotid amobarbital procedure in predicting verbal memory decline after temporal lobe resection. *Epilepsia*, *48*(3), 546–552.
- Beck, A. (1993). *Beck Anxiety Inventory*. San Antonio, TX: Psychological Corporation.
- Beck, A., Steer, R., & Brown, G. (1996). *Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.
- Bell, B. D., Davies, K. G., Hermann, B. P., & Walters, G. (2000). Confrontation naming after anterior temporal lobectomy is related to age of acquisition of the object names. *Neuropsychologia*, *38*(1), 83–92.
- Benton, A. L. (1994). Neuropsychological assessment. *Annual Review of Psychology*, *45*, 1–23.
- Benton, A., Hannay, H. J., & Varney, N. R. (1975). Visual perception of line direction in patients with unilateral brain disease. *Neurology*, *25*(10), 907–910.
- Binder, J. R., Frost, J. A., Hammeke, T. A., Cox, R. W., Rao, S. M., & Prieto, T. (1997). Human brain language areas identified by functional magnetic resonance imaging. *Journal of Neuroscience*, *17*(1), 353–362.
- Binder, J. R., Sabsevitz, D. S., Swanson, S. J., Hammeke, T. A., Raghavan, M., & Mueller, W. M. (2008). Use of preoperative functional MRI to predict verbal memory decline after temporal lobe epilepsy surgery. *Epilepsia*, *49*(8), 1377–1394.
- Binder, J. R., Swanson, S. J., Hammeke, T. A., Morris, G. L., Mueller, W. M., Fischer, M., . . . Houghton, V. M. (1996). Determination of language dominance using functional MRI: A comparison with the Wada test. *Neurology*, *46*(4), 978–984.
- Binder, J. R., Swanson, S. J., Sabsevitz, D. S., Hammeke, T. A., Raghavan, M., & Mueller, W. M. (2010). A comparison of two fMRI methods for predicting verbal memory decline after left temporal lobectomy: Language lateralization versus hippocampal activation asymmetry. *Epilepsia*, *51*(4), 618–626.
- Bonelli, S. B., Powell, R. H., Yogarajah, M., Samson, R. S., Symms, M. R., Thompson, P. J., . . . Duncan, J. S. (2010). Imaging memory in temporal lobe epilepsy: Predicting the effects of temporal lobe resection. *Brain*, *133*(Pt 4), 1186–1199.
- Brown, F. C., Tuttle, E., Westerveld, M., Ferraro, F. R., Chmielowiec, T., Vandemore, M., Gibson-Beverly, G., . . . Spencer, S. S. (2010). Visual memory in patients after anterior right temporal lobectomy and adult normative data for the Brown Location Test. *Epilepsy & Behavior*, *17*(2), 215–220.
- Busch, R. M., Frazier, T. W., Haggerty, K. A., & Kubu, C. S. (2005). Utility of the Boston naming test in predicting ultimate side of surgery in patients with medically intractable temporal lobe epilepsy. *Epilepsia*, *46*(11), 1773–1779.
- Busch, R. M., Frazier, T. W., Iampietro, M. C., Chapin, J. S., & Kubu, C. S. (2009). Clinical utility of the Boston Naming Test in predicting ultimate side of surgery in patients with medically intractable temporal lobe epilepsy: A double cross-validation study. *Epilepsia*, *50*(5), 1270–1273.
- Buschke, H. (1973). Selective reminding for analysis of memory and learning. *Journal of Verbal Learning and Verbal Behavior*, *12*, 543–550.
- Butcher, J., Dahlstorm, W., Grahram, J., Tellegen, A., & Kaemmer, B. (1989). *Minnesota Multiphasic Personality Inventory-2 (MMPI-2)*. San Antonio, TX: The Psychological Corporation.
- Chelune, G. J., Naugle, R. I., Lüders, H., & Awad, I. A. (1991). Prediction of cognitive change as a function of preoperative ability status among temporal lobectomy patients seen at 6-month follow-up. *Neurology*, *41*(3), 399–404.
- Cheung, M. C., Chan, A. S., Lam, J. M., & Chan, Y. L. (2009). Pre-and postoperative fMRI and clinical memory performance in temporal lobe epilepsy. *Journal of Neurology, Neurosurgery, and Psychiatry*, *80*(10), 1099–1106.
- Chiaravalloti, N. D., & Glosser, G. (2001). Material specific memory changes after anterior temporal lobectomy as predicted by the intracarotid amobarbital test. *Epilepsia*, *42*: 902–911.

- Concha, L., Beaulieu, C., Wheatley, B. M., & Gross, D. W. (2007). Bilateral white matter diffusion changes persist after epilepsy surgery. *Epilepsia*, *48*(5), 931–940.
- Conners, C. (2001). *Conners' continuous performance test-II*. Toronto, ON: Multi Health Systems.
- Dabbs, K., Jones, J., Seidenberg, M., & Hermann, B. (2009). Neuroanatomical correlates of cognitive phenotypes in temporal lobe epilepsy. *Epilepsy & Behavior*, *15*(4), 445–451.
- Davies, K. G., Bell, B. D., Bush, A. J., Hermann, B. P., Dohan, F. C., & Jaap, A. S. (1998). Naming decline after left anterior temporal lobectomy correlates with pathological status of resected hippocampus. *Epilepsia*, *39*(4), 407–419.
- Delis, D., Kramer, J., Kaplan, E., & Ober, B. (2000). *The California Verbal Learning Test-second edition (CVLT-II)*. San Antonio, TX: Psychological Corporation.
- Diehl, B., Busch, R. M., Duncan, J. S., Piao, Z., Tkach, J., & Lüders, H. O. (2008). Abnormalities in diffusion tensor imaging of the uncinate fasciculus relate to reduced memory in temporal lobe epilepsy. *Epilepsia*, *49*(8), 1409–1418.
- Gleissner, U., Helmstaedter, C., Schramm, J., & Elger, C. E. (2004). Memory outcome after selective amygdalohippocampectomy in patients with temporal lobe epilepsy: One-year follow-up. *Epilepsia*, *45*(8), 960–962.
- Griffith, H. R., Perlman, S. B., Woodard, A. R., Rutecki, P. A., Jones, J. C., Ramirez, L. F., . . . Hermann, B. P. (2000). Preoperative FDG-PET temporal lobe hypometabolism and verbal memory after temporal lobectomy. *Neurology*, *54*(5), 1161–1165.
- Haglund, M. M., Berger, M. S., Shamseldin, M., Lettich, E., & Ojemann, G. A. (1994). Cortical localization of temporal lobe language sites in patients with gliomas. *Neurosurgery*, *34*(4), 567–76; discussion 576.
- Hamberger, M. J., Goodman, R. R., Perrine, K., & Tamny, T. (2001). Anatomic dissociation of auditory and visual naming in the lateral temporal cortex. *Neurology*, *56*(1), 56–61.
- Hamberger, M. J., Seidel, W. T., Goodman, R. R., Perrine, K., & McKhann, G. M. (2003). Temporal lobe stimulation reveals anatomic distinction between auditory naming processes. *Neurology*, *60*(9), 1478–1483.
- Hamberger, M. J., Seidel, W. T., Goodman, R. R., Williams, A., Perrine, K., Devinsky, O., & McKhann, G. M. (2007). Evidence for cortical reorganization of language in patients with hippocampal sclerosis. *Brain*, *130*(Pt 11), 2942–2950.
- Hamberger, M. J., Seidel, W. T., McKhann, G. M., & Goodman, R. R. (2010). Hippocampal removal affects visual but not auditory naming. *Neurology*, *74*(19), 1488–1493.
- Hamberger, M. J., Seidel, W. T., McKhann, G. M., Perrine, K., & Goodman, R. R. (2005). Brain stimulation reveals critical auditory naming cortex. *Brain*, *128*(Pt 11), 2742–2749.
- Hamberger, M. J., & Seidel, W. T. (2003). Auditory and visual naming tests: Normative and patient data for accuracy, response time, and tip-of-the-tongue. *Journal of the International Neuropsychological Society*, *9*(3), 479–489.
- Hamberger, M. J., & Tamny, T. R. (1999). Auditory naming and temporal lobe epilepsy. *Epilepsy Research*, *35*(3), 229–243.
- Hammeke, T. A., Kortenkamp, S. J., & Binder, J. R. (2005). Normative data on 372 stimuli for descriptive naming. *Epilepsy Research*, *66*(1–3), 45–57.
- Hanoglu, L., Ozkara, C., Keskinliç, C., Altin, U., Uzan, M., Tuzgen, S., . . . Ozyurt, E. (2004). Correlation between 1H MRS and memory before and after surgery in mesial temporal lobe epilepsy with hippocampal sclerosis. *Epilepsia*, *45*(6), 632–640.
- Helmstaedter, C., & Elger, C. E. (1996). Cognitive consequences of two-thirds anterior temporal lobectomy on verbal memory in 144 patients: A three-month follow-up study. *Epilepsia*, *37*(2), 171–180.
- Helmstaedter, C., Hermann, B., Lassonde, M., Kahane, P., & Arzimanoglou, A. (Eds.). (in press). *Neuropsychology and its role in the care of people with epilepsy* France: John Libby Eurotext.
- Helmstaedter, C., Loer, B., Wohlfahrt, R., Hammen, A., Saar, J., Steinhoff, B. J., . . . Schulze-Bonhage, A. (2008). The effects of cognitive rehabilitation on memory outcome after temporal lobe epilepsy surgery. *Epilepsy & Behavior*, *12*(3), 402–409.

- Hermann, B. P., Gold, J., Pusakulich, R., Wyler, A. R., Randolph, C., Rankin, G., & Hoy, W. (1995). Wechsler adult intelligence scale-revised in the evaluation of anterior temporal lobectomy candidates. *Epilepsia*, 36(5), 480–487.
- Hermann, B. P., Perrine, K., Chelune, G. J., Barr, W., Loring, D. W., Strauss, E., . . . Westerveld, M. (1999). Visual confrontation naming following left anterior temporal lobectomy: A comparison of surgical approaches. *Neuropsychology*, 13(1), 3–9.
- Hermann, B. P., Seidenberg, M., Haltiner, A., & Wyler, A. R. (1995). Relationship of age at onset, chronologic age, and adequacy of preoperative performance to verbal memory change after anterior temporal lobectomy. *Epilepsia*, 36(2), 137–145.
- Hermann, B. P., Wyler, A. R., Somes, G., & Clement, L. (1994). Dysnomia after left anterior temporal lobectomy without functional mapping: Frequency and correlates. *Neurosurgery*, 35(1), 52–6; discussion 56.
- Hermann, B. P., Wyler, A. R., Somes, G., Berry, A. D., & Dohan, F. C. (1992). Pathological status of the mesial temporal lobe predicts memory outcome from left anterior temporal lobectomy. *Neurosurgery*, 31(4), 652–6; discussion 656.
- Jokeit, H., Ebner, A., Holthausen, H., Markowitsch, H. J., Moch, A., Pannek, H., . . . Tuxhorn, I. (1997). Individual prediction of change in delayed recall of prose passages after left-sided anterior temporal lobectomy. *Neurology*, 49(2), 481–487.
- Jones-Gotman, M., Smith, M. L., Risse, G. L., Westerveld, M., Swanson, S. J., Giovagnoli, A. R., . . . Piazzini, A. (2010). The contribution of neuropsychology to diagnostic assessment in epilepsy. *Epilepsy & Behavior*, 18(1–2), 3–12.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *The Boston naming test*. Philadelphia, PA: Lea & Febiger.
- Keary, T. A., Frazier, T. W., Busch, R. M., Kubu, C. S., & Iampietro, M. (2007). Multivariate neuropsychological prediction of seizure lateralization in temporal epilepsy surgical cases. *Epilepsia*, 48(8), 1438–1446.
- Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., . . . Meyer-Lindenberg, A. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of Neuroscience*, 25(49), 11489–11493.
- Kneebone, A. C., Lee, G. P., Wade, L. T., & Loring, D. W. (2007). Rey Complex Figure: Figural and spatial memory before and after temporal lobectomy for intractable epilepsy. *Journal of the International Neuropsychological Society*, 13(4), 664–671.
- Krauss, G. L., Fisher, R., Plate, C., Hart, J., Uematsu, S., Gordon, B., & Lesser, R. P. (1996). Cognitive effects of resecting basal temporal language areas. *Epilepsia*, 37(5), 476–483.
- Langfitt, J. T., & Rausch, R. (1996). Word-finding deficits persist after left anterotemporal lobectomy. *Archives of Neurology*, 53(1), 72–76.
- Lee, G. P., Westerveld, M., Blackburn, L. B., Park, Y. D., & Loring, D. W. (2005). Prediction of verbal memory decline after epilepsy surgery in children: Effectiveness of Wada memory asymmetries. *Epilepsia*, 46(1), 97–103.
- Lee, T. M. C., Yip, J. T. H., & Jones-Gotman, M. (2002). Memory deficits after resection of left or right anterior temporal lobe in humans: A meta-analytic review. *Epilepsia*, 43, 283–291.
- Leeman, B. A., Leveroni, C. L., & Johnson, K. A. (2009). Does hippocampal FDG-PET asymmetry predict verbal memory dysfunction after left temporal lobectomy? *Epilepsy & Behavior*, 16(2), 274–280.
- Lencz, T., McCarthy, G., Bronen, R. A., Scott, T. M., Insermi, J. A., Sass, K. J., . . . Spencer, D. D. (1992). Quantitative magnetic resonance imaging in temporal lobe epilepsy: Relationship to neuropathology and neuropsychological function. *Annals of Neurology*, 31(6), 629–637.
- Lezak, M., Howieson, D., & Loring, D. (1994). *Neurological assessment* (4th ed.). New York, NY: Oxford University Press.
- Lineweaver, T. T., Morris, H. H., Naugle, R. I., Najm, I. M., Diehl, B., & Bingaman, W. (2006). Evaluating the contributions of state-of-the-art assessment techniques to predicting memory outcome after

- unilateral anterior temporal lobectomy. *Epilepsia*, 47(11), 1895–1903.
- Loring, D. W., Meador, K. J., Lee, G. P., King, D. W., Nichols, M. E., Park, Y. D., . . . Smith, J. R. (1995). Wada memory asymmetries predict verbal memory decline after anterior temporal lobectomy. *Neurology*, 45(7), 1329–1333.
- Loring, D. W., Strauss, E., Hermann, B. P., Barr, W. B., Perrine, K., Trenerry, M. R., . . . Bowden, S. C. (2008). Differential neuropsychological test sensitivity to left temporal lobe epilepsy. *Journal of the International Neuropsychological Society*, 14(3), 394–400.
- Lüders, H., Lesser, R. P., Hahn, J., Dinner, D. S., Morris, H., Resor, S., & Harrison, M. (1986). Basal temporal language area demonstrated by electrical stimulation. *Neurology*, 36(4), 505–510.
- Mantoan, M. A., Caboclo, L. O., de Figueiredo Ferreira Guilhoto, L. M., Lin, K., da Silva Noffs, M. H., de Souza Silva Tudesco, I., . . . Yacubian, E. M. (2009). Correlation between memory, proton magnetic resonance spectroscopy, and interictal epileptiform discharges in temporal lobe epilepsy related to mesial temporal sclerosis. *Epilepsy & Behavior*, 16(3), 447–453.
- Martin, R. C., Sawrie, S. M., Edwards, R., Roth, D. L., Faught, E., Kuzniecky, R. I., . . . Gilliam, F. G. (2000). Investigation of executive function change following anterior temporal lobectomy: Selective normalization of verbal fluency. *Neuropsychology*, 14(4), 501–508.
- Martin, R. C., Sawrie, S., Hugg, J., Gilliam, F., Faught, E., & Kuzniecky, R. (1999). Cognitive correlates of 1H MRSI-detected hippocampal abnormalities in temporal lobe epilepsy. *Neurology*, 53(9), 2052–2058.
- Martin, R. C., Sawrie, S. M., Roth, D. L., Gilliam, F. G., Faught, E., Morawetz, R. B., & Kuzniecky, R. (1998). Individual memory change after anterior temporal lobectomy: A base rate analysis using regression-based outcome methodology. *Epilepsia*, 39(10), 1075–1082.
- McIntosh, A. M., Wilson, S. J., & Berkovic, S. F. (2001). Seizure outcome after temporal lobectomy: Current research practice and findings. *Epilepsia*, 42(10), 1288–1307.
- Milner, B., Branch, C., & Rassmussen, T. (1962). Study of short-term memory after intracarotid injection of sodium amytal. *Transactions of the American Neurological Association*, 87: 224–226.
- Ojemann, G. A. (1979). Individual variability in cortical localization of language. *Journal of Neurosurgery*, 50(2), 164–169.
- Ojemann, G. A. (1983). Brain organization for language from the perspective of electrical stimulation mapping. *Brain Behavior Research*, 6: 189–230.
- Ojemann, G. A., & Dodrill, C. B. (1981). *Predicting postoperative language and memory deficits after dominant hemisphere anterior temporal lobectomy by intraoperative stimulation mapping*. Paper presented at the 50th Annual Meeting of the American Association of Neurological Surgeons, Boston, MA.
- Osterrieth, P. (1944). Le tes de copie d'une figure complexe. *Archives de Psychologie* 30: 206–356 [Trans: Corwin, J., & Bylsma, F.W. (1993). *Clinical Neuropsychologist*, 7: 9–15].
- Patariaia, E., Lindinger, G., Deecke, L., Mayer, D., & Baumgartner, C. (2005). Combined MEG/EEG analysis of the interictal spike complex in mesial temporal lobe epilepsy. *NeuroImage*, 24(3), 607–614.
- Penfield, W. (1959). *Mapping the speech area: Speech and brain mechanisms*. Princeton, NJ: Princeton University Press.
- Piotrowski, C., & Keller, J. (1989). Psychological testing in outpatient mental health facilities: A national study. *Professional Psychology*, 20: 423–425.
- Powell, H. W., Richardson, M. P., Symms, M. R., Boulby, P. A., Thompson, P. J., Duncan, J. S., & Koepp, M. J. (2007). Reorganization of verbal and nonverbal memory in temporal lobe epilepsy due to unilateral hippocampal sclerosis. *Epilepsia*, 48(8), 1512–1525.
- Rausch, R. (1987). Anatomical substrates of interictal memory deficits in temporal lobe epileptics. *International Journal of Neurology*, 21–22, 17–32.
- Rausch, R., & Babb, T. L. (1993). Hippocampal neuron loss and memory scores before and after temporal lobe surgery for epilepsy. *Archives of Neurology*, 50(8), 812–817.
- Richardson, M. P., Strange, B. A., Duncan, J. S., & Dolan, R. J. (2003). Preserved verbal memory function in left medial temporal pathology involves reorganization of function to right medial temporal lobe.

- Neuroimage*, 20, S112–S119.
- Sabsevitz, D. S., Swanson, S. J., Hammeke, T. A., Spanaki, M. V., Possing, E. T., Morris, G. L., . . . Binder, J. R. (2003). Use of preoperative functional neuroimaging to predict language deficits from epilepsy surgery. *Neurology*, 60(11), 1788–1792.
- Sabsevitz, D. S., Swanson, S. J., Morris, G. L., Mueller, W. M., & Seidenberg, M. (2001). Memory outcome after left anterior temporal lobectomy in patients with expected and reversed Wada memory asymmetry scores. *Epilepsia*, 42(11), 1408–1415.
- Saling, M. M., Berkovic, S. F., O’Shea, M. F., Kalnins, R. M., Darby, D. G., & Bladin, P. F. (1993). Lateralization of verbal memory and unilateral hippocampal sclerosis: Evidence of task-specific effects. *Journal of Clinical and Experimental Neuropsychology*, 15(4), 608–618.
- Sass, K. J., Sass, A., Westerveld, M., Lencz, T., Novelly, R. A., . . . Spencer, D. D. (1992). Specificity in the correlation of verbal memory and hippocampal neuron loss: Dissociation of memory, language, and verbal intellectual ability. *Journal of Clinical and Experimental Neuropsychology*, 14(5), 662–672.
- Sawrie, S. M., Martin, R. C., Gilliam, F. G., Faught, R. E., Maton, B., Hugg, J. W., . . . Kuzniecky, R. I. (2000). Visual confrontation naming and hippocampal function: A neural network study using quantitative (1)H magnetic resonance spectroscopy. *Brain*, 123(Pt 4), 770–780.
- Schoene-Bake, J. C., Faber, J., Trautner, P., Kaaden, S., Tittgemeyer, M., Elger, C. E., & Weber, B. (2009). Widespread affections of large fiber tracts in postoperative temporal lobe epilepsy. *NeuroImage*, 46(3), 569–576.
- Seidenberg, M., Geary, E., & Hermann, B. (2005). Investigating temporal lobe contribution to confrontation naming using MRI quantitative volumetrics. *Journal of the International Neuropsychological Society*, 11(4), 358–366.
- Smith, A. (1982). Symbol digit modalities test (SDMT): Manual (revised). Los Angeles, CA: Western Psychological Services.
- Spencer, D. D., & Ojemann, G. A. (1993). Overview of therapeutic procedures. In J. Engel, Jr. (Ed.), *Surgical treatment of the epilepsies* (2nd ed., pp. 455–471). New York, NY: Raven Press.
- Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18(6), 643.
- Stroup, E., Langfitt, J., Berg, M., McDermott, M., Pilcher, W., & Como, P. (2003). Predicting verbal memory decline following anterior temporal lobectomy (ATL). *Neurology*, 60(8), 1266–1273.
- Télliez-Zenteno, J. F., Dhar, R., & Wiebe, S. (2005). Long-term seizure outcomes following epilepsy surgery: A systematic review and meta-analysis. *Brain*, 128(Pt 5), 1188–1198.
- Tranel, D. (2006). Impaired naming of unique landmarks is associated with left temporal polar damage. *Neuropsychology*, 20(1), 1–10.
- Trenerry, M. R., Jack, C. R., Ivnik, R. J., Sharbrough, F. W., Cascino, G. D., Hirschorn, K. A., . . . Meyer, F. B. (1993). MRI hippocampal volumes and memory function before and after temporal lobectomy. *Neurology*, 43(9), 1800–1805.
- Wada, J. (1949). A new method for the determination of the site of the cerebral speech dominance: A preliminary report on the intracarotid injection of sodium amytal in man. *Igaku to Seitbusugaki*, 14, 221–222.
- Wechsler, D. (1997). *Wechsler Memory Scale-third edition*. San Antonio, TX: The Psychological Corporation.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale-fourth edition*. San Antonio, TX: The Psychological Corporation.
- Wiebe, S., Blume, W. T., Girvin, J. P., & Eliasziw, M. (2001). A randomized, controlled trial of surgery for temporal-lobe epilepsy. *New England Journal of Medicine*, 345(5), 311–318.
- Winstanley, F. S., Swanson, S. J., Sabsevitz, D. S., et al. (2008). Category specific naming during electrical stimulation mapping identifies wider cortical language representation and predicts naming outcome. Presented at the 36th Annual Meeting of the International Neuropsychological Society.
- Yogarajah, M., Focke, N. K., Bonelli, S. B., Thompson, P., Vollmar, C., McEvoy, A. W., . . . Duncan, J. S.

- (2010). The structural plasticity of white matter networks following anterior temporal lobe resection. *Brain*, 133(Pt 8), 2348–2364.
- Yucus, C. J., & Tranel, D. (2007). Preserved proper naming following left anterior temporal lobectomy is associated with early age of seizure onset. *Epilepsia*, 48(12), 2241–2252.
- Zola-Morgan, S., & Squire, L. R. (1993). Neuroanatomy of memory. *Annual Review of Neuroscience*, 16, 547–563.

# Neuropsychological Assessment in Rehabilitation

*Jennifer P. Edidin and Scott J. Hunter*

Changes to the brain due to injury, disease, and deviations in neurodevelopment often lead to impairments in cognitive, behavioral, and psychosocial functioning. These impairments may require rehabilitation services that improve functional outcomes. Within this context, doctoral-level psychologists with specialized training in brain–behavior relationships, neuropsychologists, can assist in diagnosis and the development of a treatment plan. The objective of this chapter is to address the role of neuropsychological assessment within child and adult rehabilitation settings.

## THE REHABILITATION SETTING

To understand the role of neuropsychological assessment within the context of the rehabilitation setting, it is first helpful to understand the practice of rehabilitation. Cognitive rehabilitation is the “systematic use of well-defined structured activities designed to improve higher cerebral functioning in a subject with brain injury or to help the individual accommodate for their deficits by teaching methods of compensation” (Laatsch, Thulborn, Krisky, Shobat, & Sweeney, 2004, p. 957). The neuropsychologist is typically part of a larger treatment team comprised of physicians (physiatrists and other rehabilitation specialists), nurses, physical therapists, occupational therapists, and speech therapists, among others.

Through the completion of an evaluation, which may range from a basic

screening to a comprehensive assessment of cognitive and adaptive functioning, the neuropsychologist assists the team in various ways. First, the neuropsychologist is an integral part of the diagnostic process (Wilson, Rous, & Sopena, 2008). By using a set of qualitative and quantitative measures, the neuropsychologist provides information about a patient's strengths and weaknesses both prior to treatment and, in many situations, after treatment has been completed, to track improvement and the need for further support or accommodation. Further, the results may be used to determine a patient's ability to make decisions for herself or himself, as well as whether he or she is able to live independently, manage financial demands, return to work or school, or drive (Wilson et al., 2008).

Second, information gathered from neuropsychological assessment can be used to guide treatment. For example, a stroke patient may present with difficulties such as visual *neglect*, *apraxia*, *aphasia*, and/or memory impairment. Consequently, therapies to address these deficits, such as visual scanning to address visual neglect, cognitive linguistic therapy to address aphasia, gestural training to address apraxia, and visual imagery or external aides to address memory deficits may be recommended (Cicerone et al., 2005). Typically, a developmental model that addresses fundamental cognitive abilities prior to focusing on complex ones is advised. In addition to cognitive deficits, the neuropsychologist assesses emotional and social functioning and facilitates psychosocial supports for patients who require assistance with these areas (Myers, 2009).

Third, over the course of treatment, neuropsychologists evaluate and monitor functioning over time (Wilson et al., 2008). This longer term approach to assessment provides information about the effectiveness of treatment and whether the treatment plan may need to be amended. In children, serial evaluations conducted during the course of treatment can also be used to monitor ongoing improvements in development (Limond & Leeke, 2005). It is recommended that, whenever possible, assessments occur prior to a known potential treatment-related insult (e.g., radiation or neurosurgery) in order to provide a baseline of premorbid functioning that facilitates the tracking of deficits, recovery, and development postintervention.

Fourth, the neuropsychologist provides education to patients and their families. Specifically, the neuropsychologist provides information about the nature of the injury to the patient and his or her family. Further, the neuropsychologist will help the patient and family understand the possible



cognitive, emotional, and behavioral sequelae of the brain injury or disease and discuss the impact the injury or disease may have on daily life and functioning (Sarajuuri & Koskinen, 2006). To help the patient develop appropriate expectations about recovery, it may be appropriate to address both short-and long-term effects of the injury or disease (Sarajuuri & Koskinen, 2006). This often includes a discussion about treatment options and recommendations for accommodation and modification of the environment so that the patient understands possible approaches to improve current functioning, as well as ways to compensate for and cope with impairments (Myers, 2009; Sarajuuri & Koskinen, 2006).

Finally, the rehabilitation neuropsychologist is often involved in research. Neuropsychological assessment may be used to understand the impact of brain injury or disease on cognitive, psychosocial, and adaptive development and functioning. The development of efficacious and effective rehabilitation interventions is another area of research frequently pursued. Alternatively, neuropsychological assessment may be used to examine individual or injury factors that moderate the effectiveness of specific interventions and adaptive outcomes.

## **DEVELOPMENT AND INJURY: HOW CHILDREN DIFFER FROM ADULTS**

Adult and pediatric neuropsychologists have different approaches and emphases to their assessments within the rehabilitation domain. Typically, the principal focus in adult neuropsychology is current brain–behavior relationships and localization of injury, and how skills previously developed may be altered or lost as a result of brain injury or illness. For example, a stroke leading to a brain lesion forming in a particular region of the brain can produce specific cognitive and behavioral impairments. In assessing the patient, the adult neuropsychologist will emphasize what has been lost or altered as a result of the brain insult and how skills no longer fully “in play” may be accommodated or remediated. In contrast, there is typically less emphasis placed on localization when addressing brain–behavior relationships in pediatric neuropsychology. Developing cognitive functions are often more diffused in their localization (particularly in infants and young children) and neural specification emerges over the course of development. This leads to a greater emphasis by the pediatric neuropsychologist on trajectories of skill presentation and identifying potential means of compensation that can encourage reconfiguration or accommodation,

as possible, at the neural and systems levels. Additionally, data gathered from pediatric neuropsychological assessments are specifically analyzed and appreciated within a developmental context. Importantly though, across the life span, neuropsychologists consider and appreciate the environmental context within which the patient lives to allow for a full description of the patterns of strength and weakness that exist for that individual and to promote effective adaptation over time (Bernstein, 2010).

### **Overview of Typical Development**

Whether working with adults or children, we believe that to fully understand the impact of a brain injury on the individual, the pattern of deficits observed must be considered within a developmental framework. There is rapid development of many areas of cognitive and behavioral functioning during early and mid-childhood, which are associated with changes in structural development that unfold across time (Ewing-Cobbs et al., 1994; Williamson, 2010). Specifically, during childhood, total cerebral volume increases due to synaptic proliferation. There are increases in gray matter volume throughout childhood, until a peak in preadolescence; first in the sensorimotor areas and then in higher order association areas, which are responsible for motor planning, organization, regulation, and working memory (Blakemore & Choudhury, 2006; Giedd, 2008). At the same time, there is a steady increase in white matter volume, which continues into adolescence (Blakemore & Choudhury, 2006; Giedd, 2008). These increases are associated with greater connectivity among brain regions, enhanced speed and efficiency, and improved modulation of the timing and synchrony of neuronal firing (Giedd, 2008). These physical changes are associated with improvements in fine-motor functioning, auditory processing, and the transfer of sensory information (Ciccia, Meulenbrock, & Turkstra, 2009). As well, there is a shift in the proportion of gray matter to white matter. During adolescence, synaptic pruning, reorganization, and refinement, as well as myelination of gray matter lead to reductions in overall gray matter volume (Blakemore & Choudhury, 2006; Giedd, 2008). However, there are significant individual differences that play out, at the neural and more global developmental levels, which are important considerations when considering the impact of brain injury both in the immediate and long term.

### **The Impact of Brain Injury on Children and Adolescents**

Until recently, it was believed that children were better able to recover from

brain injury than adults. This belief, known as the Kennard Principle, was named after the physician Margaret Kennard who theorized that functional outcome after a brain injury was negatively correlated with age (Ciccio et al., 2009; Dennis, Barnes, Donnelly, Wilkinson, & Humphreys, 2009; Laatsch et al., 2004). Recent studies, however, have disproved this theory; they indicate that in fact, younger children are often more vulnerable to neural insults and have worse outcomes than adults and teenagers with similar injuries (Butler & Mulhern, 2005; Ylvisaker et al., 2005).

Early brain insult can undermine typical development of basic skills, as well as the subsequent acquisition of more complex skills (Ewing-Cobbs et al., 1994; Sohlberg & Mateer, 2001). As such, early insults can have a cumulative effect on development, with impairments seen across areas of cognition, behavior, emotion, personality, moral reasoning, and social cognition (Anderson et al., 2001; Hanten, Bartha, & Levin, 2000; Harvey & Anderson, 2007; Tranel & Eslinger, 2000). A study by Jacobs, Harvey, and Anderson (2007) found that prenatal injuries are frequently associated with the greatest neurobehavioral deficits across time. The authors hypothesized that because brain regions are not yet fully devoted to specific functions, these early lesions impact multiple areas of functioning and affect a child's ability to effectively acquire information across time (Kolb, 1995; Tranel & Eslinger, 2000; Ylvisaker et al., 2005). In fact, this kind of pervasive impairment is frequently observed in physically abused infants. Still, although many aspects of cognition may be significantly impacted due to early injury, improvement is often seen with development across time. Executive functioning, however, is one cognitive domain particularly impacted by early childhood insults; often, development of executive functions worsens over time (Dennis et al., 1996; Jacobs et al., 2007; Ylvisaker et al., 2005).

In children, traumatic brain injuries (TBIs) are most often due to diffuse axonal injury. This type of injury is associated with the stretching and tearing of axons, which occurs in response to a rapid change in speed or direction when the head and consequently the brain are jolted during an accident or insult (Sohlberg & Mateer, 2001). Still, despite the breadth of axonal injury that may occur, children are less likely to experience a loss of consciousness (Sohlberg & Mateer, 2001). In fact, on first observation, many children appear to be unharmed immediately following the injury; however, as environmental demands increase and skills are expected to develop, parents and children often notice the emergence of increasing impairment. Commonly, many youth do not

notice difficulties until adolescence, when higher order cognitive abilities, such as complex language, emotion recognition, and executive functioning begin to come more directly on-line. Consequently, it is often during adolescence that the effects of early brain injuries become most apparent (Ciccia et al., 2009; Hotz & Chapman, 2009).

It has also been observed that during middle childhood children appear to be at a lower risk for developing cognitive deficits in response to a brain injury (Jacobs et al., 2007). This is seen particularly with regard to the executive functions and has been attributed to the observation that a peak in synaptogenesis occurs at this time leading to likely compensatory responses by the brain (Jacobs et al., 2007). However, the risk of impairment following a brain injury increases again in later childhood and adolescence (Jacobs et al., 2007). It has been noted, as well, that during this time, insults also tend to have a similar impact on functioning as is seen with adults (Jacobs et al., 2007). This likely reflects the fact that much of the brain's development, outside of the frontal regions, has already taken place (Williamson, 2010).

Despite the significant impairments that can result from early insults to the brain, the plasticity of children's and adolescents' brains may also be protective (Johnston, 2009). Depending on the region of the brain that is injured, children and adolescents appear to be better able to compensate for deficits, and the brain itself appears to foster a good degree of that compensation (Sohlberg & Mateer, 2001). Additionally, because of the plasticity of children's brains, cognitive rehabilitation interventions may be more effective and lead to better outcomes when pursued earlier (Ciccia et al., 2009).

### **Differences in Referral Questions and Presenting Problems Across Development**

Although there is some overlap, the types of disorders that adults and children present when referred for a neuropsychological assessment are typically quite different. More specifically, pediatric referrals tend to focus on difficulties with ongoing development and learning and a need for recommendations on how to best support the ongoing unfolding of those processes. In contrast, adult referrals more generally address a loss of adaptive skill and or capacity. Recommendations are sought regarding how to best support independence and financial status. Regardless of the type of questions being posed, both adult and pediatric populations often require rehabilitation to address traumatic insults to the brain; interventions are sought to assist with a return to functional capacity.

With regard to non-TBIs, adults are more likely to present with cerebrovascular accident (CVA) or stroke, whereas children are more likely to present with difficulties due to seizure disorders, cancer and cancer treatments, infections, neurodevelopmental disorders, and the effects of neurotoxins. These disorders have distinct effects on functioning and consequently affect the focus of a neuropsychological assessment. Interventions and accommodations that are recommended typically reflect the developmental and adaptive needs of the individual, given her specific neurological presentation.

## Disorders in Adulthood

One of the primary reasons an adult presents to rehabilitation is to address the sequelae of a TBI. TBIs are categorized as mild, moderate, or severe; however, there is not a universal classification system and several indicators are typically used to classify TBI severity. The most commonly used indices are length of time the individual lost consciousness and degree of posttraumatic amnesia (PTA). Although loss of consciousness for 30 minutes or less is classified as a mild injury, loss of consciousness for greater than 30 minutes is characterized as a moderate to severe injury (Lucas, 2003). PTA is the inability to recall events following the head injury. When PTA is less than 1 hour, the injury is classified as mild. PTA of 1 to 24 hours is moderate, and memory disturbance that lasts more than 24 hours postinjury is described as severe (Lucas, 2003). The Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) is also commonly used to measure TBI severity, although it is more sensitive to moderate and severe injuries (Lucas, 2003). Patients are rated on a 1 to 5 scale across three domains: verbal response, eye opening, and motor response. Higher scores are associated with better functioning. A mild TBI (mTBI) is defined as a score of greater than or equal to 13 points. A moderate injury includes scores between 9 and 12 points and a severe TBI (sTBI) includes scores of less than 9 points (Lucas, 2003). Although some studies have observed subtle differences in attention and working memory in patients with mTBI, impairments are often difficult to detect using current standard neuropsychological measures and patients tend to improve to levels that are indistinguishable from healthy same-aged controls (Brenner et al., 2010; Cullum & Thompson, 1997; Vanderploeg et al., 2005). As a result, adults with mTBI rarely present for or seek out rehabilitation. Instead, individuals in the rehabilitation setting tend to present with moderate to severe TBI and its impact on their behavior and functioning.

Adult patients with moderate to severe TBI typically present with

impairments across multiple areas of cognitive, social, emotional, and adaptive functioning. Specifically, deficits in processing speed, inhibition, attention, memory, and visuospatial abilities have been identified as principal concerns following a moderate to severe TBI (Campbell et al., 2009, Jacobs & Donders, 2008; Rath et al., 2004; Sigurdardottir, Andelic, Roe, & Schanke, 2009). These cognitive impairments in turn affect adaptive and psychosocial functioning (Blakemore & Choudhury, 2006; Campbell et al., 2009; Giedd, 2008; Rath et al., 2004). With regard to recovery, many cognitive deficits resolve during the first several months postinjury, as recovery is rapid during this time (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005). After this time, recovery typically slows, although it may continue for up to 5 years postinjury (Christensen et al., 2008; Hammond, Hart, Bushnik, Corrigan, & Sasser, 2004; Temkin, Machamer, & Dikmen, 2003).

Another common cause of brain injury in adults is CVA. There are two primary classifications of CVA, *ischemic* and *hemorrhagic*. An ischemic stroke is caused by the disruption of blood flow to a specific area of the brain, which leads to cell death. A hemorrhagic stroke refers to bleeding in the cerebrum or the subarachnoid area and is usually the result of an aneurism or hypertension (Barker-Collo & Feigin, 2006). A history of a stroke is associated with lower cognitive functioning across all domains (Knopman et al., 2009). Impairments in attention, mental speed, and memory are noted, as are motor and sensory difficulties (Barrett et al., 2006; Nøkleby et al., 2008). In particular, sensory loss associated with spatial deficits, such as neglect, impaired vision, field deficits, and motor dysfunction, as well as aphasia, and apraxia may result (Barrett et al., 2006; Nøkleby et al., 2008). Further, difficulties with emotion regulation and activities of daily living are often observed (Barrett et al., 2006).

Depending on their location in the brain, strokes impact different areas of functioning (Knopman et al., 2009). This, in turn, affects the results of a neuropsychological assessment and the focus on rehabilitation (Jokinen et al., 2006). For example, right-hemisphere strokes are associated with neglect, visuospatial and visuoconstructive disorders, *aprosodia* (the inability to produce or comprehend spoken language), *anosognosia* (lack of awareness of illness/deficit), *anosodiaphoria* (indifference to illness/deficit), and social deficits (Barrett et al., 2006; Donovan et al., 2008; Mosch, Max, & Tranel, 2005). In contrast, left-hemisphere stroke is commonly associated with aphasia, mutism, buccofacial apraxia, agraphia, acalculia, ideational apraxia, left confusion, and speech impairments (Donovan et al., 2008; Mosch et al., 2005).

Whereas strokes of the posterior cerebral artery (PCA) tend to lead to color agnosia, visual agnosia, alexia, facial agnosia, and amnesia, those in the anterior cerebral artery cause deficits in planning, initiation, monitoring, concentration, and flexibility (Donovan et al., 2008). Additionally, strokes in subcortical regions of the brain also have a unique impact on cognitive functioning. Thalamic strokes are associated with impairments in arousal, attention, motivation, initiation, executive functioning, and memory (Donovan et al., 2008; Jokinen et al., 2006; Knopman et al., 2009). Impaired problem solving, attention, and memory are observed in individuals with CVAs in the caudate (Donovan et al., 2008). Individuals with limbic and paralimbic strokes are unable to learn and retain new information (Donovan et al., 2008). Despite the current state of research on the deficits that result from stroke, there is no current consensus regarding the best approach to take to assess the resulting impairments (Barrett et al., 2006).

Another cause of non-TBI is cancer and cancer treatments. A study of breast cancer patients found that 35% of the patients presented with cognitive deficits prior to the onset of chemotherapy (Wefel et al., 2004). Although learning and verbal memory abilities are most consistently impacted, other impairments may arise and are dependent on the type of cancer. For example, individuals with acute myelogenous leukemia (AML) and myelodysplastic syndrome tend to present with impaired learning and memory, processing speed, executive function, and fine-motor abilities (Meyers, Albitar, & Estey, 2005). In contrast, individuals with small cell lung cancer are more likely to show deficits in verbal memory, motor coordination, and executive function (Meyers, Byrne, & Komaki, 1995).

In addition to the direct effects of cancer, treatments have also been demonstrated to affect cognitive functioning. Neuroimaging has shown that cancer treatments are associated with changes in neuroanatomy. For example, smaller gray and white matter volumes throughout the brain have been associated with chemotherapy in breast cancer patients (Inagaki et al., 2007). White matter changes have also been appreciated in individuals treated with methotrexate, and demyelination has been found in individuals prescribed with S-adenosyl methionine (SAM) (Shuper et al., 2000; Wefel, Witgert, & Meyers, 2008). Altered circuitry in the frontal regions of the brain and disruptions in brain functioning have been noted in individuals treated with interferon and tamoxifen (TAM) (see Schaefer et al., 2002 for a review; Wefel et al., 2008).

These alterations in brain structure are associated with changes in cognitive

functioning. For example, patients may experience difficulties with processing speed, inefficient learning, short-term memory, word recall, and organization (Wefel et al., 2008). Attention and concentration may also be affected (Wefel et al., 2008). It is of note that improvements are often observed and experienced subsequent to treatment completion (Wefel et al., 2008; Weis, Poppelreuter, & Bartsch, 2009).

## Disorders in Childhood

TBI is the leading cause of death and disability in youth in the United States and frequently necessitates rehabilitation services (Hotz & Chapman, 2009). The primary causes of TBI in children are motor vehicle accidents, sports injuries, falls, and child abuse (Hotz & Chapman, 2009; Sohlberg & Mateer, 2001). Although most children with mTBI recover completely within the first year, children with severe injuries recover more slowly and tend to have poorer outcomes (Anderson et al., 2005; Wilson & Gracey, 2009). This is because the brain must not only heal, but also adapt to the injury and then resume its developmental path, however altered (Wilson & Gracey, 2009).

On neuropsychological assessments, the effects of sTBI can be observed across all areas of functioning. On measures of general cognitive functioning, children with sTBI tend to have lower Full Scale IQs (Wilson & Gracey, 2009). Performance on nonverbal tasks is particularly likely to be depressed (Wilson & Gracey, 2009; Yeates, 2000). Immediately postinjury, intellectual functioning, in conjunction with information about injury severity, is a good predictor of outcome (Wilson & Gracey, 2009). Specifically, children with sTBI are more likely than those with mTBI to present with slower processing speed, as well as impaired verbal abilities, perceptual motor skills, verbal memory, attention, and written language (Kinsella et al., 1995; Taylor et al., 1999; Wilson & Gracey, 2009). They also frequently have more behavior problems and poorer adaptive abilities (Taylor et al., 1999).

Severe TBI in children also impacts many other areas of neurocognitive functioning. During the initial phase of recovery, deficits can initially be seen in alertness and orientation. Children may experience confusion, memory loss, and other changes in mental status (Yeates, 2000). Impaired executive function is also common. Specifically severe brain injury is associated with difficulties with poor metacognitive abilities, such as planning and self-monitoring (Cicerone et al., 2005; Dennis et al., 1996). Further, children may demonstrate weaknesses in cognitive flexibility, verbal fluency, and concept formation (Yeates, 2000).



These difficulties may be particularly notable for children who sustain frontal lobe injuries ([Dennis et al., 1996](#)).

Language is another area of functioning that is often impacted by sTBI. Unlike adults, there is significantly more variability in anatomical organization and localization of language in children, as well as cerebral plasticity ([Dall'Oglio, Bates, Volterra, Di Capua, & Pezzini, 1994](#); [Hertz-Pannier et al., 2002](#)). Consequently, there is not a direct relation between area of injury and subsequent deficits. Another difference between adults and children is that in children the left hemisphere is more resilient to damage. The brains of young children can also reorganize so that the right hemisphere can assume some components of language. As such, children with a lesion in a specific brain region may experience more diffuse language deficits, which may be receptive or expressive in nature. Despite this, some patterns in impairment have been documented. Long-term deficits in language comprehension, for example, have been found in children with an injury to the left temporal lobe ([Dennis et al., 1996](#)). Additionally, children who sustain an injury to the frontal lobe tend to present with impaired language production and verbal fluency ([Dennis et al., 1996](#); [Yeates, 2000](#)).

Whereas the left hemisphere is fairly resilient to injury, the right hemisphere is more vulnerable, as nonverbal skills are commonly impacted by sTBI. Specifically, many children exhibit visuoperceptual and constructional impairments ([Yeates, 2000](#)). Memory may also be affected by brain injury. Children may have difficulty with word recognition, recall of information regardless of whether structure is provided, and explicit memory ([Yeates, 2000](#)). Deficits in sensorimotor abilities may also be observed, particularly fine-motor skills. Further, children with TBI may also experience stereognosis, difficulty with finger localization, and graphesthesia ([Yeates, 2000](#)).

Impairments in cognitive functioning are often associated with academic difficulties. This may be a direct result of changes in cognitive functioning, as poor performance on cognitive measures postinjury is predictive of changes in academic placement; however, for many children, placement in a special education environment is due to behavioral sequelae and poor adaptive functioning ([Kinsella et al., 1995](#); [Yeates, 2000](#)). Despite these changes, academic skills acquired prior to the injury are relatively resilient to decline ([Kinsella et al., 2005](#); [Wilson & Gracey, 2009](#)).

The effect of TBI on the family is often not considered in children or adults, yet it can have a significant impact on recovery. Studies have found that

family burden and parental distress after TBI predict outcome (Taylor et al., 1999). Many families become distressed by the continued cognitive, emotional, and behavioral symptoms associated with the injury. This can be particularly difficult if the family members are unaware of the prognosis or expect full recovery (Yeates et al., 2001). Family dysfunction can also impact recovery (Taylor et al., 1999). Research indicates that emotional and behavioral symptoms postinjury can be affected by parent adjustment (Yeates et al., 2001).

In addition, another cause of brain injury in children is cancer and its treatment. Unlike the effect that it can have in adults, which often dissipates over time, cancer and cancer treatments can have long-term effects on development and overall functioning in children. Studies have found that brain tumors can cause deficits in attention, executive functioning, and processing speed, as well as in fluid and crystallized intellectual abilities (Mulhern & Butler, 2004). The impact of cancer is influenced by age and gender, with younger children and girls being at increased risk for more severe deficits (Butler & Mulhern, 2005; Ellenberg et al., 2009; Mulhern & Butler, 2004). For brain tumors, the severity of the impairments is dependent on the degree to which the tumor has invaded normal brain tissue. The more healthy tissue involved, the greater the risk for impairment (Mulhern & Butler, 2004). Brain tumors can also lead to seizures and hydrocephalus, which further increase risk for neurocognitive dysfunction (Mulhern & Butler, 2004).

Cancer treatments can also significantly impact all areas of cognitive functioning. Studies have found that children treated for brain tumors exhibit deficits in intelligence, attention, concentration, executive functioning, nonverbal memory, processing speed, and memory (Butler & Copeland, 2002; Butler & Mulhern, 2005; Mulhern & Butler, 2004). These impairments, in turn, lead to academic difficulties (Butler & Copeland, 2002; Butler & Mulhern, 2005). Additionally, children and adults treated as children experience social problems and a poorer quality of life (Butler & Mulhern, 2005, Mulhern & Butler, 2004).

There are various different kinds of treatments for childhood cancers, including surgery, cranial irradiation (CRT), and intrathecal injection of chemotherapy, which are associated with their own risks and deficits (Butler & Copeland, 2002; Butler & Mulhern, 2005, Mulhern & Butler, 2004). Surgical methods may be used to remove brain tumors, which can directly damage adjacent areas of the brain and cause impairment (Butler & Mulhern, 2005; Mulhern & Butler, 2004). CRT has the most negative impact on the developing

brain, as it causes demyelination and has been linked to smaller volume of white matter in the central nervous system (Butler & Copeland, 2002). The negative effects of CRT may develop within 2 years of treatment, but may be delayed for up to 7 years posttreatment (Jankovic et al., 1994). Because of this, many pediatric oncologists use intrathecal and systemic chemotherapy instead (Mulhern & Butler, 2004). Although these appear to cause less damage, 20% to 30% of children who undergo these treatments experience neurocognitive changes (Mulhern & Butler, 2004). These noninvasive treatments for cancer can last for years and, therefore, can have a pervasive effect on development (Butler & Mulhern, 2005).

Stroke is another cause of non-TBI in children. Children who experience unilateral strokes present with similar deficits as adults (Mosch et al., 2005). Although left-hemisphere stroke in adults is associated with abnormal speech and language, these abilities remain grossly intact in children (Mosch et al., 2005). When they are impacted, children tend to recover better than adults (Mosch et al., 2005). Left-hemisphere stroke is associated with impaired visuomotor integration (Mosch et al., 2005). Stroke in the right hemisphere causes poorer anterograde memory in children than in adults; however, children tend to be more significantly impacted. In contrast, children's social functioning tends to be less significantly impacted by left-hemisphere stroke than it is in adults (Mosch et al., 2005).

There are several other less common causes of brain injury in children. Specifically, seizures and other lesions are often caused by structural abnormalities (Byars et al., 2007). Studies indicate that these anomalies are associated with lower intellectual functioning, as well as impaired processing speed, executive functioning, and verbal memory (Byars et al., 2007). Neurodevelopmental disorders, such as cerebral palsy and spina bifida, are also associated with physical and cognitive impairments that may be addressed in rehabilitation. Additionally infections, such as meningitis and encephalitis, as well as environmental toxins can also lead to changes in the brain that affect neurocognitive functioning (Hotz & Copeland, 2009). Neuropsychological assessment can be used to assess the type and degree of impairment caused by these insults and guide rehabilitation, including the need for specific interventions, such as cognitive rehabilitation for attention, working memory, and problem solving, as well as assist in guiding efforts by physical, occupational, and speech therapists.

### **Considerations With Rehabilitation Neuropsychological**

## Considerations with Rehabilitation Neuropsychological Assessment

### Approaches to Assessment

Neuropsychological assessments may be conducted for a variety of reasons, which inform the neuropsychologist's choice of battery. The purpose of the assessment may range from a brief evaluation that provides basic data about a few areas of functioning to a more comprehensive evaluation that measures multiple domains in depth (Sattler, 2001). Within the context of rehabilitation, the brief assessment is often used as a screen to determine whether an individual has a disorder that necessitates rehabilitation, is eligible to participate in a particular program, or requires a more comprehensive evaluation (Sattler, 2001). In contrast, a comprehensive assessment provides more detailed information, which is typically used to offer a diagnosis, answer a diagnostic question, track progress over time, or determine eligibility for placement in a particular program (Sattler, 2001).

Within the context of brief and comprehensive batteries, there are three main types, each with its advantages and disadvantages: *fixed*, *flexible*, and *mixed* (Léon-Carrión, Taaffe, & Barroso y Martin, 2006). The fixed battery uses a single set of concurrently standardized measures that assess multiple domains of cognitive functioning. Examples include the *Halstead-Reitan Neurological Test Battery (HRB)*; Reitan & Wolfson, 1993), which is used most typically to assess multiple cognitive functions associated with potential broad-based dysfunction, like dementia, and the *Luria-Nebraska Neuropsychological Battery (LNNB)*; Golden, Hammeke, & Purisch, 1991), which is designed to assess a wide range of cognitive functions and is specifically designed to assist in developing rehabilitation plans (Sattler & D'Amato, 2002). Fixed battery approaches are often theoretically determined such that the tasks administered are meant to be interpreted in tandem and as a representation of an underlying theoretical model concerning brain function. They often also provide a summary determination regarding functional status. Ultimately, the emphasis of the fixed battery is on diagnosis. As such, the purpose of the assessment is to identify the probable location of a lesion (or lesions) in the brain and how that lesion affects functioning (Léon-Carrión et al., 2006). The primary advantage of a fixed battery is its ability to allow the neuropsychologist to rely on a large normative database against which performances across all subtests can be compared. It also provides a specified set of domains to be assessed. This allows for direct

comparison of performance across the specific domains examined (Sohlberg & Mateer, 2001) and across individuals with a similar set of concerns. In contrast to other options, however, reliance on a unitary battery to assess all cognitive domains, regardless of the specific concerns being addressed clinically, can reduce the neuropsychologist's flexibility in selecting and utilizing a more appropriate and germane set of tests to answer the referral question at hand (Sohlberg & Mateer, 2001). This can be problematic because the fixed battery may not measure all domains equally, and specific areas requiring more detailed investigation may be overlooked.

In contrast, a flexible battery allows the neuropsychologist to choose measures that more directly address for the referral question being posed. The primary objective of the flexible battery approach is to identify an individual's specific pattern of strengths and weaknesses, and to more directly outline specific domains of difficulty (Léon-Carrión et al., 2006). Further, other information from the assessment, such as behavioral observations, informs the interpretation of scores (Léon-Carrión et al., 2006). The benefits of this approach are that the examiner has greater flexibility in regard to both the depth and breadth with which different domains of cognitive functioning are assessed (Sohlberg & Mateer, 2001). It also allows the neuropsychologist to incorporate additional tests to supplement the existing data if it is deemed necessary and is germane to the clinical question at hand (Sohlberg & Mateer, 2001). Consequently, the flexible approach may enable the neuropsychologist to better evaluate the referral question (Sohlberg & Mateer, 2001). Despite these advantages, the use of multiple measures with different norms can affect comparisons of performance across tests (Sohlberg & Mateer, 2001).

It is also possible to combine fixed and flexible batteries, which can be accomplished in one of two ways. First, a fixed battery can be used to provide a core set of scores, which can then be supplemented with additional measures, as needed (Sohlberg & Mateer, 2001). Alternatively, there are several measures with subtests that assess multiple domains that can be administered in a flexible manner, but use the same normative sample. The Neuropsychological Assessment Battery (NAB) and the NEPSY II are examples of measures that integrate the advantages of fixed and flexible approaches. Because of the many advantages of the flexible battery, it is more commonly used than the fixed approach in rehabilitation settings (Sohlberg & Mateer, 2001); this is specifically the case when efforts to understand change over time are a priority.

Informants

## Informants

Although gathering data directly from the patient is essential to a neuropsychological evaluation, collecting information from other sources is also recommended. This can be particularly important in the context of rehabilitation when the patient's awareness of his or her impairments may be compromised. Whereas in adult populations, collateral information is often provided by spouses, children, and siblings, in pediatric populations, it is more likely to be provided by parents and teachers. Generally, the use of multiple informants provides a comprehensive representation of the patient's strengths and weaknesses, as well as clarifies.

## Clinical Interview

Regardless of the assessment approach and who provides information, it is important to obtain a thorough history as part of a neuropsychological assessment. Different areas may be emphasized depending on the age of the patient. One of the most important pieces of information within the rehabilitation setting is the nature of the brain injury (Sohlberg & Mateer, 2001). Specifically, the examiner should enquire about age at the time of injury, location and severity of the injury (if known), and how it has affected the patient (Frank & Elliot, 2000; Sohlberg & Mateer, 2001). Additionally, information about the patient's current functioning and environment should be gathered. This may include learning more about the current patient's support network and coping skills (Sohlberg & Mateer, 2001).

To better understand these changes, it is necessary to gather information about preinjury functioning. This includes cognitive, behavioral, emotional, social, and academic functioning (Sohlberg & Mateer, 2001). It is also important to enquire about the patient's strengths and weaknesses prior to the injury (Sohlberg & Mateer, 2001). For children, this may include academic functioning and, for adults, this may include a combination of academic and occupational functioning (Léon-Carrión et al., 2006). Information about developmental history should also be collected. When working with youth, a more detailed developmental history may be appropriate. This may include information about pregnancy and delivery, as well as about motor and language milestones. A medical and mental health history should also be included (Léon-Carrión et al., 2006).

It is also important to gather information about the patient's environment, as it accounts for a significant amount of the variance in postinjury outcomes

(Yeates, 2000). This includes gathering information about social supports, including from friends and family (Frank & Elliot, 2000; Sohlberg & Mateer, 2001). Information about the patient's family demographics, including socioeconomic status, family size, and structure of the family should be obtained as available (Frank & Elliot, 2000; Yeates, 2000). It may also be useful to inquire about the patient's and family's cultural background, as it may inform treatment (Frank & Elliot, 2000). Further, an understanding of the availability of supports is essential (Frank & Elliot, 2000). For both adult and pediatric populations, it is helpful to assess the types of services (e.g., educational or vocational, mental health, and support groups) to which the individual has access (Frank & Elliot, 2000). In addition to these basic facts about the family, it is also helpful to determine environmental stressors the patient may experience, as this can interfere with recovery (Sohlberg & Mateer, 2001).

Given that one component of the neuropsychologist's role in rehabilitation is to educate the patient and his or her family about the nature of the injury and its impact on functioning, it can be invaluable in assessing the patient's understanding of the injury and its effects (Sohlberg & Mateer, 2001). Additionally, the neuropsychologist should determine the patient's and family's expectations about recovery, as this can contribute to subsequent family stress and difficulties with adaptation and functioning (Sohlberg & Mateer, 2001). This includes getting a detailed understanding of short- and long-term expectations, as possible, and developing with the family an appreciation for the potential trajectories of recovery.

## Standardized Measures

In addition to gathering a complete patient history, standardized measures that assess cognitive, behavioral, emotional, academic, and adaptive functioning are typically administered as part of a comprehensive, tailored neuropsychological assessment. Cognitive and academic functioning are typically assessed using paper-and-pencil or computerized measures. Rating scales or semistructured interviews are used to gather data about emotional, behavioral, and adaptive functioning. Depending on the scale, the patient, a family member, and/or teacher may supply information about functioning. Gathering data from multiple informants and across environments can augment the patient's report, as it can provide insight into the patient's levels of awareness of his or her impairments (Sohlberg & Mateer, 2001). Further, it enables the examiner to look at differences in functioning across environments (Sohlberg & Mateer, 2001).

## Observations

Although often difficult to do, in vivo observations of the patient can complement the data collected in the office. As standardized measures do not always directly translate to the ability to complete daily tasks, observations provide the examiner with the opportunity to assess the patient's ability to perform everyday tasks in his or her real-life environment (Sohlberg & Mateer, 2001). For example, the examiner can observe the patient's ability to dress and prepare meals independently (Sohlberg & Mateer, 2001). Observations may be structured or unstructured and may be conducted in any number of environments. They are typically performed at the patient's home, school, or place of work (Sohlberg & Mateer, 2001).

## Neuropsychological Assessment: Domains and Measures

The cognitive component of a neuropsychological battery typically assesses multiple domains, which may include attention, executive function, memory, language, visuospatial skills, and sensorimotor abilities. Additional tests may be used to gather supplementary data about a particular domain in order to answer the referral question. Neuropsychological evaluations in a rehabilitation setting will include these core domains, but may also measure other areas of cognitive functioning that are pertinent to the patient's current presentation and needs. The tests described in this chapter are not an exhaustive list; rather they are ones that are widely used to assess functioning within each domain.

## Injury Severity

Initially following a brain injury and generally within the emergency room setting, level of injury severity is determined through the assessment of loss of consciousness, duration of loss of consciousness, duration of amnesia, and extent of coma (Yeates, 2000). The Glasgow Coma Scale (Teasdale & Jennett, 1974) is the most commonly used measure to assess the severity of TBI (Léon-Carrión et al., 2006; Sigurdardottir et al., 2009; Yeates, 2000). The scale assesses eye opening, motor response, and verbal response. Scores can range from 3 to 15, with lower scores reflecting more severe injuries (Teasdale & Jennett, 1974). A modified version of the scale, called the Children's Coma Scale, has been developed (Baron, 2004; Ewing-Cobbs et al., 1998). It assesses motor and verbal responses; however, for children younger than 36 months of age, it allows for appropriate nonverbalized responses to be taken under consideration (Baron,



2004; Hahn et al., 1988).

## Alertness and Orientation

Mental status often varies in the period immediately following a TBI and is referred to as PTA (Yeates, 2000). This can also be used as another indicator of trauma severity (Yeates, 2000). These measures tend to assess arousal, alertness, and orientation.

*Adults.* There are a number of scales that can be used to assess mental status in adults. One of the more commonly used measures is the Galveston Orientation and Amnesia Test (GOAT; Levin, O'Donnell, & Grossman, 1979), which assesses degree and duration of disorientation to person, place, and time, as well as amnesia (Bode, Heinemann, & Semik, 2000; León-Carrión et al., 2006; Sohlberg & Mateer, 2001). It consists of 100 questions and is scored based on the number of questions answered correctly on a scale of 0 to 100 with higher scores being indicative of higher functioning. As this measure can be difficult to administer to patients who are not verbal, it can be difficult to use in rehabilitation settings (Novack et al., 2000). Additionally, some questions may be difficult to answer even for verbal patients and it can be difficult to score (León-Carrión et al., 2006). Another scale often used to measure orientation is the Orientation Log test (O-Log). Like the GOAT, it can be used over time and used to estimate recovery time. The O-Log has 10 questions, with scores that range from 0 to 30 (León-Carrión et al., 2006).

*Child.* Similar tests of orientation are available for children. The Children's Orientation and Amnesia Test (COAT; Ewing-Cobbs, Levin, Fletcher, Miner, & Eisenberg, 1990) was designed as the children's counterpart of the Glasgow Coma Scale, but is used to measure orientation and memory (Baron, 2004; Ewing-Cobbs et al., 1990). It consists of 16 items and can be used with children between the ages of 3 and 15 years of age (Ewing-Cobbs et al., 1990).

## Intellectual and Overall Cognitive Functioning

Deficits in intellectual functioning may be noted postinjury. As intellectual functioning immediately following a TBI can provide information about outcome, assessment of overall cognitive functioning is important. These tests typically assess verbal and nonverbal abilities, as well as memory and processing speed. Depending on the age of the patient, it may also include measures of motor development.

*Adult.* The most commonly used measure for adults is the *Wechsler Adult*

*Intelligence Scale, Fourth Edition (WAIS-IV; Wechsler, 2008)*. It can be used with individuals between the ages of 16 years and 90 years, 11 months. The Full Scale IQ (FSIQ) is composed of four indices—verbal comprehension, perceptual reasoning, working memory, and processing speed. Ten core factors contribute to these indices; however, an additional five subtests may be administered. Scores on the WAIS-IV range from 40 to 160, with a mean of 100 and a standard deviation (SD) of 15 (Wechsler, 2008). The verbal subtests of the WAIS-IV appear to be less sensitive to brain damage than nonverbal tests. This has been attributed to the “over learned” and crystallized nature of the information assessed in the verbal subtests. An advantage of using this measure is that it has been normed with the *Wechsler Individual Achievement Test, Third Edition (WIAT-III, Wechsler, 2009a)* and *Wechsler Memory Scale, Fourth Edition (WMS; Wechsler, 2009b)*.

Another test of intelligence is the *Stanford-Binet, Fifth Edition (SB5; Roid, 2003)*. It can be used with individuals from 2 to 85 years of age. Like the WAIS-IV, it provides an FSIQ that is composed of two domains, nonverbal and verbal, as well as five factors scores that are based on specific subtests (Strauss, Sherman, & Spreen, 2006). The factors include fluid reasoning, knowledge, quantitative reasoning, visual-spatial processing, and working memory (Strauss et al., 2006). Scores range from 40 to 160, with a mean of 100 and an SD of 15. Age equivalents are also available (Strauss et al., 2006).

When it is not possible or necessary to conduct a comprehensive assessment of general cognitive functioning, the *Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990)* can be used. It takes approximately 15 to 30 minutes to administer. Like the SB5, it can be administered to children and adults. The age range is 4 through 90 years of age (Strauss et al., 2006). The test has a mean of 100 and an SD of 15. The K-BIT consists of two tests, which measure verbal and nonverbal abilities (Strauss et al., 2006). Another brief test of intelligence is the *Wechsler Abbreviated Test of Intelligence (WASI; The Psychological Corporation, 1999)*. Like the K-BIT it can be administered to children and adults, with an age range of 6 to 89 years old. The WASI provides somewhat more flexibility than the K-BIT, as it allows for the administration of either two or four tests, which measure verbal and nonverbal functioning. These take 15 to 30 minutes to administer (Strauss et al., 2006).

There are also several measures that can be used to assess intelligence for adult patients who are nonverbal or who have significant speech or communication impairments. The *Test of Nonverbal Intelligence, Third Edition*

(TONI-3; [Brown et al., 1997](#)) is used to assess nonverbal reasoning individuals between the ages of 6 and 89 years, 11 months. Items consist of matrix or nonverbal analogic reasoning tests ([Strauss et al., 2006](#)). Directions are pantomimed by the examiner and the examinee uses gestures to communicate responses ([Strauss et al., 2006](#)). The *Comprehensive Test of Nonverbal Intelligence* (CTONI; [Hammill et al., 1997](#)) can also be used with nonverbal patients to assess intelligence. It can be used with children from the age of 6 years to adults of age 90 years ([Baron, 2004](#)). Like the TONI-3, instructions for the CTONI are pantomimed and gestures are used by the examinee to respond ([Baron, 2004](#)).

*Child.* The *Wechsler Intelligence Scale for Children, Fourth Edition* (WISC-IV; [Wechsler, 2003](#)) is the most commonly used measure to assess general cognitive functioning in children. It can be used for children 6 years to 16 years, 11 months of age ([Strauss et al., 2006](#)). The WISC-IV is composed of the same four factor structure as the WAIS-IV, with the same mean and SD. Although the WISC-IV also contains 10 core subtests, they differ from those of the WAIS-IV. Studies of children with TBI indicate that all indices will be somewhat depressed, but the Processing Speed Index (PSI) appears to be most impacted and the Perceptual Reasoning Index (PRI) least affected. For younger children, aged 2 years, 6 months to 7 years, 3 months, the *Wechsler Preschool and Primary Scale of Intelligence, Third Edition* (WPPSI-3, [Wechsler, 2002](#)) can be used.

Another measure of intellectual functioning for children is the *Differential Abilities Scale, Second Edition* (DAS-2; [Elliot, 2007](#)). It can be used with children aged 2 years, 6 months through 17 years, 11 months. Scores range from 30 to 170, with a mean of 100 and an SD of 15. It provides a General Ability Composite, which is composed of one to three clusters depending on the child's age. Whereas for young preschool children there is only one cluster that taps verbal and nonverbal abilities, for older preschool children there are two separate clusters for verbal and nonverbal abilities ([Baron, 2004](#)). The form for school-age children includes three clusters, which assess verbal, nonverbal reasoning, and spatial abilities ([Baron, 2004](#)).

When working with infants and young children, there are two tests of cognitive functioning and development that are typically used, the *Bayley Scales of Infant Development, Third Edition* (Bayley-3; [Bayley, 2006](#)) and the *Mullen Scales of Early Learning* ([Mullen, 1995](#)). The Bayley-3 assesses motor, mental, and behavioral development in children between the ages of 1 and 42 months

([Baron, 2004](#); [Bayley, 2006](#)). The *Mullen Scales* can be used with children from birth to 68 months of age. It assesses fine and gross motor development, receptive and expressive language, and visuospatial skills.

## Attention

Attention is a multifaceted domain, which includes the ability to orient and focus on a stimulus, vigilance, shifting attention among stimuli, selective attention, divided attention, and distractibility ([Baron, 2004](#); [Limond & Leeke, 2005](#)). Because attention is not mediated by a specific region of the brain, but rather is composed of distinct networks, most traumatic and nontraumatic brain injuries affect attentional abilities (Fernandez-Duque & Posner, 2001). It is also possible for other factors, such as mood and motivation, to affect performance on measures of attention ([Baron, 2004](#)).

There is a wide range of measures that assess attention in pediatric and adult populations. Most measures of general cognitive functioning tap attentional abilities and can act as a screening tool for more significant deficits ([Strauss et al., 2006](#)). For example, the *WISC-IV* and *WAIS-IV* include several measures of working memory, as well as tests that tap attention to detail. A more comprehensive assessment of this domain will likely be needed in the context of rehabilitation using specialized measures of attention. Most tests assess one of four areas of attention: divided, sustained, shifting, and selective.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Brief Test of Everyday Attention</i>	17 to 82 years		Divided attention
<i>Children's Paced Auditory Serial Addition Test (CHIPSAT)</i>	8 to 14 years, 6 months		Processing speed Sustained attention Divided attention
<i>Continuous Performance Test, Second edition (CPT-2)</i>	6 to 55 years		Sustained attention
<i>Delis-Kaplan Executive Functioning System (DKEFS)</i>	8 to 89 years	Color-Word Interference Test Trail Making Test	Selective attention Visual scanning Sequencing Motor speed Switching attention
NEPSY-2	3 to 16 years	Auditory Attention and Response Set Statue	Sustained attention
<i>Paced Auditory Serial Addition Test (PASAT)</i>	16 to 74 years		Processing speed Sustained attention Divided attention
<i>Stroop Color-Word Test</i>	5 to 94 years <sup>a</sup>		Selective attention Cognitive flexibility Inhibition

<i>Test of Everyday Attention (TEA)</i>	18 to 80 years	Elevator Counting Elevator Counting with Distraction Elevator Counting with Rehearsal Lottery Map Search Telephone Search Telephone Switch and Counting Visual Elevator	Selective attention Divided attention Sustained attention Shifting attention
<i>Test of Everyday Attention for Children (TEA-Ch)</i>	6 to 16 years	Code Transmission Creature Counting Map Mission Opposite Words Score Score DT Sky Search Sky Search DT Walk-Don't Walk	Selective attention Divided attention Sustained attention Shifting attention
<i>Trail Making Test (TMT)</i>	9 to 89 years	Trails A: Connect Numbers Trails B: Alternate between Connecting Numbers and Letters	Visual scanning Sequencing Motor speed Switching attention
<i>WAIS-IV</i>	16 to 90 years, 11 months	Cancellation Coding Digit Span Symbol Search	Working memory Attention to detail Selective attention
<i>WISC-IV</i>	6 to 16 years, 11 months	Cancellation Coding Digit Span Letter Number Sequencing Symbol Search	Working memory Attention to detail Selective attention Divided attention

Note: See [Baron \(2004\)](#) and [Strauss et al. \(2006\)](#) for a more complete overview of the tests.

<sup>a</sup> Depends on version used.

## Executive Functioning

Like attention, executive function is a complex construct, which has been conceptualized in various ways. Ida Sue [Baron \(2004\)](#) defines executive function as:

the metacognitive capacities that allow an individual to perceive stimuli from his or her environment, respond adaptively, flexibly change direction, anticipate future goals, consider sequences, and respond in an integrated or common-sense way, utilizing all of these capacities to serve a common purposive goal.

To this definition, the emotional components of executive function, specifically the ability to regulate one's emotional response can be added. Although executive functioning abilities are primarily controlled by the frontal lobe, other regions of the brain are involved. As mentioned previously, insults in

childhood to any number of brain regions can also interfere with the development of executive function. Unfortunately, these difficulties are often not apparent until adolescence when executive function typically comes online. A comprehensive evaluation of executive function can assist in assessing the severity of the deficit and can help guide treatment regardless of developmental stage.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Category Tests</i>	5 to 80 years <sup>a</sup>	5 to 7 Subtests	Cognitive flexibility Concept formation
<i>Behavioral Dyscontrol Scale (BDS)</i>	21 to 102 years		Self-monitoring Inhibition
<i>Brief</i>	5 to 18 years		Planning Organization Initiation Inhibition Cognitive flexibility Emotional control Working memory Self-monitoring
<i>Cambridge Neuropsychological Assessment Test Automated Batteries (CANTAB)</i>	4 to 90 years	Big Little Circle Intra-Extra Dimensional Shift Matching to Sample Motor Screening Paired Associate Learning Pattern Recognition Memory Rapid Visual Information Processing Reaction Time Spatial Recognition Memory Spatial Span Spatial Working Memory Stockings of Cambridge	Learning and memory Working memory Attention Planning Cognitive flexibility Motor speed
<i>Controlled Oral Word Association Test</i>	5 to 89 years		Fluency Self-monitoring Inhibition Initiation

<i>DKEFS</i>	8 to 89 years	Card Sort Color-Word Interference Test Design Fluency Proverbs Tower Trail Making Task Twenty Questions Verbal Fluency Word Context Test	Cognitive flexibility Planning Fluency Concept formation and categorization Sequencing Inhibition Motor speed
<i>Go/No-Go Tasks</i>			Inhibition
<i>NEPSY-2</i>	3 to 16 years	Animal Sorting Auditory Attention and Response Set Clocks Design Fluency Inhibition	Cognitive flexibility Fluency Concept formation and categorization Inhibition
<i>Porteus Maze Test (PMT)</i>	3 years and older		Planning Motor speed
<i>Rey Complex Figure Test (RCFT)</i>	6 to 93 years	Copy	Organization Planning
<i>Stroop Test</i>	5 to 94 years	Word Color Color-Word Interference	Selective attention Cognitive flexibility Inhibition
<i>Tower Tests: Hanoi London</i>	4 to 8 years, 7 to 12 years		Planning
<i>Wisconsin Card Sort Test (WCST)</i>	6 years, 6 months to 89 years	Color Form Number	Cognitive flexibility Concept formation Sustained attention

<sup>a</sup> Age range can be tested using one of three versions: child, intermediate, and adult.

## Language, Speech, and Communication

There is not a universally accepted definition of language; however, current definitions include the use of combinations of sounds in order to communicate, which are guided by rules so that they are meaningful (Baron, 2004; Kolb & Wishaw, 2008; Larson, 1999). Speech, on the other hand, is the motor component of verbal communication. These skills can be broken down further into *expressive language* and *receptive language*, which are the abilities to effectively produce and comprehend language. These components of language can be affected by brain injuries. It is of note that in children, who are in the process of developing language and in which language control is more pervasive, an insult to a specific region in the brain may impact multiple areas of functioning.

When language impairment is suspected, the neuropsychological assessment should evaluate both expressive and receptive language skills. An



evaluation should also include an assessment of expressive language skills, such as *prosody* (intonation that can affect the meanings of words and sentences), fluency, word retrieval, and the ability to correctly name letters, words, and objects, as well as the ability to communicate through writing. Receptive language should also be assessed through measures of verbal and written comprehension. Further, *phonological processing* (the use of sounds in processing language), *phonological awareness* (the ability to appreciate the components of language), *syntax* (the rules about how words are put together), and *semantics* (the meaning of words) should be assessed. Depending on performance, additional measures of language should be included to assess particular areas of language in more depth.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Boston Naming Test, Second Edition (BNT-2)</i>	5 to 13 years ≥18 years		Naming
<i>Boston Diagnostic Aphasia Examination, Third Edition (BDAE-3)</i>	≥16 years	50 Subtests	Conversational and expository speech Auditory comprehension Oral expression Reading Writing Praxis
<i>Clinical Evaluation of Language Fundamentals, Fourth Edition (CELF-4)</i>	5 to 21 years	Concepts and Following Directions Expressive Vocabulary Familiar Sequences Formulating Sentences Number Repetition Phonological Awareness Rapid Automatic Naming	Naming Verbal memory Working memory Semantics Syntax Phonological awareness

		Recalling Sentences Semantic Relations Sentence Assembly Sentence Structure Understanding Spoken Paragraphs Word Associations Word Classes: Receptive, Expressive, Total Word Definitions Word Structure	
<i>Expressive Oral Word Vocabulary Test, Third Edition (EOWVT-3)</i>	2 to 18 years, 11 months		Word retrieval Naming
<i>Expressive Vocabulary Test (EVT)</i>	≥2 years, 6 months		Word retrieval Naming
<i>Halstead-Wepman Aphasia Screening Test</i>	≥5years*		Naming Spelling Writing Verbal memory Reading Visuospatial Calculation Comprehension
<i>Multilingual Aphasia Examination (MAE)</i>	2 to 12 years 16 to 97years	Aural Comprehension of Words and Phrases Block Letter Spelling Controlled Oral Word Association Oral Spelling Rating of Articulation Rating of Praxis Features of Writing Reading Comprehension of Words and Phrases Sentence Repetition Token Test Visual Naming Written Spelling	Naming Verbal memory Comprehension Verbal fluency Spelling

<i>NEPSY-2</i>	3 to 16 years	Body Part Naming and Identification Comprehension of Instructions Oromotor Sequences Phonological Processing Repetition of Nonsense Words Speeded Naming Word Generation	Phonological awareness Verbal memory Receptive language Naming Comprehension Word retrieval Oromotor coordination
<i>Peabody Picture Vocabulary Test, Third Edition (PPVT-3)</i>	≥2 years, 6 months		Receptive vocabulary
<i>Receptive One-Word Picture Vocabulary Test (ROWPVT)</i>	2 to 18 years, 11 months		Receptive vocabulary
<i>Token Test and Token Test for Children</i>	≥5 years	Parts A to F	Verbal comprehension

<sup>a</sup> Age range can be tested using one of three versions of the test.

## Learning and Memory

Memory is a multifaceted construct that includes the processes of taking in new information, encoding it, storing it, and recognizing it. It has been categorized and classified in numerous ways; however, this chapter will focus on those processes that are typically measured in the context of rehabilitation. Before one can recall information, it must be acquired and encoded through the process of *learning*. The process responsible for holding information in mind for a short period of time so that the information can be used is called *working memory* (Loring, 1999). There is some confusion and disagreement about short-term and long-term memory, as there is not an exact moment when encoded information shifts from one process to another. Neuropsychological measures generally assess the ability to remember information immediately after it was presented, *immediate memory*, and after a delay (typically 20–30 minutes), *delayed recall* or *delayed memory* (Loring, 1999). These processes may be further divided into the ability to recall information without cues, *free recall*, and with cues, *cued recall*, as well as the ability to identify previously presented information, *recognition* (Loring, 1999).

Depending on the region that is injured, individuals may only be able to remember some categories of information or information presented through a specific modality. The most general categories of this are *explicit and implicit memory* (Limond & Leeke, 2005; Loring, 1999). Whereas explicit memory refers to the conscious recall of events and experiences, implicit memory occurs at an unconscious level. Explicit memory can be further classified into information about specific events, *episodic memory*, and information that reflects general knowledge, *semantic memory* (Limond & Leeke, 2005; Loring, 1999). Although there are several kinds of implicit memory, within the context of neuropsychological assessment the focus tends to be on procedural memory, which is well-learned knowledge of how to perform tasks (Limond & Leeke, 2006; Loring, 1999). The ability to learn and recall information visually or auditorily may also be impacted by a brain lesion and, therefore, should also be assessed.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Benton Visual Retention Test, Fifth Edition</i>	6 to 11 years 17 to 97 years		Visual memory Visuoperceptual Perceptual Organizational
<i>California Verbal Learning Test, Children's Edition (CVLT-C)</i>	5 to 16 years, 11 months		Verbal learning Verbal immediate memory Verbal delayed recall Delayed recognition Interference
<i>California Verbal Learning Test, Second Edition (CVLT-2)</i>	16 to 89 years		Verbal learning Verbal immediate memory Verbal delayed recall Delayed recognition Interference
<i>Children's Memory Scale (CMS)</i>	5 to 16 years, 11 months	Dot Locations Faces Family Pictures Stories Word Lists Word Paris	Verbal immediate memory Verbal delayed memory Visual immediate memory Visual delayed memory Interference Delayed recognition
<i>NEPSY-2</i>	3 to 16 years	List Memory Memory for Designs Memory for Faces Memory for Names Narrative Memory Narrative Memory Sentence Repetition Word List Interference	Verbal memory Verbal learning Visual memory Working memory Spatial memory Immediate memory Delayed recall Recognition

<i>Rey Auditory Verbal Learning Test (RAVLT)</i>	6 to 97 years		Immediate memory Interference Delayed recall Delayed recognition
<i>Rey Complex Figure Test (RCFT)</i>	6 to 93 years		Visual immediate memory Visual delayed recall Delayed recognition Visuoperceptual Perceptual organizational
<i>Test of Memory and Learning, Second Edition (TOMAL-2)</i>	5 to 19 years	Abstract Visual Memory Digits Backward Digits Forward Facial Memory Letters Backward Letters Forward Manual Imitation Memory for Location Memory for Stories Object Recall Paired Recall Visual Selective Reminding Visual Sequential Memory Word Selective Reminding	Verbal immediate memory Verbal delayed memory Visual immediate memory
<i>Wechsler Memory Scale, Fourth Edition (WMS-IV)</i>	16 to 89 years	Design Memory General Cognitive Screener Logical Memory I and II Spatial Addition Symbol Span Verbal Paired Associates I and II Visual Reproduction I and II	Verbal immediate memory Verbal delayed memory Visual immediate memory Visual delayed memory Interference Delayed recognition Working memory
<i>Wide Range Assessment of Memory and Learning, Second Edition (WRAML-2)</i>	5 to 17 years, 11 months	Design Memory Finger Windows Number/Letter Memory Picture Memory Sentence Memory Sound Symbol Story Memory Verbal Learning Visual Learning	Working memory Verbal memory Visual memory Recognition

## Visuospatial, Visuoperceptual, and Perceptual Organizational Skills

In children and adults, the abilities to perceive, represent, organize, and integrate visual information can be negatively affected by brain injury. Deficits in these skills are typically reflective of damage to the right hemisphere, although not exclusively. When selecting neuropsychological measures to assess visuospatial skills, it is important to note that tests may also assess other areas of functioning, such as executive functioning and sensorimotor abilities. Consequently, difficulties on the test may not be specifically due to a right-hemisphere lesion, which is the reason a comprehensive neuropsychological battery is important to tease out the specific area of weakness. This potentially allows for rehabilitation and other interventions to be more focused and effective.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Beery Developmental Test of Visual Motor Integration</i>	3 to 17 years	VMI Visual Motor	Visuomotor integration Visuoperceptual
<i>Bender Gestalt</i>	≥3 years		Neglect Visuomotor integration
<i>Clock Drawing Test</i>	6 to 12 years Adults		Visuomotor Visuospatial Executive control Visual inattention
<i>Hooper Visual Organization Test (VOT)</i>	5 to 91 years		Neglect Visual integration Visuoperceptual
<i>Judgment of Line Orientation (VLOT)</i>	7 to 96 years		Visuoperceptual
<i>NEPSY-2</i>	3 to 16 years	Arrows Block Construction Design Copy Geometric Puzzles Picture Puzzles Route Finding	Visuoperceptual processing Visuospatial processing Visuomotor Visual discrimination Spatial localization
<i>Rey Complex Figure Test</i>	6 to 93 years		Visual immediate memory Visual delayed recall Delayed recognition Visuoperceptual Perceptual organizational
<i>Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV)</i>	6 to 16 years, 11 months	Block Design Cancellation	Visuospatial Neglect
<i>Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV)</i>	16 to 90 years, 11 months	Block Design Cancellation	Visuospatial Neglect

## Sensorimotor and Praxis Functions

As individuals with TBI often present with difficulties performing certain activities of daily living, assessment of sensorimotor skills and *praxis* should be assessed. Within this domain, neuropsychological evaluations may assess various aspects of sensorimotor skills. Subtle signs of an impaired nervous system may be appreciated through observation or standardized measures, such as difficulties with coordination, dysarthria, and dyskinesia (Baron, 2004). It is also important to note the patient's dominant hand, as it can provide clues about lateralization and risk for certain neurological impairments (Baron, 2004). Additionally, evaluations may assess motor speed, motor strength, and motor dexterity, as well as the ability to perform specific movements (Baron, 2004). It may also be appropriate to assess the patient's ability to perceive and recognize sensory information (Baron, 2004).

## Emotion, Behavior, and Personality

Patients with brain injury frequently present with changes in mood, personality, and behavior. Although these changes may be directly caused by alterations in brain structure and function, they may also be associated with a response to the effects of injury. As such, it is important to not only assess current emotional and behavioral functioning, but also preinjury functioning. Depending on the presenting problems and referral questions, it may be appropriate to use a measure that assesses a broad range of symptoms to screen for emotional or behavioral difficulties. Alternatively, more indepth assessment of depression or anxiety may be needed. Although the use of standardized measures is important to diagnosis, it should not be used in place of information obtained through the patient interview and throughout the assessment.

TESTS	AGE RANGE	SUBTESTS	SKILLS ASSESSED
<i>Beery Developmental Test of Visual Motor Integration</i>	3 to 17 years	Visual VMI Motor	Visuomotor integration Visuoperceptual
<i>Finger Tapping Test (FTT)</i>	5 to 85 years		Motor dexterity Motor speed
<i>Grip Strength Test</i>	6 to 8 years		Motor strength
<i>Grooved Pegboard Test</i>	6 to 85 years		Motor dexterity Motor speed
<i>Halstead-Reitan Battery</i>	≥5 years <sup>a</sup>	Finger Tapping Test Tactual Performance Test Reitan-Klove Sensory- Perceptual Examination	Sensory recognition Motor speed Motor dexterity
<i>NEPSY-2</i>	3 to 16 years	Finger Tapping Imitating Hand Movements Manual Motor Sequences Visuomotor Precision	Motor speed Motor dexterity
<i>Pantomime Recognition Test</i>	3 to 8 years		Praxis
<i>Tactual Performance Test (TPT)</i>	5 to 85 years		Sensory recognition

<sup>a</sup> Age range can be tested using one of three versions of the test.

TESTS	AGE RANGE	AREAS ASSESSED
<i>Behavior Assessment System for Children, Second Edition (BASC-2)</i>	2 to 21 years, 11 months	Internalizing symptoms Externalizing symptoms Activities of daily living Functional communication
<i>Beck Depression Inventory, Second Edition (BDI-2)</i>	13 to 80 years	Depression
<i>Becky Anxiety Inventory (BAI)</i>	17 to 80 years	Anxiety
<i>Children's Behavior Checklist, Youth Self Report</i>		Internalizing symptoms Externalizing symptoms
<i>Children's Depression Inventory (CDI)</i>	7 to 17 years	Depression
<i>Multidimensional Anxiety Scale for Children (MASC)</i>	8 to 19 years	Anxiety
<i>Mini-International Neuropsychiatric Interview (MINI) &amp; MINI International Neuropsychiatric Interview for Children and Adolescents (MINI-KID)</i>		Adult psychopathology Child psychopathology Adolescent psychopathology
<i>Minnesota Multiphasic Personality Inventory, Second Edition (MMPI-2)</i> <i>Minnesota Multiphasic Personality Inventory, Adolescent Version (MMPI-A)</i>	≥18 years 14 to 18 years	Adult psychopathology Adolescent psychopathology 10 clinical scales 31 clinical subscales
<i>Personality Assessment Inventory (PAI) &amp; Personality Assessment Inventory, Adolescent Version (PAI-A)</i>	18 to 89 years 12 to 18 years	Adult psychopathology Adolescent psychopathology 22 scales: clinical, treatment, interpersonal
<i>Revised Children's Manifest Anxiety Scale, Second Edition (RCMAS-2)</i>	6 to 19 years	Anxiety
<i>Symptoms Checklist 90-R</i>	≥13 years	Wide range test of psychopathology and psychological symptoms

## Adaptive Functioning

Adaptive functioning may be affected by any number of insults in both children and adults. It is, therefore, essential to examine during a neuropsychological assessment. Some of the commonly used measures of adaptive functioning are rating scales, but there are also several semistructured interviews available, which enable the examiner to gain a more comprehensive understanding of the patient's skills. Measures may assess cognitive and behavioral functioning, as well as the ability to independently perform daily tasks, such as brushing teeth, cooking, and dressing. They may also evaluate the patient's ability to effectively communicate needs and desires. Others include items that assess the patient's ability to function within the community and socialization skills.



TESTS	AGE RANGE	AREAS ASSESSED
<i>Barthel Index</i>	Adults	Activities of daily living
<i>Disability Rating Scale</i>	Older Children and Adults	Assessment of general functioning after brain injury
<i>Functional Assessment Measure</i>	Adults	Cognitive functioning Behavioral functioning Communication Community functioning
<i>Functional Independence Measure</i>	Child Form Adult Form	Independent functioning Activities of daily living
<i>Glasgow Outcome scale</i>	≥16 years	Assessment of general functioning after brain injury
<i>Scales of Independent Behavior, Revised (SIB-R)</i>	Infant to 85 years	Motor skills Broad independence Communication Community living Personal living Problem behaviors
<i>Vineland Adaptive Behavior Scales (VABS)</i>	Birth to 90 years	Communication Activities of daily living Motor skills Maladaptive behaviors Socialization

## Academic Skills

Generally more specific to child assessment, as children are in the process of going to school and learning, academic functioning is an important component of a neurological assessment. Acquired cognitive deficits can have a significant impact on the patient's academic performance. Although higher order reading abilities tend to be more vulnerable to brain injury than basic learning skills, a thorough assessment of reading, writing, and mathematics abilities is recommended (Hanten et al., 2009). For adults, academic achievement may significantly impact their ability to perform basic activities of daily living. Regardless of age, results from a neuropsychological may be able to guide treatment and acquire appropriate supports.

TESTS	AGE RANGE	SKILLS ASSESSED
<i>Wechsler Individual Achievement Test, Second Edition (WIAT-2)</i>	4 to 85 years	Reading Writing Math Oral expression
<i>Wide Range Achievement Test (WRAT)</i>	5 to 64 years	Reading Writing Math
<i>Woodcock Johnson Tests of Achievement, Third Edition (WJ-3)</i>	2 to 90years	Reading Writing Math

## Other Domains

In addition to the principal domains addressed earlier, there are several other areas of functioning that are essential to assess. Neuropsychological assessments are frequently required to determine a patient's readiness to return to work, school, and community living (e.g., independent living and driving), particularly in the rehabilitation setting. Although there are not specific measures available to assess this construct, information gathered from a comprehensive neuropsychological assessment can provide the clinician with information to answer these types of referral questions.

Another area that may need to be assessed is motivation and malingering. These behaviors can be observed in a neuropsychological assessment. Standardized measures, such as the Test of Motivation/Malingering (TOMMS) can be used to assess these constructs; however, behavioral observations and patient interview should also be used to guide interpretation of the testing results.

Finally, as mentioned previously, family functioning can significantly impact outcome after a brain injury. Assessment of family burden and adjustment is essential, as it potentially provides important information about whether intervention is needed and where it should be targeted. In addition to information gathered through the patient interview, there are several standardized scales that assess family functioning. Specifically, the Family Burden of Injury Interview, Family Assessment Device, Impact on Family Scale, and the Family Adaptability and Cohesion Scale are commonly used measures within in the neuropsychological rehabilitation setting to assess these constructs.

## Interventions: Cognitive Rehabilitation and Psychotherapy

Acquired brain injuries and disorders require a multidisciplinary approach to

intervention to address the various cognitive, behavioral, emotional, and physical impairments with which patients may present (Prigatano & Naar-King, 2007). Although a review of current empirically supported approaches is beyond the scope of this chapter, an overview of the guiding principles of rehabilitation and remediation for adults and children will be provided. Cognitive rehabilitation programs typically fall into one of three categories—cognitive, behavioral, or holistic interventions, which integrate cognitive and behavioral approaches with a focus on psychosocial functioning (Butler, 2007). Holistic interventions are multimodal in nature and, therefore, are implemented across the various environments in which a patient functions (Butler, 2007). There are a number of commonly used techniques to address impairments in functioning, which may include the acquisition of compensatory strategies, restoration of skills through practice and exercise, modification of the environment, and the development of metacognition so that individuals can monitor their thinking and improve their ability to problem solve (Andrews, 2005; Butler & Copeland, 2002; Butler & Mulhern, 2005; Farmer, Kanne, Grissom, & Kemp, 2010; Sohlberg & Mateer, 2001; Ylvisaker et al., 2005). Psychotherapy may complement these strategies, as it can improve verbal mediation of tasks, self-control, and problem solving (Butler & Mulhern, 2005). Additionally, psychotherapy can provide strategies to manage emotional reactions to the individual's experience and address family issues (Butler & Mulhern, 2005).

Interventions with adults are targeted to address specific areas of deficit. For individuals with neglect or a related disorder, scanning therapy, selective sensory stimulation, or environmental motor retraining may be implemented (Barrett et al., 2006). Scanning therapy promotes visual scanning of the environment to help the patient orient himself or herself (Butler et al., 2006). The goal of selective sensory stimulation is to improve the perceptual information that is perceived on the side of the body that is neglected (Butler et al., 2006). In environmental-motor remapping, patients respond to feedback produced by the perceived discrepancy between somatic and visual information (Barrett et al., 2006). Although there is no adequate support for specific interventions to improve attention for adults, studies support improvement in complex attentional processes (Cicerone et al., 2005). To address language and communication disorders, studies indicate that practice, cuing, and semantic techniques are effective (Cicerone et al., 2006). Compensatory strategies, visual imagery, and the use of assistive devices, such as alarms, are often used to address memory impairments (Cicerone et al., 2006). Finally, rehabilitation of

executive functioning may incorporate strategies to improve problem solving, self-regulation, and metacognition (Cicerone et al., 2006).

Although there is a dearth of randomized controlled trials that examine the efficacy and effectiveness of rehabilitation and remediation interventions for children, particularly for TBI, there are several interventions that show promise (Limond & Leeke, 2005). One example is the Cognitive Remediation Program (CRP; Butler & Copeland, 2002), which has been developed to improve cognitive and academic functioning in youth who have been treated for cancer. It incorporates repetition and drills; learning of new skills and strategies; and cognitive-behavior therapy to improve memory, attention, self-awareness, and metacognitive skills (Butler, 2007; Butler & Copeland, 2002). Efficacy studies of CRP have found improvements in attention and academic achievement (Butler et al., 2008). Rehabilitation with children also emphasizes school-based interventions (Sohlberg & Mateer, 2001). Youth may receive a 504 Plan or Individualized Education Plan (IEP) to support rehabilitation and remediation of skills. Although a 504 provides the child with academic accommodations, an IEP is a formal arrangement that includes specific guidelines about objectives and goals across a range of areas, as well as details about when and how they will be met (Butler, 2007). Both types of educational plans, however, provide direction about strategies to facilitate learning. This may include the provision of clear instruction and feedback, as well as the opportunity for repetition and practice (Sohlberg & Mateer, 2001). Youth with acquired brain injuries also may require more individualized instruction by a teacher who is trained to work with children with disabilities that impact learning, as they may be better able to provide instruction at the appropriate level (Sohlberg & Mateer, 2001).

## **SUMMARY AND FUTURE DIRECTIONS**

Neuropsychological assessments are an important component of the rehabilitation process and the information obtained can be used by both the neuropsychologist and the rehabilitation team to provide diagnostic information, guide treatment, determine competency, and track development and progress. Neuropsychologists must be familiar with various types of medical disorders and injuries that are frequently seen in this context and how they can impact cognitive, behavioral, emotional, and adaptive functioning. Neuropsychologists' knowledge of the intersection of medical and psychological functioning should inform their choice of measures. They should be intimately aware of the best approach to use to assess different domains of functioning given a patient's

presenting deficits. The results of the neuropsychological assessment should be communicated clearly to the patient and his or her family, as should expectations for improvement and treatment recommendations.

Despite the significant amount of research that has been conducted in recent decades, future research is needed to evolve the practice of neuropsychological assessment in the rehabilitation setting, and to better link assessment and intervention programming. First, as current tests are not particularly sensitive to the mild symptoms patients present, more effective measures to assess dysfunction associated with mTBI are needed (Barrett et al., 2006; Sigurdardottir et al., 2009). Second, neuropsychological assessment should be better integrated into studies examining efficacy of treatments (Mulhern & Butler, 2004). Third, there is a relative paucity of research on neuropsychological assessment in pediatric rehabilitation settings; therefore, further study is much needed. This includes studies that tease apart the unique effects of primary versus secondary brain injury on brain and cognitive development in children. Finally, development of tests to effectively assess functioning in real-world settings is needed. Progress in the area of neuropsychological research within the rehabilitation setting will require the ongoing collaboration of physicians, neuropsychologists, and others involved in the rehabilitation process.

## REFERENCES

- Anderson, V. A., Catroppa, C., Haritou, F., Morse, S., Pentland, L., Rosenfeld, J., & Stargatt, R. (2001). Predictors of acute child and family outcome following traumatic brain injury in children. *Pediatric Neurosurgery*, *34*(3), 138–148.
- Anderson, V., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2005). Functional plasticity or vulnerability after early brain injury? *Pediatrics*, *116*(6), 1374–1382.
- Andrews, K. (2005). Rehabilitation practice following profound brain damage. *Neuropsychological Rehabilitation*, *15*(3–4), 461–472.
- Barker-Collo, S., & Feigin, V. (2006). The impact of neuropsychological deficits on functional stroke outcomes. *Neuropsychology Review*, *16*(2), 53–64.
- Baron, I. S. (2004). *Neuropsychological evaluation of the child*. New York, NY: Oxford University Press.
- Barrett, A. M., Buxbaum, L. J., Coslett, H. B., Edwards, E., Heilman, K. M., Hillis, A. E., . . . Robertson, I. H. (2006). Cognitive rehabilitation interventions for neglect and related disorders: Moving from bench to bedside in stroke patients. *Journal of Cognitive Neuroscience*, *18*(7), 1223–1236.
- Bayley, N. (2006). *Bayley scales of infant and toddler development* (3rd ed.). San Antonio, TX: Harcourt Assessment.
- Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society*, *11*(3), 215–227.
- Bernstein, J. H. (2010). Developmental models in pediatric neuropsychology. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 17–40). New York, NY:

- Cambridge University Press.
- Blakemore, S. J., & Choudhury, S. (2006). Development of the adolescent brain: Implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 47(3–4), 296–312.
- Bode, R. K., Heinemann, A. W., & Semik, P. (2000). Measurement properties of the Galveston Orientation and Amnesia Test (GOAT) and improvement patterns during inpatient rehabilitation. *Journal of Head Trauma Rehabilitation*, 15(1), 637–655.
- Brenner, L. A., Terrio, H., Homaifar, B. Y., Gutierrez, P. M., Staves, P. J., Harwood, J. E., . . . Warden, D. (2010). Neuropsychological test performance in soldiers with blast-related mild TBI. *Neuropsychology*, 24(2), 160–167.
- Brown, L., Sherbenou, R. J., & Johnsen, S. K. (1997). *Test of Nonverbal Intelligence (TONI-3): A language-free measure of cognitive ability*. Austin, TX: Pro-Ed.
- Butler, R. W. (2007). Cognitive rehabilitation. In S. J. Hunter & J. Donders (Eds.), *Pediatric neuropsychological intervention* (pp. 444–464). New York, NY: Cambridge University Press.
- Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review*, 26(1), 17–31.
- Butler, R. W., & Copeland, D. R. (2002). Attentional processes and their remediation in children treated for cancer: A literature review and the development of a therapeutic approach. *Journal of the International Neuropsychological Society*, 8(1), 115–124.
- Butler, R. W., Copeland, D. R., Fairclough, D. L., Mulhern, R. K., Katz, E. R., Kazak A. E., . . . Sahler, O. J. Z. (2008). A multicenter, randomized clinical trial of a cognitive remediation program for childhood survivors of a pediatric malignancy. *Journal of Consulting and Clinical Psychology*, 76(3), 367–378.
- Butler, R. W., & Mulhern, R. K. (2005). Neurocognitive interventions for children and adolescents surviving cancer. *Journal of Pediatric Psychology*, 30(1), 65–78.
- Byars, A. W., deGrauw, T. J., Johnson, C. S., Fastenau, P. S., Perkins, S. M., Egelhoff, J. C., . . . Austin, J. K. (2007). The association of MRI findings and neuropsychological functioning after the first recognized seizure. *Epilepsia*, 48(6), 1067–1074.
- Campbell, T. A., Nelson, L. A., Lumpkin, R., Yoash-Gantz, R. E., Pickett, T. C., & McCormick, C. (2009). Neuropsychological measures of processing speed and executive functioning in combat veterans with PTSD, TBI, and comorbid TBI/PTSD. *Psychiatric Annals*, 39, 797–803.
- Christensen, B. K., Colella, B., Inness, E., Hebert, D., Monette, G., Bayley, M., & Green, R. E. (2008). Recovery of cognitive function after traumatic brain injury: A multilevel modeling analysis of Canadian outcomes. *Archives of Physical Medicine and Rehabilitation*, 89(Suppl. 12), S3–15.
- Ciccia, A. H., Meulenbroek, P., & Turkstra, L. S. (2009). Adolescent brain and cognitive developments: Implications for clinical assessment in traumatic brain injury. *Topics in Language Disorders*, 29, 249–265.
- Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., Kneipp, S., . . . Catanese, J. (2005). Evidence-based cognitive rehabilitation: Updated review of the literature from 1998 through 2002. *Archives of Physical Medicine and Rehabilitation*, 86(8), 1681–1692.
- Cullum, C. M., & Thompson, L. L. (1997). Neuropsychological diagnosis and outcome in mild traumatic brain injury. *Applied Neuropsychology*, 4(1), 6–15.
- Dall’Oglio, A. M., Bates, E., Volterra, V., Di Capua, M., & Pezzini, G. (1994). Early cognition, communication and language in children with focal brain injury. *Developmental Medicine and Child Neurology*, 36(12), 1076–1098.
- Dennis, M., Barnes, M. A., Donnelly, R. E., Wilkinson, M., & Humphreys, R. P. (1996). Appraising and managing knowledge: Metacognitive skills after childhood head injury. *Developmental Neuropsychology*, 12, 77–103.
- Donovan, N. J., Kendall, D. L., Heaton, S. C., Kwon, S., Velozo, C. A., & Duncan, P. W. (2008). Conceptualizing functional cognition in stroke. *Neurorehabilitation and Neural Repair*, 22(2), 122–135.
- Ellenberg, L., Liu, Q., Gioia, G., Yasui, Y., Packer, R. J., Mertens, A., . . . Zeltzer, L. K. (2009).

- Neurocognitive status in long-term survivors of childhood CNS malignancies: A report from the Childhood Cancer Survivor Study. *Neuropsychology*, 23(6), 705–717.
- Elliot, C. D. (2007). *Differential Ability Scales—second edition (DAS-II)*. Minneapolis, MN: Pearson Assessments.
- Ewing-Cobbs, L., Kramer, L., Prasad, M., Canales, D. N., Louis, P. T., Fletcher, J. M., . . . Cheung, K. (1998). Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics*, 102(2 Pt 1), 300–307.
- Ewing-Cobbs, L., Levin, H. S., Fletcher, J. M., Miner, M. E., & Eisenberg, H. M. (1990). The Children's Orientation and Amnesia Test: Relationship to severity of acute head injury and to recovery of memory. *Neurosurgery*, 27(5), 683–691; discussion 691.
- Ewing-Cobbs, L., Thompson, N. M., Miner, M. E., & Fletcher, J. M. (1994). Gunshot wounds to the brain in children and adolescents: Age and neurobehavioral development. *Neurosurgery*, 35(2), 225–233; discussion 233.
- Farmer, J. E., Kanne, S. M., Grissom, M. O., & Kemp, S. (2010). Pediatric neuropsychology in medical rehabilitation settings. In R. G. Frank, M. Rosenthal, & Bruce Caplan (Eds.), *Handbook of rehabilitation psychology* (2nd ed, pp. 315–328.). Washington, DC: American Psychological Association.
- Frank, R. G., & Elliott, T. R. (2000). Rehabilitation psychology: Hope for a psychology of chronic conditions. *Handbook of rehabilitation psychology* (pp. 3–8) Washington, DC: American Psychological Association.
- Giedd, J. N. (2008). The teen brain: Insights from neuroimaging. *Journal of Adolescent Health*, 42(4), 335–343.
- Golden, C. J., Purisch, A. D., & Hammeke, T. A. (1991). *Luria-Nebraska neuropsychological battery (LNNB)*. Los Angeles, CA: WPS/Western Psychological Services.
- Hahn, Y. S., Chyung, C., Barthel, M. J., Bailes, J., Flannery, A. M., & McLone, D. G. (1988). Head injuries in children under 36 months of age. *Child's Nervous System*, 4(1), 34–39.
- Hammill, D. D., Pearson, N. A., & Wiederholt, J. L. (1997). *C-TONI Comprehensive Test of Nonverbal Intelligence: Manual*. Austin, TX: Pro-Ed.
- Hammond, F. M., Hart, T., Bushnik, T., Corrigan, J. D., & Sasser, H. (2004). Change and predictors of change in communication, cognition, and social function between 1 and 5 years after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 19(4), 314–328.
- Hanten, G., Bartha, M., & Levin, H. S. (2000). Metacognition following pediatric traumatic brain injury: A preliminary study. *Developmental Neuropsychology*, 18(3), 383–398.
- Hanten, G., Li, X., Newsome, M. R., Swank, P., Chapman, S. B., Dennis, M., . . . Levin, H. S. (2009). Oral reading and expressive language after childhood traumatic brain injury: Trajectory and correlates of change over time. *Topics in Language Disorders*, 29, 236–248.
- Hertz-Pannier, L., Chiron, C., Jambaqué, I., Renaux-Kieffer, V., Van de Moortele, P. F., Delalande, O., . . . Le Bihan, D. (2002). Late plasticity for language in a child's non-dominant hemisphere: A pre-and post-surgery fMRI study. *Brain*, 125(Pt 2), 361–372.
- Hotz, G., & Chapman, S. B. (2009). Brain-based assessment and treatment in pediatric brain injury. *Topics in Language Disorders*, 29, 204–206.
- Inagaki, M., Yoshikawa, E., Matsuoka, Y., Sugawara, Y., Nakano, T., Akechi, T., . . . Uchitomi, Y. (2007). Smaller regional volumes of brain gray and white matter demonstrated in breast cancer survivors exposed to adjuvant chemotherapy. *Cancer*, 109(1), 146–156.
- Jacobs, M. L., & Donders, J. (2008). Performance discrepancies on the California Verbal Learning Test: Second Edition (CVLT-II) after traumatic brain injury. *Archives of Clinical Neuropsychology*, 23(1), 113–118.
- Jacobs, R., Harvey, A. S., & Anderson, V. (2007). Executive function following focal frontal lobe lesions: Impact of timing of lesion on outcome. *Cortex*, 43(6), 792–805.
- Jankovic, M., Brouwers, P., Valsecchi, M. G., Van Veldhuizen, A., Huisman, J., Kamphuis, R., . . .

- Ferronato, L. (1994). Association of 1800 cGy cranial irradiation with intellectual function in children with acute lymphoblastic leukaemia. ISPACC. International Study Group on Psychosocial Aspects of Childhood Cancer. *Lancet*, 344(8917), 224–227.
- Johnston, M. V. (2009). Plasticity in the developing brain: Implications for rehabilitation. *Developmental Disabilities Research Reviews*, 15(2), 94–101.
- Jokinen, H., Kalska, H., Mäntylä, R., Pohjasvaara, T., Ylikoski, R., Hietanen, M., . . . Erkinjuntti, T. (2006). Cognitive profile of subcortical ischaemic vascular disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, 77(1), 28–33.
- Kaufman, A. S., & Kaufman, N. L. (1990). *Kaufman Brief Intelligence Test: KBIT*. Circle Pines, MN: AGS/American Guidance Service.
- Kinsella, G., Prior, M., Sawyer, M., Murtagh, D., Eisenmajer, R., Anderson, V., . . . Klug, G. (1995). Neuropsychological deficit and academic performance in children and adolescents following traumatic brain injury. *Journal of Pediatric Psychology*, 20(6), 753–767.
- Knopman, D. S., Roberts, R. O., Geda, Y. E., Boeve, B. F., Pankratz, V. S., Cha, R. H., . . . Petersen, R. C. (2009). Association of prior stroke with cognitive function and cognitive impairment: A population-based study. *Archives of Neurology*, 66(5), 614–619.
- Kolb, B. (1995). *Brain plasticity and behavior*. Mahwah, NJ: Lawrence Erlbaum.
- Kolb, B., & Whishaw, I. Q. (2008). *Fundamentals of human neuropsychology (6th ed.)*. New York, NY: Worth.
- Laatsch, L. K., Thulborn, K. R., Krisky, C. M., Shobat, D. M., & Sweeney, J. A. (2004). Investigating the neurobiological basis of cognitive rehabilitation therapy with fMRI. *Brain Injury*, 18(10), 957–974.
- Larson, R. G. (1999). *The structure and rheology of complex fluids*. New York, NY: Oxford University Press.
- Léon-Carrión, J., Taaffe, P. J., & Barroso y Martin, J. M. (2006). Neuropsychological assessment of persons with acquired brain injury. In J. Léon-Carrión, K. R. H. von Wild, & G. A. Zitnay (Eds.), *Brain injury treatment: Theories and practices* (pp. 275–312) New York, NY: Taylor & Francis.
- Levin, H. S., O'Donnell, V. M., & Grossman, R. G. (1979). The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *Journal of Nervous and Mental Disease*, 167(11), 675–684.
- Limond, J., & Leeke, R. (2005). Practitioner review: Cognitive rehabilitation for children with acquired brain injury. *Journal of Child Psychology and Psychiatry*, 46, 339–352.
- Loring, D. W. (1999). *INS Dictionary of Neuropsychology*. New York, NY: Oxford University Press.
- Lucas, J. A. (1998). Traumatic brain injury and postconcussive syndrome. In P. J. Snyder & P. D. Nussbaum (Eds.), *Clinical neuropsychology: A pocket handbook for assessment* (pp. 243–265). Washington, DC: American Psychological Association.
- Lucas, J. A. (2003). Traumatic brain injury and postconcussive syndrome. In P. J. Snyder & P. D. Nussbaum (Eds.), *Clinical neuropsychology*. Washington, DC: American Psychological Association.
- Meyers, C. A., Albitar, M., & Estey, E. (2005). Cognitive impairment, fatigue, and cytokine levels in patients with acute myelogenous leukemia or myelodysplastic syndrome. *Cancer*, 104(4), 788–793.
- Meyers, C. A., Byrne, K. S., & Komaki, R. (1995). Cognitive deficits in patients with small cell lung cancer before and after chemotherapy. *Lung Cancer*, 12(3), 231–235.
- Mosch, S. C., Max, J. E., & Tranel, D. (2005). A matched lesion analysis of childhood versus adult-onset brain injury due to unilateral stroke: Another perspective on neural plasticity and recovery of social functioning. *Cognitive and Behavioral Neurology*, 18(1), 5–17.
- Mulhern, R. K., & Butler, R. W. (2004). Neurocognitive sequelae of childhood cancers and their treatment. *Pediatric Rehabilitation*, 7(1), 1–14; discussion 15.
- Mullen, E. M. (1995). *Mullen scales of early learning* (AGS edn). Circle Pines, MN: American Guidance Service.
- Myers, J. S. (2009). Chemotherapy-related cognitive impairment. *Clinical Journal of Oncology Nursing*, 13(4), 413–421.



- Nøkleby, K., Boland, E., Bergersen, H., Schanke, A. K., Farner, L., Wagle, J., & Wyller, T. B. (2008). Screening for cognitive deficits after stroke: A comparison of three screening tools. *Clinical Rehabilitation*, 22(12), 1095–1104.
- Novack, T. A., Dowler, R. N., Bush, B. A., Glen, T., & Schneider, J. J. (2000). Validity of the Orientation Log, relative to the Galveston Orientation and Amnesia Test. *Journal of Head Trauma Rehabilitation*, 15(3), 957–961.
- Prigatano, G. P., & Naar-King, S. (2007). Neuropsychological rehabilitation of school-age children: An integrated team approach to individualized interventions. In S. J. Hunter & J. Donders (Eds.), *Pediatric neuropsychological intervention* (pp. 465–475). New York, NY: Cambridge University Press.
- Rath, J. F., Langenbahn, D. M., Simon, D., Sherr, R. L., Fletcher, J., & Diller, L. (2004). The construct of problem solving in higher level neuropsychological assessment and rehabilitation. *Archives of Clinical Neuropsychology*, 19(5), 613–635.
- Roid, G. H. (2003). *Stanford-Binet Intelligence Scales (SB5)*. Rolling Meadows, IL: Riverside.
- Sarajuuri, J. M., & Koskinen, S. K. (2006). Holistic neuropsychological rehabilitation in Finland: The INSURE program—a transcultural outgrowth of perspectives from Israel to Europe via USA. *International Journal of Psychology*, 41, 362–370.
- Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed.). San Diego, CA: Jerome M. Sattler.
- Sattler, J. M., & D’Amato, R. (2002). Brain injuries: Formal batteries and informal measures. In J. M. Sattler (Ed.), *Assessment of children: Behavioral and clinical applications* (4th ed., pp. 440–469). San Diego, CA: Jerome M. Sattler.
- Schaefer, M., Engelbrecht, M. A., Gut, O., Fiebich, B. L., Bauer, J., Schmidt, F., . . . Lieb, K. (2002). Interferon alpha (IFNalpha) and psychiatric syndromes: A review. *Progress in Neuro-psychopharmacology & Biological Psychiatry*, 26(4), 731–746.
- Shuper, A., Stark, B., Kornreich, L., Cohen, I. J., Aviner, S., Steinmetz, A., . . . Yaniv, I. (2000). Methotrexate treatment protocols and the central nervous system: Significant cure with significant neurotoxicity. *Journal of Child Neurology*, 15(9), 573–580.
- Sigurdardottir, S., Andelic, N., Roe, C., & Schanke, A. K. (2009). Cognitive recovery and predictors of functional outcome 1 year after traumatic brain injury. *Journal of the International Neuropsychological Society*, 15(5), 740–750.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach*. New York, NY: Guilford Press.
- Strauss, E., Sherman, E. M., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd edn). New York, NY: Oxford University Press.
- Taylor, H. G., Yeates, K. O., Wade, S. L., Drotar, D., Klein, S. K., & Stancin, T. (1999). Influences on first-year recovery from traumatic brain injury in children. *Neuropsychology*, 13(1), 76–89.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81–84.
- Temkin, N. R., Machamer, J. E., & Dikmen, S. S. (2003). Correlates of functional status 3–5 years after traumatic brain injury with CT abnormalities. *Journal of Neurotrauma*, 20(3), 229–241.
- Tranel, D., & Eslinger, P. J. (2000). Effects of early onset brain injury on the development of cognition and behavior: Introduction to the special issue. *Developmental Neuropsychology*, 18(3), 273–280.
- Vanderploeg, R. D., Curtiss, G., & Belanger, H. G. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society*, 11(3), 228–236.
- Wechsler, D. (1999). *Wechsler Abbreviated Test of Intelligence*. San Antonio, TX: Psychological Corporation.
- Wechsler, D. (2002). *Wechsler Preschool and Primary Scale of Intelligence* (3rd ed.). San Antonio, TX: Harcourt Assessment.
- Wechsler, D. (2003). *WISC-IV: Administration and Scoring Manual*. San Antonio, TX: Psychological

- Corporation.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale, Fourth Edition*. San Antonio, TX: Psychological Corporation.
- Wechsler, D. (2009a). *Wechsler Individual Achievement Test* (3rd edn). San Antonio, TX: NCS Pearson.
- Wechsler, D. (2009b). *Wechsler Memory Scale—fourth edition (WMS-IV)*. Minneapolis, MN: Pearson Assessments.
- Wefel, J. S., Lenzi, R., Theriault, R., Buzdar, A. U., Cruickshank, S., & Meyers, C. A. (2004). “Chemobrain” in breast carcinoma?: A prologue. *Cancer*, *101*(3), 466–475.
- Wefel, J. S., Witgert, M. E., & Meyers, C. A. (2008). Neuropsychological sequelae of non-central nervous system cancer and cancer therapy. *Neuropsychology Review*, *18*(2), 121–131.
- Weis, J., Poppelreuter, M., & Bartsch, H. H. (2009). Cognitive deficits as long-term side-effects of adjuvant therapy in breast cancer patients: “Subjective” complaints and “objective” neuropsychological test results. *Psycho-oncology*, *18*(7), 775–782.
- Williamson, J. (2010). A lifespan review of developmental neuroanatomy. In J. Donders & S. J. Hunter (Eds.), *Principles and practice of lifespan developmental neuropsychology* (pp. 3–16). New York, NY: Cambridge University Press.
- Wilson, B. A., Gracey, F., Evans, J. J., & Bateman, A. (2009). Towards a comprehensive model of neuropsychological rehabilitation. *Neuropsychological rehabilitation: Theory, models, therapy and outcome* (pp. 1–21). New York, NY: Cambridge University Press.
- Wilson, B. A., Rous, R., & Sopena, S. (2008). The current practice of neuropsychological rehabilitation in the United Kingdom. *Applied Neuropsychology*, *15*(4), 229–240.
- Yeates, K. O. (2000). Closed head injury. In K. O. Yeates, M. D. Ris, & H. G. Taylor (Eds.), *Pediatric neuropsychology: Research, theory, and practice* (pp. 92–116). New York, NY: Guilford Press.
- Yeates, K. O., Taylor, H. G., Barry, C. T., Drotar, D., Wade, & Stancin, T. (2001). Neurobehavioral symptoms in childhood closed-head injuries: Changes in prevalence and correlates during the first year postinjury. *Journal of Pediatric Psychology*, *26*, 79–91.
- Ylvisaker, M., Adelson, P. D., Braga, L. W., Burnett, S. M., Glang, A., Feeney, T., . . . Todis, B. (2005). Rehabilitation and ongoing support after pediatric TBI: Twenty years of progress. *Journal of Head Trauma Rehabilitation*, *20*(1), 95–109.

# Utilization of Neuroimaging in Rehabilitation

*Shawn D. Gale and Ramona O. Hopkins*

Neuroimaging is widely used in neurological disorders and brain injury to assist in clinical diagnosis. Neuroimaging is also used to detect lesions and assess the extent of neural damage that can be correlated with patient outcomes such as cognitive and psychiatric outcomes in a variety of neurological disorders and injuries. However, the contributions of neuroimaging to guide rehabilitation and recovery are more limited. The overarching aim of this chapter is to address the contributions that neuroimaging provides to the field of rehabilitation. Given the complexity of neuroimaging and rehabilitation, the focus of this chapter will be on the synergy between the two, rather than an in-depth review of either field. Certainly there are entire tomes, including the edited volume in which this chapter appears, devoted to very specific topics in each field. To this end, the technical details regarding neuroimaging techniques will only receive the briefest mention. Similarly, there are many neurological disorders and injuries that are amenable to neuroimaging techniques, but we simply do not have the space to cover all of them. Thus, the major focus in this chapter will be neuroimaging as it relates to rehabilitation in traumatic brain injury (TBI), cerebral vascular accidents (CVA), and anoxia. Finally, although it would be interesting to also include in-depth information on the unique challenges in the pediatric population, we simply cannot do justice to this large and complex area with given space limitations. In this regard, we refer interested individuals to texts specific to this field, such as *Pediatric Traumatic Brain Injury: New Frontiers in Clinical and Translational Research* edited by [Anderson and Yeates](#)

(2010) and *Treating Neurodevelopmental Disabilities: Clinical Research and Practice* edited by Farmer, Donders, and Warschausky (2005).

## STRUCTURAL NEUROIMAGING

### CT and MRI

The two most common forms of imaging used to evaluate the structural integrity of the cerebrum are computed tomography (CT) and magnetic resonance imaging (MRI). Both CT and MRI are effective methods that are used to identify and localize lesions in a variety of neurological disorders and following neural trauma (Flanagan, Cantor, & Ashman, 2008). These are the frontline imaging modalities used daily in clinical practice and each has unique strengths and weaknesses. CT scans are based on X-ray technology, and therefore use radiation to obtain data. Because X-ray data is based on information collected in sections, it is referred to as tomography. These sectional data are then reconstructed with the assistance of a computer (i.e., computer-assisted tomography [CAT]) to provide usually two-dimensional (2D) and, more recently, three-dimensional (3D) images. CT is particularly good at identifying bone and bone damage (i.e., skull fractures), as well as hemorrhage or bleeding. CT is also used to identify evidence of gross neuropathological changes such as mass effect, midline shift, or ventricular collapse or enlargement, which might be secondary to such things as edema, hemorrhage, tumors, or the presence of foreign objects (i.e., bullets, shrapnel) or bone fragments. Acute brain injuries on CT appear as areas of either hyperintense (increased) or hypointense (decreased) signal (Little, Kraus, Jiam, et al., 2010). Thus, CT is extremely important in cerebral trauma, where the initial goal is to determine diagnosis and establish whether emergent neurosurgical intervention is warranted. In addition, CT also has the advantage of being less expensive, is much more resistant to motion artifact than MRI, and can be used when placing the patient in a magnetic field would be contraindicated, such as in the presence of metal fragments or pacemaker (Ketonen & Berg, 1997).

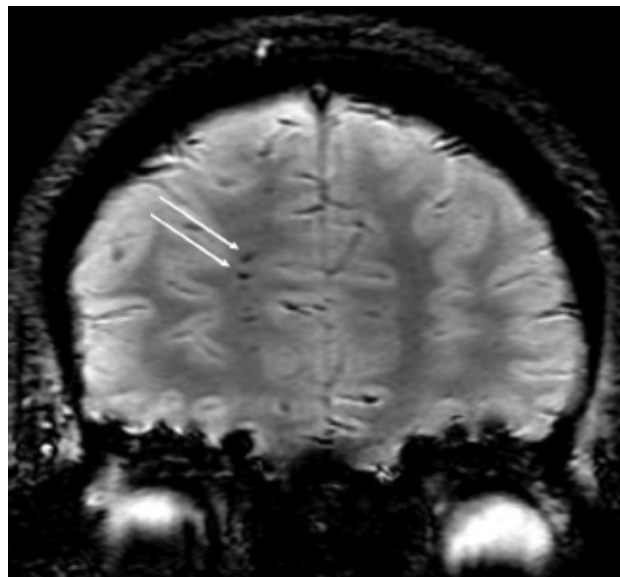
MRI is based on the fact that atoms resonate or precess when flooded with energy via radio waves, which can be accurately measured in the presence of an applied magnetic field (Mori & Barker, 1999). As a portion of the atoms align their rotations within that magnetic field and release energy as they return to their normal state, the energy is detected as a change within the magnetic field. This energy release or change is quantitatively different for different types of

tissue/substances. For example, in the brain, white matter, grey matter, and cerebrospinal fluid (CSF) are differentiated, as these consist of differing amounts of water and fat. For a review of MRI physics (and functional magnetic resonance imaging [fMRI]) see [Matthews \(2001\)](#).

MRI has better spatial resolution than CT and through the utilization of different scanning protocols or sequences various aspects of tissues can be emphasized in order to differentiate among different lesion types, as well as stages of lesion evolution or resolution. For example, following hemorrhage, hemoglobin breaks down. There are various stages of hemoglobin breakdown that are time dependent ([Bradley, 1993](#)). For example, oxyhemoglobin is present in the hyperacute stage (<24 hours), deoxyhemoglobin is present in the acute stage (1–3 days), methemoglobin can occur early (>3 days) or later (>7 days), and, finally, hemosiderin is present in the chronic stage (>14 days). Hemosiderin can be detected using certain MR sequences such as gradient recalled echo (GRE). Thus, identification of the stages of hemorrhage gives both spatial information (i.e., which area of the brain was injured) as well as temporal information (i.e., type of hemoglobin present) in that its appearance and subsequent disappearance are time dependent. Thus, radiological investigation can typically determine whether a given lesion is acute, subacute, or chronic, which provides important information when combined with the patient's clinical history. Different MRI sequences have implications for treatment of various disorders in terms of etiology or differential diagnosis (i.e., hemorrhage can be parenchymal, epidural, subdural, subarachnoid, or intraventricular), and evolution or resolution over time in response to either specific treatments or spontaneous recovery. Using a trauma example, MRI is much more likely to find lesions not present on CT in terms of both quantity and quality ([Duckworth & Stevens, 2010](#)). For example, [Figure 13.1](#) shows a coronal GRE sequence obtained in a young man following mild head trauma with brief loss of consciousness. Initial CT was negative, and he was diagnosed with concussion. However, he continued to have difficulty and high-resolution MRI was obtained 7 weeks postinjury, including a GRE sequence that identified several small foci in the right frontal lobe (image is in radiologic orientation with right hemisphere appearing on the left) consistent with remote hemorrhagic shear injury. Unfortunately, GRE sequences may not be obtained in many clinical cases when there is no reason to suspect hemorrhage, as in the case just illustrated ([Wardlaw & Statham, 2000](#)). For a review of the implications of mild head trauma and postconcussion syndrome see [Prigatano and Gale \(2011\)](#).

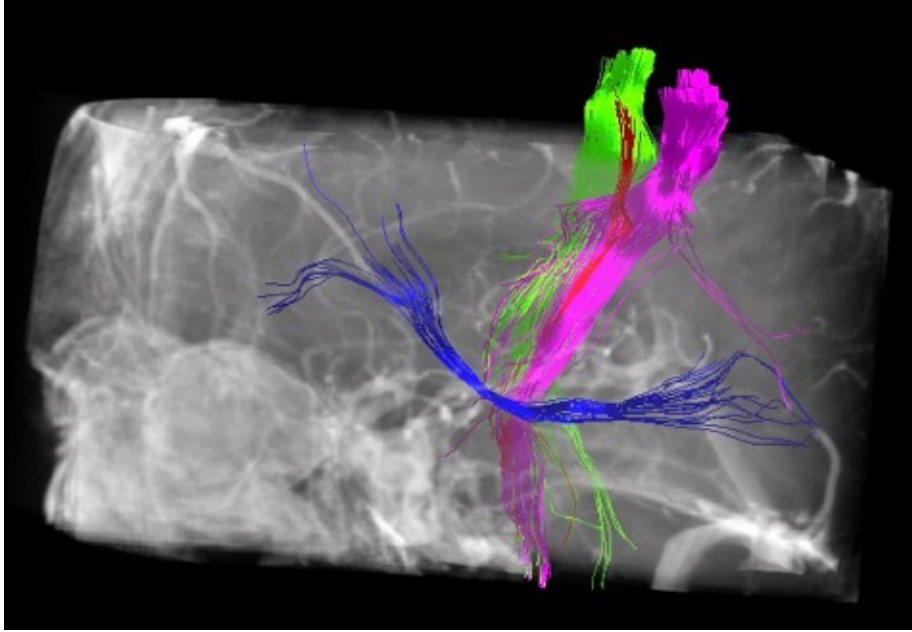
## DWI and DTI

Diffusion weighted imaging (DWI) was first developed in the 1980s (e.g., see [Taylor & Bushell, 1985](#)) and has been used extensively in acute stroke because DWI is very sensitive to acute ischemia ([Ueda et al., 1999](#)). DWI is based on the idea that water molecules are more likely to disperse or diffuse at equal rates in all directions (i.e., isotropically, or in a sphere) unless there is structure (e.g., axons), which causes unequal or directional (anisotropic, or in an ellipsoid) diffusion. Thus, water molecules will diffuse rapidly along a bundle of axons and much more slowly perpendicular to those axons ([Basser, Mattiello, & LeBihan, 1994](#)). More recently, diffusion tensor imaging (DTI) has been used to not only identify areas of acute ischemia, but also to evaluate the integrity of white matter tracts. Observing increased diffusion or isotropy in an area of bundled axons suggests compromised neuronal integrity; specifically, structurally compromised white matter tracts ([Ulug, Moore, Bojko, & Zimmerman, 1999](#)). There are a variety of ways to measure and characterize the amount of diffusion using DTI, including fractional anisotropy (FA), radial diffusivity (RD), and calculating an apparent diffusion coefficient (ADC). In order to calculate a tensor, ADC in at least six directions must be obtained ([Mukherjee, Berman, Chung, Hess, & Henry, 2008](#)).

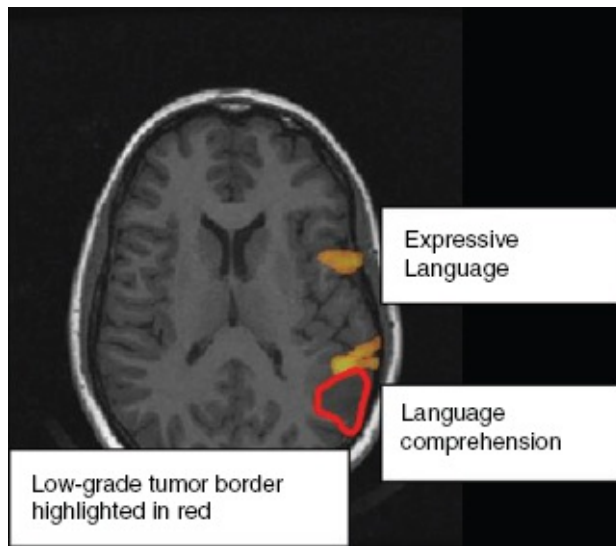


**FIGURE 13.1** Coronal GRE sequence demonstrating hemosiderin deposits in the frontal lobe (arrows) representing traumatic shear injury secondary to head trauma. This young man had brief loss of consciousness, initial CT was negative, and he was diagnosed with concussion. This MRI was obtained 7 weeks postinjury.

DTI can also be used to track neuronal fiber pathways and their interconnections (Conturo et al., 1999). For example, DTI can inform the neurosurgeon regarding how a brain tumor might affect white matter tracts via edema, infiltration, displacement, or disruption. In this sense, DTI can identify the preferred pathway to the neurosurgical target, reducing the amount of surgical damage, and help determine which white matter tracts have or have not been compromised prior to surgery (Yen et al., 2009). A case in point, Figure 13.2 shows an individual who was imaged prior to neurosurgical intervention for neoplasm. In this figure, red represents motor fibers related to the tongue, purple represents finger motor tracts, green represents toe motor tracts, and blue represents the arcuate fasciculus, which is the white matter pathway that connects Wernicke's and Broca's language areas. Even though DTI may identify which tracts may or may not be affected, it is common for atypical cerebral organization to occur, or at least displacement of cognitive functions associated with eloquent cortex in the context of brain tumor. Figure 13.3 shows an individual with a neoplasm (tumor) and the fMRI shows activity in areas of expressive language and language comprehension. Note that the area of the brain associated with language comprehension is on the border of the low-grade tumor. The information obtained via fMRI, in this case, has several implications related to treatment planning. Given the proximity to a brain area associated with language and type of tumor, the patient may decide to have gamma knife (radiosurgery), rather than traditional surgery. Additionally, in traditional surgery, tissue on the margins of tumors is usually also resected to decrease the potential of tumor recurrence, but, in this case, a particularly conservative approach may be warranted. Finally, this information can be used to inform the patient of potential negative cognitive effects of tumor removal. Coupled with fMRI-based cognitive testing, neuropsychologists can now map cortical functioning to minimize postsurgical neurocognitive impairment. Without the accompanying fMRI-based localization of critical areas of cognitive function (e.g., language, motor), the DTI data may be of limited use in determining patients' cognitive outcomes. It should be remembered that individual brains, to at least some degree, have individualized organization (Bigler & Wilde, 2010). Furthermore, from a clinical standpoint, atypical neural organization in neurologic disease appears to be common, suggesting a greater need for functional imaging to be coupled with structural and DTI data to inform treatment decisions.



**FIGURE 13.2** DTI tracking of motor pathways in a patient with brain tumor. In this figure, red represents motor fibers related to tongue, purple represents finger, green represents toe, and blue represents the arcuate fasciculus connecting Wernicke's and Broca's areas. *Source:* Provided by Leslie Baxter. Used with permission.



**FIGURE 13.3** The fMRI study in a patient with brain tumor. This figure is in radiologic orientation (left hemisphere is on the right). Brain activation on measures of expressive language and language comprehension appear in orange. A low-grade tumor is outlined in red and is on the border of the area activated on a language comprehension task.

*Source:* Provided by Leslie Baxter. Used with permission.



## FUNCTIONAL NEUROIMAGING

In addition to structural brain imaging, as mentioned earlier, functional imaging techniques are used both clinically and for research purposes to evaluate the integrity of the brain functioning rather than assess structural integrity. Functional imaging techniques include positron emission tomography (PET), single-photon emission computed tomography (SPECT), magnetic resonance spectroscopy (MRS), fMRI, electroencephalography (EEG), magnetoencephalography (MEG), and perfusion-related protocols for both MRI and CT. Functional imaging provides information about neural function, and it is able to detect functional abnormalities associated with neural injury (Jantzen, Anderson, Steinberg, & Kelso, 2004). Entire books have been written on these constantly evolving brain mapping techniques, making a comprehensive review beyond the scope of this chapter. Many excellent review articles have been written on functional imaging. For example, an article by Eliassen *et al.* (2008) reviews the preceding functional imaging techniques as applied to stroke rehabilitation. Functional imaging methods differ in the type of information they provide about the brain, as well as the underlying mechanisms involved in data acquisition. Each functional technique differs in spatial and temporal resolution. Table 13.1 is adapted from Carey and Seitz (2007b) and Matthews (2001) and summarizes the spatial and temporal characteristics of the functional imaging methods.

**TABLE 13.1 Summary of Temporal and Spatial Resolution of Various Neuroimaging Techniques**

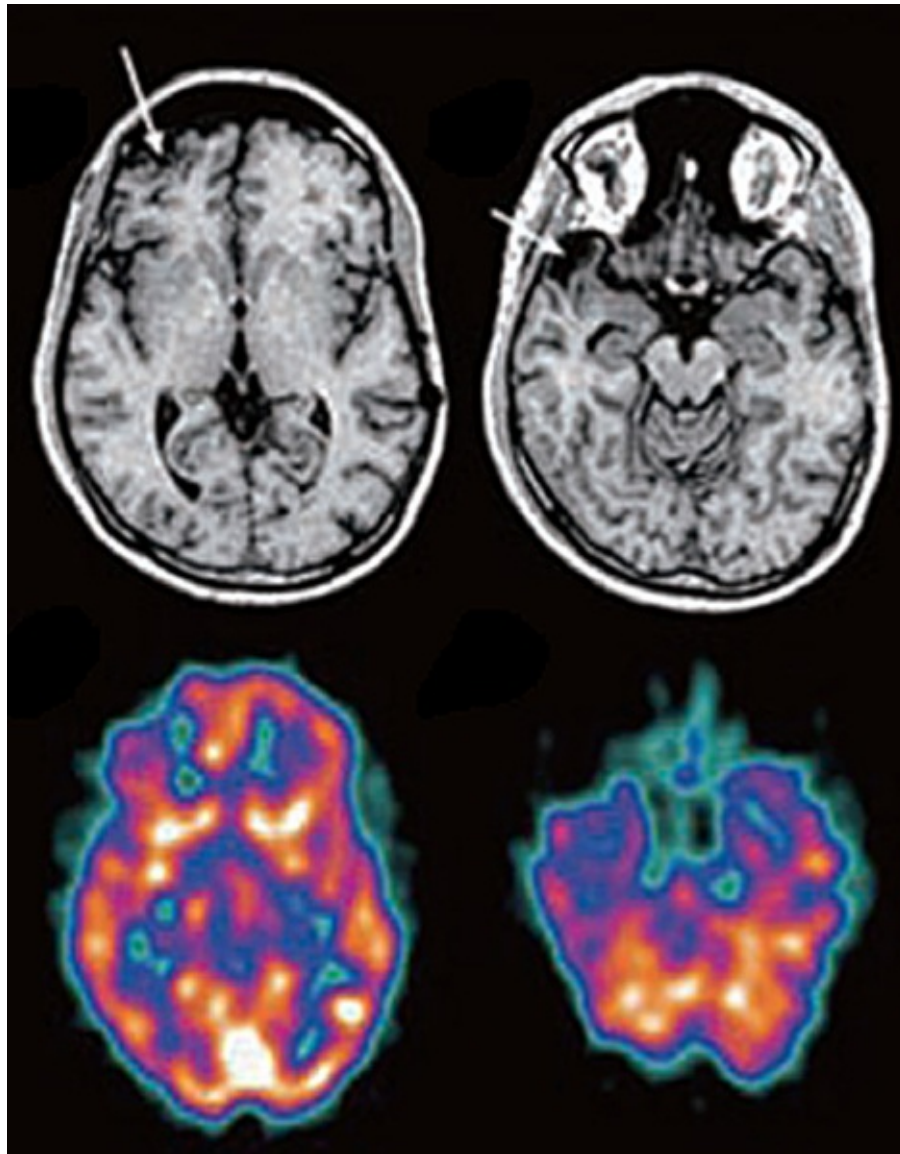
TECHNIQUE	TEMPORAL RESOLUTION	SPATIAL RESOLUTION	ADVANTAGES AND DISADVANTAGES
CT	Low	Medium	Anatomic, bone, acute blood, radiation
MRI	Low	High	Anatomic, high resolution, neuropathology, no radiation
fMRI	Medium	Low	Function, cognition, noninvasive, postprocessing takes much time
PET	Low	Low	Function, metabolism, receptors, transmitters, cognition, invasive
EEG	High	Low	Electrical activity
MEG	High	Medium	Electrical activity, better spatial resolution than EEG

CT: computed tomography; MRI: magnetic resonance imaging; fMRI: functional magnetic resonance imaging; PET: positron emission tomography; EEG: electroencephalography; MEG: magnetoencephalography

Source: Adapted from Carey and Seitz (2007b) and Matthews (2001).

## PET and SPECT

PET and SPECT are nuclear medicine imaging modalities that involve intravenous administration of radioactive isotopes (tracers) that are measured as they are taken up by various cerebral structures. One method of transporting a tracer is by attaching it to a molecule that is biologically utilized in the brain, such as using fluorodeoxyglucose (FDG), which combines with glucose. Lower uptake of FDG in some cerebral regions compared to others suggests hypometabolism in those regions, likely reflecting abnormal neuronal functioning. Metabolism is not the only function that can be measured; neurotransmitters, among other things, can also be radioactively labeled. Although PET and SPECT have lower spatial resolution, they can be mapped onto, or coregistered, with more traditional structural imaging techniques such as CT. Of considerable interest to the field of rehabilitation is the fact that methods such as PET can identify abnormalities in tissue that may “appear normal” on standard structural MRI or CT imaging. Furthermore, even when a specific lesion is identified, it is often the case that the area of dysfunctional tissue is usually much larger than the observed lesion. [Figure 13.4](#) offers an example of both structural (MRI) and functional (SPECT) data obtained at the same time in a patient with a history of TBI. Arrows correspond to regions of frontal (left) and temporal (right) atrophic changes. Note on the SPECT, darker colors represent lower baseline metabolism, that the areas of hypometabolism are larger than suggested on conventional MRI. Finally, in addition to identifying the cellular makeup and functions of various cerebral regions, PET can be used to investigate cognitive function, as areas that change in response to a cognitive demand are likely to be involved in the carrying out of that cognitive function. Neuronal function that can be studied includes cerebral blood flow, site-specific metabolic activity, and even site-specific oxygen utilization. The main difference between PET and SPECT is in the ligands used, as well as the method of detecting the radioactive tracers.



**FIGURE 13.4** Axial MRI (top row) and SPECT (bottom row) images in a patient with remote TBI. The MRI shows trauma-related encephalomalacic changes and the SPECT suggest the area of dysfunctional frontal and temporal tissue is larger than suggested by MRI.

### EEG and MEG

In contrast to PET and SPECT techniques that indirectly measure neuronal activity, EEG and MEG directly measure the electrophysiological activity of neurons. Because electrophysiological activity is measured through electrodes placed on the scalp, some distance from the cortical surface, the obtained spatial information of neuronal firing is limited. The temporal information of EEG and MEG is quite good. In addition to measuring passive electrical activity with EEG, a stimulus can be presented which is then correlated temporally with the

EEG activity. This method of “evoking” a change in the EEG pattern is referred to as evoked potentials (EP). Similarly, more complicated behavioral tasks can be performed and electrical activity measured, resulting in an event-related potential (ERP). Rather than measure the electrical activity of groups of neurons, MEG actually measures the magnetic fields that are generated by these currents. MEG has better spatial resolution than EEG.

## MRS

Proton magnetic resonance spectroscopy ( $^1\text{H}$  MRS), or simply MRS, is an MRI method that evaluates aspects of the neuronal structure and neurochemical function of cerebral tissue. MRS assesses biologic markers that represent aspects of neuronal and glial integrity or dysfunction, metabolites, neurotransmitters, and even biomarkers typically only seen following injury. Common biomarkers used in MRS include the neuronal marker N-acetyl aspartate (NAA); myoinositol, a glial marker; neurotransmitters glutamate and glutamine; energy metabolites lactate and creatine (Cre); and membrane markers such as choline (Cho; Babikian et al., 2006). In MRS analyses, single or multiple voxels are selected from predetermined regions of interest with many studies using ratios (e.g., NAA/Cre) to determine the integrity of a particular brain region. For example, Babikian et al. (2006) found that regional NAA/Cre ratios correlated with cognitive function after pediatric TBI. For a review of MRS and its use in hypoxicischemic injury see Little, Kraus, Jiam, et al. (2010).

## fMRI

The fMRI takes advantage of the physiologic fact that the magnetic characteristics of hemoglobin are different depending on whether hemoglobin is oxygenated or deoxygenated. Briefly, fMRI capitalizes on the fact that neuronal activity is linked to hemodynamic changes and active neurons utilize more oxygen resulting in greater concentration of deoxyhemoglobin, which is paramagnetic and thus causes distortions in the applied magnetic field (Ogawa, Lee, Kay, & Tank, 1990). This effect is referred to as blood-oxygenation-level-dependent (BOLD) contrast. For a detailed account of this technique, from physics and physiology to experimental design and interpretation of data, we suggest *Functional MRI: An Introduction to Methods* edited by Jezzard, Matthews, and Smith (2001).

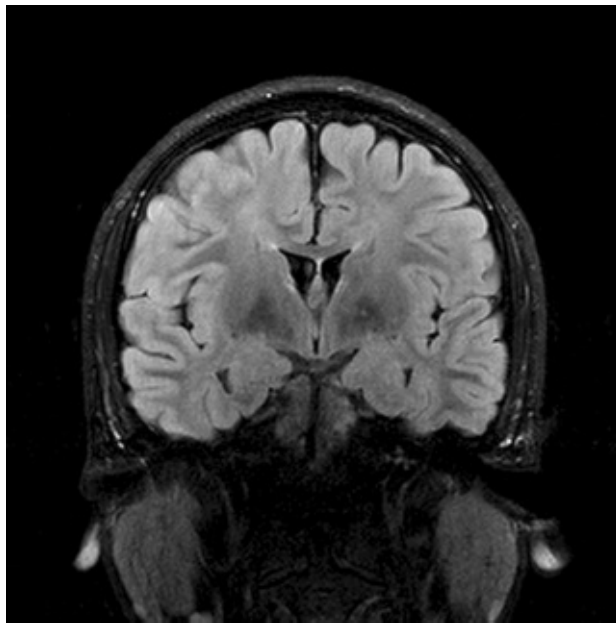
As will be discussed in this chapter, the multimodal acquisition of

information (multiple imaging modalities in conjunction with neuropsychological assessment) may provide the best clinical information in neurological disorders and brain injury. For example, certain conditions such as epilepsy, even if refractory, may present with either normal or abnormal EEG, normal or abnormal MRI, and normal or abnormal PET; the various combinations of these results have implications for diagnosis, treatment (particularly for surgical intervention), and outcome, including rehabilitation. Neuropsychology has a very important role in evaluating diagnosis, management, recovery, and rehabilitation in patients with neurological disorders and injuries.

### **DIFFERENTIAL DIAGNOSIS AND DEFINING THE LESION/PATHOLOGY: NEUROIMAGING AS A BIOMARKER OF INJURY**

Although it is clear that MRI, for example, can be extremely useful in identifying the pathology associated with a given condition such as TBI, it is important for the clinician to understand the implications of this information, which may inform the approach to evaluation, management, and treatment. Furthermore, neuroimaging can help elucidate the evolving role of the lesion as it relates to the brain–behavior relationships. For example, basal ganglia lesions and neuropsychological impairments are common following anoxic brain injury, including carbon monoxide (CO) poisoning. Lesions of the basal ganglia ([Figure 13.5](#)) are associated with cognitive impairments in memory, executive function, visual–spatial deficits, and generalized cognitive decline. For example, six patients with CO poisoning who underwent MRI at multiple time points following CO poisoning showed marked variability in lesion presentation ([Hopkins, Fearing, Weaver, & Foley, 2006](#)). Of the six patients, two patients had normal initial post-CO scans and one patient developed bilateral “Swiss cheese” basal ganglia lesions on MRI at 2 weeks that remained present 6 months later. The second patient had perfusion defects in the basal ganglia at 4 months. Three of the patients had bilateral globus pallidus lesions on initial scans that remained present 15 days and 1 year later. Bilateral globus pallidus lesions were observed on MRI at 2.5 years, 5 years post-CO poisoning in two patients. One patient had a left globus pallidus lesion on initial brain CT but had a normal MRI 1.5 years post-CO poisoning. Thus, some patients had lesions immediately following CO poisoning, whereas other patients did not. The lesions varied in presentation and included bilateral globus pallidus, unilateral globus pallidus, or bilateral small

lesions with the appearance of Swiss cheese. Four patients' lesions persisted months to years following CO poisoning; however, one patient's lesions resolved 1.5 years later. The above findings suggest marked variability in onset, location, and presentation of basal ganglia lesions following CO poisoning (Hopkins, Fearing, et al., 2006).



**FIGURE 13.5** DTI sequence showing bilateral globus pallidus lesions following carbon monoxide poisoning.

*Source:* Provided by Erin D. Bigler. Used with permission.

There are times when imaging may fail to identify a lesion or damage in contrast to clinical observations, such as cognitive impairments. That is, structural neuroimaging is normal, but patients have cognitive impairments on neuropsychological testing and/or changes in neurobehavioral status. In some cases, such as in the instance of [Figure 13.1](#), there was a suggestion that initial neuroimaging may not have captured the true nature of the injury; the neurobehavioral and neurocognitive difficulties were beyond what would have been expected, given the initial estimates of severity of injury. In this case, fortunately, additional neuroimaging studies were completed several weeks later, which clarified the extent of injury of the initial trauma. Taken together, imaging does not replace or supersede neuropsychology, but rather complements it. In fact, imaging is increasingly moving past simple lesion identification and toward

becoming a measure of cognitive functioning, and is used to identify specific treatments and monitor treatment efficacy. In this section, we will discuss some of the diagnostic issues neuropsychologists should be acquainted with, as well as future directions for neuroimaging in the context of rehabilitation.

## Neuroimaging and TBI

Moderate to severe TBI is associated with cognitive and physical deficits. Neuroimaging plays a central role in acute medical management of TBI and is often used to determine the extent and severity of TBI. As noted previously, acute CT scans are used for decision making regarding the surgical intervention of space-occupying lesions, determining the presence and nature of focal intracranial lesions and/or diffuse axonal injury, and as a surrogate marker of intracranial pressure. Although traumatic axonal injury, also referred to as DAI, can be widespread, it is typically found in areas where the white matter and gray matter interface. Specific neuroanatomical regions where DAI may have manifested include the subcortical white matter, corpus callosum, and brainstem (Povlishock & Katz, 2005). Other methods of evaluating the effects of DAI include T2\* gradient echo imaging, which is more sensitive to hemorrhage than standard T2-weighted imaging and can identify microbleeding associated with DAI. Diffuse axonal injuries do not resolve with time. Diffuse axonal injury-related lesions can be detected several years after TBI (Scheid, Preul, Gruber, Wiggins, & von Cramon, 2003). In addition to DAI, TBI can produce focal injury, including contusions and lesions throughout the brain but most commonly found in the frontal and temporal regions (Bigler, 2001; Povlishock & Katz, 2005). Disruption of axonal transport is not limited to time of injury; within hours of the injury neuronal transport is impaired resulting in swelling, disconnection, and accumulation of various molecules at the axoplasmic transport site (Stone et al., 2002). Furthermore, secondary injury occurs from other mechanisms such as metabolic (i.e., edema, excitotoxicity), vascular (i.e., hemorrhage, hypoxia), and mechanistic changes (e.g., increased intracranial pressure) (Povlishock & Katz, 2005; Smith, Meaney, & Shull, 2003). Neuroimaging can be used to detect secondary neural injuries.

Predicting TBI outcome is complicated due to the timing of primary and secondary neural injury and because the effects of the injury are often not limited to the original lesion site. Three examples follow. First, Wallerian degeneration, axonal and myelin degeneration of the neuron distal to the injury site, may not be detectable for several months following injury (Povlishock &

Katz, 2005). The loss of neurons may result in atrophic changes, which may not be present until months following brain injury. Second, in a landmark study of temporal lobe morphology in both aging and TBI, it was discovered that TBI disproportionately affected the white matter of the temporal lobes, suggesting the importance of evaluating white matter in TBI (Bigler, Anderson, & Blatter, 2002). Furthermore, the potential effects of white matter injury in the temporal lobes (i.e., decreasing neuronal function throughout the temporal stem) may result in restricted information processing in limbic pathways (e.g., hippocampus, fornix, etc.), which contributes to the memory and mood-related morbidities associated with TBI. Third, from a developmental and aging perspective, injury not only affects the current state of brain development, but can change the developmental trajectory (this is especially true in pediatric brain injury). The developmental effects of the injury can manifest as inhibited growth; for example, an injury at a young age might prevent normal age-related increases in white matter connections and in older age an injury might hasten or exacerbate normal age-related cognitive decline. Severe TBI in children not only damaged the corpus callosum, but also prevented the normal age-related increase in corpus callosum volume, which occurred in less severely injured children (Levin et al., 2000). Information is accumulating that suggests individuals with a history of TBI do not “age as well” as healthy adults without a history of TBI. A landmark study on the effects of aging following cerebral injury by Corkin, Rosen, Sullivan, and Clegg (1989) found penetrating head injury in adolescence exacerbated cognitive aging in late life (Corkin et al., 1989). This 30-year longitudinal study compared nondemented World War II veterans with remote history of penetrating head injury to those who had sustained remote history of peripheral nerve injury. Differences in the individuals’ cognitive function in 1950 compared to 1980 showed a generalized cognitive decline in the TBI group, irrespective of lesion site, although analysis by lesion site also showed focal impairments. Because the cognitive decline in the TBI group was above and beyond that expected, given stable cognitive function and maximal recovery, the authors hypothesized that an interaction between the penetrating head injury and the aging process might exist. For an excellent review of structural imaging and cognitive outcomes following TBI, the reader is referred to the chapter by Bigler (2011), which appears in the *Textbook of Traumatic Brain Injury* edited by Silver, McAllister, and Yudofsky.

Although structural imaging can identify anatomic evidence of injury, it does not detect impaired function in tissue that appears “normal.” Functional



imaging can detect abnormal neural function, assess task-specific function, and may help link the anatomic lesions to behavioral and cognitive consequences of TBI ([Jantzen et al., 2004](#)). Prospective memory, the ability to remember and carry out actions in the future, relies on a neural network that includes the frontal lobes and medial temporal lobes. A study in TBI patients using DAI found prospective memory was positively correlated with FA values in the left parahippocampal gyrus, left inferior parietal lobe, and left anterior cingulate gyrus in individuals with TBI ([Kondo et al., 2010](#)). These brain regions are part of the neural circuit that is involved in prospective memory.

### Neuroimaging and CVA

The initial diagnosis of CVA essentially starts by determining whether the lesion is hemorrhagic or ischemic, which is crucial as treatment is dramatically different depending on the type of CVA (e.g., anticoagulant therapy, surgical repair of blood vessels, or evacuation of blood). A detailed review of the pathophysiology of ischemic stroke (>80% of all stroke cases) is provided by [Doyle, Simon, and Stenzel-Poore \(2008\)](#). [Doyle and colleagues \(2008\)](#) describe the pathophysiology of ischemic stroke as a “complex interplay of excitotoxicity, acidosis, inflammation, oxidative stress, peri-infarct depolarization and apoptosis” (p. 9). It is important to note that these multiple pathophysiological processes, which also relate to structural changes such as Wallerian degeneration, each has its own unique time course. During the initial diagnostic phase, in addition to the underlying mechanisms (e.g., ischemic vs. hemorrhagic), the size of the lesion, specific lesion location, lesion characteristics (e.g., small vessel, large vessel), and their associations with the clinical deficit(s), it is important to determine how much “savable” tissue may be present in the area surrounding the lesion, which is known as the ischemic penumbra. It is in the ischemic penumbra that pharmacologic interventions are most likely to be successful. Neuroimaging plays an important role not only in measuring the original infarct zone (or, in the case of TBI, original contusion or hemorrhage), but also in assessing how potentially treatable areas of tissue respond to treatment ([Doyle et al., 2008](#); [Fabricius et al., 2006](#); [Lauritzen et al., 2011](#)). In reviewing neuroimaging studies of CVA, the informed neuropsychologist should consider the potential effect of minimal or maximal dysfunction in these perilesional zones in determining which neuropsychological tests may best assess the patient’s cognitive function. Considerations of tests should include how the initial area of infarct and the surrounding perilesional

areas might affect cognitive function, and ultimately cognitive and functional outcome. Of course, test selection will also depend on the condition of the brain prior to the most recent stroke.

A brain with severe underlying microvascular ischemic disease, prior infarct, or hemorrhage will have a different clinical course and outcome than a more “pristine” brain (for example, see [Bejot et al., 2011](#), regarding risk factors associated with early dementia following stroke). Furthermore, other variables that influence outcomes include age, a progressive disease course, and unique clinical manifestations or syndromes that may be observed depending on the underlying neuropathology and size and location of the lesion. There are entire studies devoted to reviewing clinical outcomes in specific stroke syndromes such as thalamic hemorrhage. A retrospective analysis of 175 consecutive patients with thalamic hemorrhage classified these patients into four anatomical subtypes: anterior, posteromedial, posterolateral, and global (entire thalamus involved), and found subtype differences in symptoms, morbidity, and clinical outcomes ([Chung et al., 1996](#)). Similarly, [Carrera and Bogousslavsky \(2006\)](#) described distinct behavioral syndromes associated with thalamic infarction depending on the four main arterial thalamic territories. In past decades, neuroimaging studies were not refined enough in terms of spatial and temporal information to specifically map out characteristic lesion locations and surrounding hypometabolic tissue of common pathways among patients who were associated with these neurobehavioral symptoms. As the technology has improved, this possibility is starting to be realized. A recent study of six cases with left anterior thalamic infarction found that the patients had associated neurobehavioral and neuropsychological sequelae, including impairments in verbal memory, language function (e.g., anomia), and apathy ([Nishio, Hashimoto, Ishii, & Mori, 2011](#)). [Nishio et al. \(2011\)](#) carried out stereotactic lesion location using MRI through a linked anatomic atlas, then measured cerebral blood flow via PET imaging, and carried out neuropsychological assessment. The mammillothalamic tract, part of the Papez circuit, was compromised in all subjects, which likely contributed to the memory deficits. Whereas, the internal medullary lamina was affected in half of the subjects, causing the thalamus to disconnect from the anterior temporal lobe and likely accounts for the presence of language deficits. Finally, apathy was thought to be due to hypometabolism in the left anterior cingulate gyrus ([Nishio et al., 2011](#)). Along these same lines, using DTI, decreased FA values in the fornix of patients with thalamic hemorrhage suggested possible neuronal loss in the fornix, which

supports of the role of Papez circuit disruption in memory loss (Yeo & Jang, 2011). Future coupling of multiple neuroimaging techniques might help explain cognitive and behavioral outcomes common to many patients with the same injury or neurological disorder, as well as help explain individual clinical differences.

For a review of rehabilitation, neuroimaging, and outcome in stroke, the reader is referred to Carey and Seitz (2007b). Additionally, the reader is referred to an excellent book by Festa and Lazar (2009), *Neurovascular Neuropsychology*.

### Neuroimaging Following Anoxia

Anoxic brain injury (ABI) can result in both focal and diffuse lesions and atrophy in the cortical and subcortical structures. A review article found that of the 90 patients with ABI across the studies analyzed, 44% of patients had cortical edema or atrophy, 33% had cerebellar lesions, 22% had basal ganglia lesions, 21% had hippocampal atrophy, and 3% had thalamic lesions (Caine & Watson, 2000). Both necrotic processes and demyelination following ABI were observed in the cerebellum (O'Donnell, Buxton, Pitkin, & Jarvis, 2000). Further, the medial temporal lobes, including the hippocampus, are selectively vulnerable to ABI. Lesions following ABI are common in neural areas at the end of the vascular supply, including the hippocampal (Manns, Hopkins, Reed, Kitchener, & Squire, 2003), basal ganglia, and cerebellum (Armengol, 2000). Hippocampal atrophy is also common and is associated with impaired declarative memory (Goodrich-Hunsaker & Hopkins, 2010). The temporal horns of the lateral ventricles are sensitive markers of temporal lobe damage and indicate temporal lobe and/or hippocampal atrophy (Bigler, 2001). The cerebellum is also vulnerable to ABI, especially Purkinje cell death due to excitotoxicity (Welsh et al., 2002). Cerebellar damage results in ataxia or poor motor coordination, problems initiating or ending purposeful movements, impaired balance, difficulty maintaining posture, and problems with gait (Heimer, 1995). Cognitive deficits secondary to cerebellar pathology include impaired executive function, visuospatial abilities, language, memory, and emotional changes (Schmahmann & Pandya, 1997).

In addition to gray matter damage, ABI also effects white matter damage. White matter lesions following ABI occur in the cerebellum (Mascalchi, Petrucci, & Zampa, 1996) and subcortical and periventricular white matter pathways (Parkinson et al., 2002). Similar to basal ganglia lesions discussed

above, white matter hyperintensities onset and duration are heterogeneous. For example, white matter hyperintensities were present on Day 1 (Jackson et al., 2003) and 7 days following ABI on DWI (Chalela, Wolf, Maldjian, & Kasner, 2001). Further, the fornix, which is the major output pathway from the hippocampus to the mammillary bodies, is a common site of atrophy and is associated with verbal memory impairments following ABI (Kesler, Hopkins, Blatter, Edge-Booth, & Bigler, 2001). Thus, the memory impairments in ABI are likely due to hippocampal cell loss or due to anterograde degeneration of the hippocampal axons in the fornix following hippocampal neuronal loss. Atrophy of the corpus callosum also occurs following ABI (Porter, Hopkins, Weaver, Bigler, & Blatter, 2002). The above data indicate that ABI results in diffuse and focal injuries and atrophy in a wide range of cortical and subcortical structures.

### Neuroimaging Following Critical Illness

Unlike TBI, CVA, and anoxia, the role of neuroimaging in critically ill populations is understudied. Recently studies have begun to assess the effects of critical illness on neurological structure and function. Mortality following critical illness has substantially declined over the last 20 years (Williams et al., 2008) resulting in increased number of survivors, many of whom have significant neurologic morbidities. Although we have been long aware that individuals with TBI and CVA have cognitive impairments, recent studies report that a high percentage of survivors of critical illness develop cognitive impairments (Hopkins, Weaver, et al., 2005), which appear similar to cognitive impairments observed following TBI and anoxic brain injury. Medical technological advances have improved central nervous system (CNS) monitoring in critically ill patients, allowing physicians to diagnose delirium, seizures, and encephalopathy quickly and reliably. Brain imaging techniques such as CT, MRI, and fMRI techniques are increasingly used as part of a group of diagnostic tools that are used to evaluate neurologic function during and following critical illness.

Early neuroimaging investigations failed to find significant abnormalities on brain imaging in medical intensive care unit (ICU) (critically ill) populations (Young, 1995). For example, an early neuroimaging study found no abnormalities on brain imaging in critically ill patients with encephalopathy due to sepsis (Jackson, Gilbert, Young, & Bolton, 1985). More recently, neuroimaging has been used to guide short-term medical decision making (e.g., bleeding, infection, etc.), guide therapy, and assess long-term prognosis in

critically ill patients, similar to more well-studied neurologic disorders and brain injuries. A randomized trial in mechanically ventilated patients found changes in mental status explained by neuroimaging about 25% of the time (Kress, Pohlman, O'Connor, & Hall, 2000). A recent study that assessed the relationship between brain MRI and clinical treatment in eight delirious critically ill patients found six (75%) had white matter hyperintensities and one (12%) patient had mild atrophy (Morandi et al., 2010). Although brain imaging (MRI and CT) studies did not result in a change in diagnoses or change in medical or surgical treatment for any patient in this study, lesions on neuroimaging were associated with severe impairments in memory, executive function, and attention (Morandi et al., 2010).

A study of neuroimaging using CT and MRI in 64 critically ill patients who were imaged for diminished level of consciousness, confusion, altered mental status, coma, or prolonged delirium found 17 (39%) patients had normal scans and 28 (41%) had abnormalities on brain imaging (Suchyta, Jephson, & Hopkins, 2010). These findings suggest that neuropathological findings not only occur in critically ill patients with changes in mental status, but it is notable that patients with focal CNS insults and preexisting neurologic or neurodegenerative disease were excluded from the study (Suchyta et al., 2010). Imaging and pathological studies find white matter lesions are common in critically ill patients with sepsis (Sharshar et al., 2007). A small quantitative brain imaging study in critically ill patients found substantial generalized brain atrophy and structural lesions in cortical and subcortical structures (e.g., hippocampus) that persisted months post ICU discharge (Hopkins, Gale, & Weaver, 2006). Thus, critical illness is associated with significant brain atrophy and cortical and subcortical lesions. The abnormalities on brain imaging are correlated with cognitive impairments, similar to that observed following TBI or anoxic brain injury.

Recent studies find delirium is associated with cognitive impairments in critically ill patients. An investigation that assessed the relationship of delirium and cognitive outcomes in critically ill patients found the duration of delirium independently predicted cognitive impairment 12 months post ICU discharge (Girard et al., 2010). Increased duration of delirium is also associated with smaller brain volumes at hospital discharge and 3-month follow-up in ICU survivors (Gunther et al., 2010). Several studies have found that noncritically ill patients with delirium have reduced regional cerebral blood flow in the frontal and temporal lobes (Fong et al., 2006; Yokota, Ogawa, Kurokawa, &

[Yamamoto, 2003](#)). White matter abnormalities are risk factors for the development of delirium in older patients who underwent cardiac surgery ([Shioiri et al., 2010](#)). Thus, delirium is associated with neurological abnormalities and cognitive impairments. The above findings indicate that critical illness and/or its treatment contributes to brain injury. Neuroimaging is beginning to play an important role not only in diagnosis of critically ill patients, but also in assessing outcomes and in the future may play a role in guiding treatments and rehabilitation.

## **NEUROIMAGING AS A BIOMARKER OF REHABILITATION EFFECTIVENESS**

Although there is a long history of studying the effects of brain injury with neuroimaging techniques, this was typically done to learn more about the underlying neuropathology of these injuries and how these might correlate with outcome (e.g., in TBI correlating initial injury severity with lesion volume, atrophic changes, or likelihood of return to work), rather than using the neuroimaging to measure the effects of rehabilitation. The potential roles that neuroimaging, particularly functional neuroimaging, can play in recovery and rehabilitation are evolving rapidly. [Seitz \(2010\)](#), in regard to stroke, describes it this way: “Functional imaging is a physiological tool which allows the study of brain activity related to specific activation states as well as its changes related to adaptation to the lesion, to deficit compensation, and to relearning” ([Seitz, 2010](#), p. 79). Research in animals and humans has had significant impact on recovery, neuroplasticity, and rehabilitation ([Turkstra, Holland, & Bays, 2003](#)). Recent developments in neuroimaging have made it possible to study plasticity, recovery, and rehabilitation in vivo. For example, fMRI studies detailing cortical finger representations in primary motor cortex of blind individuals who read Braille since they were young were significantly different from individuals who became blind and learned to read Braille as adults ([Pascual-Leone et al., 1993](#)). In this section, we will discuss studies, some in rehabilitation and other fields, which describe the use of quantitative and functional imaging and how they relate to the fields of rehabilitation and neuropsychology.

Quantitative neuroimaging techniques have not only been used to identify neuropathological changes in neurologic disorders, but have also been utilized to demonstrate differences between healthy individuals due to both uncontrollable (e.g., sex, age) and controllable (e.g., task mastery) variables. A landmark study demonstrated that healthy individuals with “extensive navigation experience”

due to driving a taxi in a very challenging environment (i.e., London) had larger posterior hippocampal volumes compared to healthy controls without this experience (Maguire et al., 2000). Furthermore, years of experience was positively correlated with larger hippocampal volumes. These findings were thought to reflect the potential for “local plastic change” in the human brain. Focal increases in gray matter occurred in humans in response to acquiring a new motor skill such as juggling (Draganski & May, 2008). Similarly, training-induced changes were found in the white matter of healthy adult humans (increased FA as measured by DTI) in response to learning how to juggle (Scholz, Klein, Behrens, & Johansen-Berg, 2009). Changes are observed in both gray and white matter in individual monkeys trained to use tools for the first time (Quallo et al., 2009). For a review of training-induced changes in the brain see Draganski and May (2008). Thus, experience changes the structural makeup of the brain and neuroimaging techniques can detect structural changes. These studies use an imaging technique known as voxel-based morphometry (VBM). Conventional structural high-resolution MRI scans are amenable to 3D reconstruction (e.g., high number of very thin slices throughout the entire brain), and the VBM method is a postprocessing method. Each voxel, or 3D pixel, in the images is subject to a statistical procedure mapping the probability that it belongs to white matter, gray matter, or cerebrospinal fluid creating a tissue-specific segmented image. Gray matter, for example, can be segmented out and the resultant gray matter map of the brain prior to the new learning can be statistically compared voxel by voxel to the postlearning gray matter map and the differences and volumetric changes can be calculated. This technique has been successfully applied to various neurologic disorders, including TBI (Gale, Baxter, Roundy, & Johnson, 2005; Gale & Prigatano, 2010). The advantage of VBM over tracing is that it is fully automated and is not restricted to anatomical boundaries; each voxel is considered and the threshold can be set for change based on statistical and/or minimum cluster size (number of connected voxels).

Other than the potential challenges of mapping an injured brain which changes over time due to the effects of injury, these techniques can be used in rehabilitation in the same way they have been utilized in healthy controls showing learning-associated or rehabilitation-associated changes. For example, DTI and fMRI findings are better predictors of improved reading in subjects with dyslexia than were standard behavioral measures (Hoeft et al., 2011). A recent study demonstrated the usefulness of DTI compared to conventional MRI techniques in determining motor outcome after ischemic middle cerebral artery

(MCA) stroke at 30 days (Puig et al., 2010). DTI-FA values were more sensitive than conventional MRI in detecting Wallerian degeneration in the cortical spinal tract and correlated with motor outcome (Puig et al., 2010). As can be seen by these examples, there are a variety of techniques that can be utilized and the most useful may depend on the clinical question, neural system, or specific outcome measured. The overarching theme here is that the future role of neuroimaging in rehabilitation might include individual markers of not only severity of injury, but also neural changes (i.e., physiological and structural) associated with treatment in order to understand the underlying mechanisms of change (or absence of change) with any given rehabilitative treatment and serve as a marker of the treatment's effectiveness.

Neural markers of rehabilitation effectiveness may be very important for many reasons, some of which may be counterintuitive. For example, it may be that interventions at certain (critical) periods may be less effective or even harmful (Boake et al., 2007; Humm, Kozlowski, James, Gotts, & Schallert, 1998). In the field of multiple sclerosis (MS), the standard is that imaging has not only been used for diagnosis and understanding the underlying neuropathology of the disease, but also used to monitor the development and even of the response to treatment using serial scans (Bermel & Fox, 2010). Though MS is a special case, the idea of neuroimaging biomarkers of treatment response might be very helpful in the field of rehabilitation, particularly in the context of functional imaging that assesses brain changes associated with recovery to specific treatments. Additionally, structural neuroimaging can be used to ascertain the overall effect on the brain of an acute treatment or surgical procedure in TBI, CVA, or anoxia. A recent example demonstrated increased cortical thickness in patients with severe cerebrovascular steno-occlusive disease that underwent surgical revascularization that was assessed using automated quantitative MRI measurements using Freesurfer software (<http://surfer.nmr.mgh.harvard.edu>; Fierstra et al., 2011). Approximately 11 months following surgery, cortical thickness increased by an average of 5.1% in the revascularized hemisphere. It should be noted that this study employed an fMRI BOLD technique allowing identification of regional deficiencies in cerebral blood flow, the likely underlying physiologic reason for cortical thinning in these same regions. Thus, this study utilized conventional MRI that was subsequently processed via automated software to measure regional cortical thickness and fMRI technique was used to measure regional cerebral blood flow. The combination of these techniques helped identify target areas for intervention



and then allowed for measurement of the effect of the surgical intervention on brain structure over time.

### **Neuroimaging and Rehabilitation Outcome in TBI**

Acute CT scans are vital to emergency management of TBI; in addition, these scans are also important to rehabilitation clinicians as they are frequently the only available neuroimaging data from which clinicians can make inferences, conclusions, and prognostic predictions (Gale & Hopkins, 2004). Acute abnormalities on CT scans predict functional outcomes following TBI (Cowen et al., 1995). For example, acute CT abnormalities are associated with increased need for assistance with ambulation and activities of daily living, increased need for global supervision, and decreased ability to return to work following TBI (Reider et al., 2002). Few studies have examined the predictive utility of initial CT abnormalities in predicting rehabilitation outcomes. Several studies show that the day-of-injury abnormalities on CT predict cerebral atrophy, but inconsistently predict rehabilitation outcomes (Bigler, Ryser, Gandhi, Kimball, & Wilde, 2006; Brown et al., 2005). Although MRI clearly has better resolution than CT, the ability of acute MRI to predict functional outcome following TBI is also quite limited (Bigler, 2011). This is likely due to a variety of factors, including the fact that both the primary and secondary effects of TBI may or may not be evident on the initial scan. Even less obvious on initial scanning are the ultimate effects that lesions have on underlying brain structure; as atrophic changes take time to be manifested on CT and MRI. Still, many studies have demonstrated the usefulness of neuroimaging findings, typically obtained in the postacute or chronic stages of TBI, and their association to a variety of clinical outcomes. For example, structural neuroimaging findings in TBI are associated with cognitive performance (Bigler et al., 1996; Blatter et al., 1997; Gale et al., 2005; Gale, Burr, Bigler, & Blatter, 1993; Gale, Johnson, Bigler, & Blatter, 1994, 1995; Hopkins, McCourt, & Cleavinger, 2003; Johnson, Bigler, Burr, & Blatter, 1994; Johnson, Pinkston, Bigler, & Blatter, 1996; Kraus et al., 2007), neurobehavioral outcome (Barker et al., 1999; Bigler, 1999; Vasa et al., 2004), ultimate level of global functioning or recovery (Levin et al., 1997, 2008; Pierallini et al., 2000; Wilde et al., 2005), and even parental ratings of social reintegration in children with TBI (Gale & Prigatano, 2010). Reviewing the above-mentioned studies, one will notice a distinct trend from earlier studies measuring global markers of neurotrauma (e.g., total ventricular volume as it relates to total brain volume) to more local markers investigating specific

networks and anatomical pathways, which is partly due to both increased resolution as well as increasingly subtle techniques. Similarly, prior studies necessarily highlighted gross or macrostructural anatomical changes, whereas more recent studies assess microstructural changes not previously appreciated. Decreased thalamic volumes following TBI might represent transneuronal degeneration as a consequence of TBI, because even patients without visible thalamic lesions had decreased volumes (Anderson, Wood, Bigler, & Blatter, 1996). More recently, DTI was used to measure the microstructural integrity of the thalamus following TBI, including measuring seven different thalamic regions (Little, Kraus, Joseph, et al., 2010). This type of analysis simply could not have been carried out with prior technology. Little, Kraus, Joseph, et al. (2010) found, among other things, that although DTI measures of the ventral posterior lateral nucleus were associated with performance on memory measures, the anterior thalamic nucleus and ventral anterior thalamic nucleus were associated with attention (Little, Kraus, Joseph, et al., 2010). Further, impairment on executive measures correlated with cortical-subcortical damage rather than being limited to frontal cortical injury, which may explain why executive dysfunction does not necessarily require gross lesions in the frontal cortex (Little, Kraus, Joseph, et al., 2010).

MRS in pediatric TBI patients predicted adverse neurological outcomes, specifically lower NAA/Cre ratio or NAA/Cho ratio or high Cho/Cre ratio were associated with poor neurologic outcomes (Ashwal et al., 2000). Similarly, single-voxel MRS predicted poor neuropsychological outcome in individuals with pediatric TBI (Brenner, Freier, Holshouser, Burley, & Ashwal, 2003). A study in children and adolescents with TBI found NAA values correlated with cognitive scores and the mean NAA/Cre ratio explained over 40% of the variance in cognitive scores, beyond demographic or clinical variables (Babikian et al., 2006). Thus, MRS appears to improve prediction of cognitive recovery and may be a valuable tool in evaluating the effect of cognitive rehabilitation on neuropsychological outcomes following TBI.

### **Neuroimaging and Rehabilitation Outcome in CVA**

Stroke is among the most common causes of death and adult disability, including cognitive and motor impairments (Stinear, 2010). Recent studies have demonstrated subtle acute changes that were correlated with motor outcome at 3 months. In one stroke study utilizing DWI, reduced ADC coefficients in the ipsilesional cerebral peduncle predicted worse motor outcome (poor recovery) at

3 months (DeVetten et al., 2010). A study that used fMRI poststroke to assess motor activity found positive correlations between the amount of task-related fMRI activity in the first 48 hours after stroke and the amount of motor recovery at 3-month follow-up (Marshall et al., 2009). A review of motor function poststroke notes that functional imaging predicts motor impairment months later, and motor impairment and motor recovery are important predictors of functional outcomes and influence the ability to live independently (Stinear, 2010). Given that DTI and fMRI can evaluate and predict recovery of function poststroke, it follows that these techniques could be used to assess the effectiveness and gains induced by rehabilitation, including rehabilitation of motor and cognitive function. There are several recent review papers that address the current and potential uses of structural and functional neuroimaging in recovery and neurorehabilitation post-CVA (Carey & Seitz, 2007a; Eliassen et al., 2008; Seitz & Donnan, 2010).

Regarding rehabilitation, transcranial magnetic stimulation has been used to stimulate corticomotor excitability in the motor system. Increased corticomotor excitability was associated with recovery of independence in activities of daily living (feeding, bathing, dressing, toileting, continence, and transfer) at 6 months (Di Lazzaro et al., 2010). Similarly, arm-focused therapy for 2 weeks resulted in increased brain activation on fMRI, which was associated with functional improvement in arm and hand motion (Dong, Dobkin, Cen, Wu, & Winstein, 2006). Recovery of language following CVA using daily melodic intonation speech therapy was assessed pretreatment and posttreatment using DTI; and language therapy resulted in increased fibers and volumes of the right arcuate fasciculus and significant improvement in speech (Schlaug, Marchina, & Norton, 2009). Similar changes are reported in healthy individuals. Poor readers who underwent 100 hours of intensive reading instruction had significantly increased FA values on DTI in the left anterior centrum semiovale posttreatment compared to pretreatment values that were associated with improvement in reading (Keller & Just, 2009).

### **Neuroimaging and Rehabilitation Outcome in Anoxia**

Rehabilitation outcomes following anoxia are less well studied than those following TBI or CVA, and current data suggest that outcomes postanoxia are variable. Although studies of the effectiveness of rehabilitation are limited, research indicates that the majority of anoxic patients have poor outcomes (Bachman & Katz, 1997). For patients with severe anoxia coma (greater than 24

hours), both survival and recovery of functional abilities is poor (Groswasser, Cohen, & Costeff, 1989). Survival rates following postanoxic coma range from 9% to 40% (Bedell, Delbanco, Cook, & Epstein, 1983). Cognitive and functional recovery was assessed in comatose patients following severe anoxia ( $N = 31$ ) and only 13 patients were independent in activities of daily living, 2 patients returned to their preinjury level of cognitive function, and 1 patient returned to previous employment. The best outcomes occurred in younger patients who were less than 25 years old (Groswasser et al., 1989), which is a concern as patients with ABI tend to be older than patients with TBI. A study of eight individuals postanoxia, who underwent intensive rehabilitation, found 100% of patients had moderate-to-severe cognitive impairments (Armengol, 2000), suggesting that the rehabilitation may be less effective in this population. Similarly, a case study of severe anoxia found significant cognitive impairments and emotional dysregulation that negatively impacted the ability of the patient to participate in rehabilitation (Parkin, Miller, & Vincent, 1987). Another study in 12 patients with ABI found all patients had cognitive impairments, including impaired memory and executive dysfunction, and no relationship between the cognitive impairments and medical or injury data was found (Pesquine, Picq, & Pradat-Diehl, 2004). Individuals with higher preinjury functional status who underwent inpatient rehabilitation had the best outcomes; however, few patients returned to work or resumed their previous level of function (Schmidt, Drew-Cates, & Dombovy, 1997). A 49-year-old man with high premorbid function underwent rehabilitation following severe ABI. His cognitive function was reportedly within normal limits at 8 weeks, although the author indicated that his cognitive function was likely worse than his premorbid cognitive abilities (Kaplan, 1999). The author suggested that “Relatively good cognitive function within the first month postanoxia likely indicates improved recovery and benefit from continued rehabilitation” (Kaplan, 1999, p. 305). The finding of recovery of cognitive function appears to be uncommon, especially for severe hypoxia or anoxia.

Good cognitive recovery following ABI appears to be the exception rather than the rule, as most of the available studies show significant cognitive impairments that persist years following the injury. Survivors of ABI ( $N = 12$ ) were followed 2 years after intensive rehabilitation and all patients had permanent cognitive deficits, which were classified as amnesic and moderate to severe dementia syndromes (Pusswald, Fertl, Faltl, & Auff, 2000). A recent study that followed 93 patients with ABI found individuals with ABI were older

(males mean age 60 years), progressed more slowly in rehabilitation compared to other patients receiving rehabilitation, had worse functional outcomes, a higher percentage of patients had cognitive impairments and the impairments tended to be more severe, and the patients were more likely to be discharged from rehabilitation to a residential care facility (Fitzgerald, Aditya, Prior, McNeill, & Pentland, 2010). Similarly, a study that randomized patients with hypoxic, chronic obstructive pulmonary disease to cognitive training for attention, learning, and executive function compared to no cognitive training found that the training had no effect on cognitive function 1 to 6 months posttraining (Incalzi et al., 2008).

Several studies suggest that recovery following ABI is worse than recovery following TBI. A study that compared 15 patients with anoxic brain injury to 15 patients with TBI found that the anoxic patients' motor, cognition, and total scores on the Functional Independence Measure were lower than patients with TBI, and their rate of recovery during rehabilitation was slower than the TBI patients during inpatient rehabilitation (Cullen, Crescini, & Bayley, 2009). A second study that compared postrehabilitation outcomes of 10 patients with anoxic brain injury with matched TBI patients found that the anoxic patients had worse cognitive impairments and functional outcomes than did patients with TBI (Cullen & Weisz, 2011). These studies highlight the significant, permanent cognitive impairments following anoxic brain injury, despite acute and postacute intensive rehabilitation therapy and suggest that outcome tends to be worse in this group compared to TBI. As with TBI, the cognitive deficits following ABI adversely affect the patients' behavioral functioning, limiting their ability to return to work and lead normal social lives.

The role of imaging in assessing the effectiveness of rehabilitation in the anoxic brain injury population is not well understood. One study compared cognitive outcomes in patients with ABI to demographically matched patients with TBI (Hopkins, Tate, & Bigler, 2005). Although neither group had focal lesions, both groups had significant generalized and specific hippocampal atrophy and the cognitive impairments were similar between these two etiologically distinct groups. Hippocampal atrophy was not associated with memory impairments for either the TBI or ABI group but was associated with intellectual function. Interestingly, the amount of brain atrophy, not injury etiology, predicted cognitive outcomes (Hopkins, Tate, et al., 2005). Unfortunately, we were unable to identify any studies that used neuroimaging to assess the effectiveness of rehabilitation following anoxic brain injury. Studies

that incorporate neuroimaging with rehabilitation following anoxic brain injuries should be the focus of future research.

### **Neuroimaging and Rehabilitation Outcome in Critical Illness**

We are just beginning to understand the extent and severity of cognitive impairments as well as the duration of the impairments, and their effect on functional abilities following critical illness. A 2-year outcome study in ICU survivors found that nearly a third (32%) of the patients were unemployed, retired, or were receiving new disability (Hopkins, Weaver, et al., 2005). Further, 31 patients (42%) were referred for inpatient rehabilitation of which 23 referrals were for physical weakness or deconditioning and 8 referrals were for limb fractures or amputations. Only nine patients (12%) were subsequently identified as having cognitive impairments and received cognitive rehabilitation on the rehabilitation unit (Hopkins, Weaver, et al., 2005). Studies are just beginning to systematically investigate potential interventions in critically ill populations. Studies suggest that medications such as sedatives given during hospitalization adversely impact on cognitive function even after hospital discharge. A recent study compared long-term neurocognitive outcomes in critically ill, mechanically ventilated patients (Girard et al., 2010). A longer duration of delirium predicted worse cognitive impairment at 3 and 12 months (Girard et al., 2010). A second study assessed cognitive outcomes in patients treated with a protocol that interrupts and reduces sedative exposure with breathing trials as compared to spontaneous breathing trials alone, and found cognitive impairments were less common in the group who received the wake-up and breathe intervention (Jackson, Girard, et al., 2010).

Both the implementation and study of cognitive rehabilitation in survivors of critical illness is in its infancy. The only cognitive rehabilitation study conducted to date is a small Phase II clinical trial. Survivors of critical illness were randomized to receive either usual care (sporadic rehabilitation) compared to a 3-month program of physical and cognitive rehabilitation using Goal Management Training (Jackson, Clune, et al., 2010). All patients enrolled in the study had cognitive impairments at hospital discharge. The group that received cognitive rehabilitation had better cognitive function, including improved performance on measures of executive function at 3-month follow-up (Jackson, Clune, et al., 2010). The study by Jackson *et al.* suggests that survivors of critical illness may benefit from cognitive rehabilitation and therefore efforts should be made to facilitate rehabilitation in this population and increase

awareness of the need for rehabilitation in critical care and rehabilitation providers. Cognitive rehabilitation following critical illness is limited and studies are needed to determine the effectiveness in this population.

## SUMMARY

Neuroimaging is used routinely in the diagnosis of neurological injury and disease. Conventional methods of both structural and functional measures of cortical integrity are readily available to treating clinicians and can inform rehabilitation efforts. Over time, a plethora of studies have attempted to utilize standard clinical imaging data to investigate factors associated with initial injury severity, differential diagnosis, and biomarkers of functional outcome. More recently, the development of increasingly sophisticated imaging techniques has allowed the analysis of subtle changes (e.g., regional cortical thickness, spectroscopy of ipsilesional and contralesional cortex) providing previously unavailable information. The successful coupling of structural and functional data will further enhance our understanding of not only the underlying pathophysiology and spontaneous changes over time following injury, but also will increasingly be used as a measure of rehabilitation effectiveness and individual response to treatment.

## REFERENCES

- Anderson, C. V., Wood, D. M., Bigler, E. D., & Blatter, D. D. (1996). Lesion volume, injury severity, and thalamic integrity following head injury. *Journal of Neurotrauma*, 13(2), 59–65.
- Anderson, V., & Yeates, K. O. (2010). *Pediatric traumatic brain injury: New frontiers in clinical and translational research*. New York, NY: Cambridge University Press.
- Armengol, C. G. (2000). Acute oxygen deprivation: neuropsychological profiles and implications for rehabilitation. *Brain Injury*, 14(3), 237–250.
- Ashwal, S., Holshouser, B. A., Shu, S. K., Simmons, P. L., Perkin, R. M., Tomasi, L. G., . . . Hinshaw, D. B. (2000). Predictive value of proton magnetic resonance spectroscopy in pediatric closed head injury. *Pediatric Neurology*, 23(2), 114–125.
- Babikian, T., Freier, M. C., Ashwal, S., Riggs, M. L., Burley, T., & Holshouser, B. A. (2006). MR spectroscopy: Predicting long-term neuropsychological outcome following pediatric TBI. *Journal of Magnetic Resonance Imaging*, 24(4), 801–811.
- Bachman, D., & Katz, D. I. (1997). Anoxic-hypotensive brain injury and encephalitis. In V. M. Mills & D. I. E. Katz (Eds.), *Neurologic rehabilitation: A guide to diagnosis, prognosis, and treatment planning* (pp. 145–176). Malden, MA: Blackwell Science.
- Barker, L. H., Bigler, E. D., Johnson, S. C., Anderson, C. V., Russo, A. A., Boineau, B., and Blatter, D. D. (1999). Polysubstance abuse and traumatic brain injury: Quantitative magnetic resonance imaging and neuropsychological outcome in older adolescents and young adults. *Journal of the International Neuropsychological Society*, 5(7), 593–608.
- Basser, P. J., Mattiello, J., & LeBihan, D. (1994). MR diffusion tensor spectroscopy and imaging. *Biophysical Journal*, 66(1), 259–267.

- Bedell, S. E., Delbanco, T. L., Cook, E. F., & Epstein, F. H. (1983). Survival after cardiopulmonary resuscitation in the hospital. *New England Journal of Medicine*, *309*(10), 569–576.
- Bejot, Y., Aboa-Eboule, C., Durier, J., Rouaud, O., Jacquin, A., Ponavoy, E., . . . Giroud, M. (2011). Prevalence of early dementia after first-ever stroke: A 24-year population-based study. *Stroke*, *42*(3), 607–612.
- Bermel, R. A., & Fox, R. J. (2010). *MRI in multiple sclerosis*. Philadelphia, PA: Lippincott Williams & Wilkins.
- Bigler, E. D. (1999). Neuroimaging in pediatric traumatic head injury: Diagnostic considerations and relationships to neurobehavioral outcome. *Journal of Head Trauma Rehabilitation*, *14*(4), 406–423.
- Bigler, E. D. (2001). Quantitative magnetic resonance imaging in traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *16*(2), 117–134.
- Bigler, E. D. (2011). Structural neuroimaging. In T. W. McAllister, S. C. Yudofsky, & J. M. Silver (Eds.), *Textbook of traumatic brain injury* (2nd ed., pp. 73–91). Arlington, TX: American Psychiatric Publishing.
- Bigler, E. D., Anderson, C. V., & Blatter, D. D. (2002). Temporal lobe morphology in normal aging and traumatic brain injury. *American Journal of Neuroradiology*, *23*(2), 255–266.
- Bigler, E. D., Johnson, S. C., Anderson, C. V., Blatter, D. D., Gale, S. D., Russo, A. A., . . . Abildskov, T. J. (1996). Traumatic brain injury and memory: The role of hippocampal atrophy. *Neuropsychology*, *10*(3), 333–342.
- Bigler, E. D., Ryser, D. K., Gandhi, P., Kimball, J., & Wilde, E. A. (2006). Day-of-injury computerized tomography, rehabilitation status, and development of cerebral atrophy in persons with traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation*, *85*(10), 793–806.
- Bigler, E. D., & Wilde, E. A. (2010). Quantitative neuroimaging and the prediction of rehabilitation outcome following traumatic brain injury. *Frontiers in Human Neuroscience*, *4*, 228.
- Blatter, D. D., Bigler, E. D., Gale, S. D., Johnson, S. C., Anderson, C. V., Burnett, B. M., . . . Bailey, B. J. (1997). MR-based brain and cerebrospinal fluid measurement after traumatic brain injury: Correlation with neuropsychological outcome. *American Journal of Neuroradiology*, *18*, 1–10.
- Boake, C., Noser, E. A., Ro, T., Baraniuk, S., Gaber, M., Johnson, R., . . . Levin, H. S. (2007). Constraint-induced movement therapy during early stroke rehabilitation. *Neurorehabilitation and Neural Repair*, *21*(1), 14–24.
- Bradley, W. G., Jr. (1993). MR appearance of hemorrhage in the brain. *Radiology*, *189*(1), 15–26.
- Brenner, T., Freier, M. C., Holshouser, B. A., Burley, T., & Ashwal, S. (2003). Predicting neuropsychologic outcome after traumatic brain injury in children. *Pediatric Neurology*, *28*(2), 104–114.
- Brown, A. W., Malec, J. F., McClelland, R. L., Diehl, N. N., Englander, J., & Cifu, D. X. (2005). Clinical elements that predict outcome after traumatic brain injury: A prospective multicenter recursive partitioning (decision-tree) analysis. *Journal of Neurotrauma*, *22*(10), 1040–1051.
- Caine, D., & Watson, J. D. (2000). Neuropsychological and neuropathological sequelae of cerebral anoxia: A critical review. *Journal of the International Neuropsychological Society*, *6*(1), 86–99.
- Carey, L. M., & Seitz, R. J. (2007a). Functional neuroimaging in stroke recovery and neurorehabilitation: Conceptual issues and perspectives. *International Journal of Stroke*, *2*, 245–264.
- Carey, L. M., & Seitz, R. J. (2007b). Functional neuroimaging in stroke recovery and neurorehabilitation: Conceptual issues and perspectives. *International Journal of Stroke*, *2*(4), 245–264.
- Carrera, E., & Bogousslavsky, J. (2006). The thalamus and behavior: Effects of anatomically distinct strokes. *Neurology*, *66*(12), 1817–1823.
- Chalela, J. A., Wolf, R. L., Maldjian, J. A., & Kasner, S. E. (2001). MRI identification of early white matter injury in anoxic-ischemic encephalopathy. *Neurology*, *56*(4), 481–485.
- Chung, C. S., Caplan, L. R., Han, W., Pessin, M. S., Lee, K. H., & Kim, J. M. (1996). Thalamic haemorrhage. *Brain*, *119*(Pt 6), 1873–1886.
- Conturo, T. E., Lori, N. F., Cull, T. S., Akbudak, E., Snyder, A. Z., Shimony, J. S., . . . Raichle, M. E. (1999). Tracking neuronal fiber pathways in the living human brain. *Proceedings of the National*



- Academy of Sciences USA*, 96(18), 10422–10427.
- Corkin, S., Rosen, T. J., Sullivan, E. V., & Clegg, R. A. (1989). Penetrating head injury in young adulthood exacerbates cognitive decline in later years. *Journal of Neuroscience*, 9(11), 3876–3883.
- Cowen, T. D., Meythaler, J. M., DeVivo, M. J., Ivie, C. S., III, Lebow, J., & Novack, T. A. (1995). Influence of early variables in traumatic brain injury on functional independence measure scores and rehabilitation length of stay and charges. *Archives of Physical Medicine and Rehabilitation*, 76(9), 797–803.
- Cullen, N. K., Crescini, C., & Bayley, M. T. (2009). Rehabilitation outcomes after anoxic brain injury: A case-controlled comparison with traumatic brain injury. *Physical Medicine and Rehabilitation*, 1(12), 1069–1076.
- Cullen, N. K., & Weisz, K. (2011). Cognitive correlates with functional outcomes after anoxic brain injury: A case-controlled comparison with traumatic brain injury. *Brain Injury*, 25(1), 35–43.
- DeVetten, G., Coutts, S. B., Hill, M. D., Goyal, M., Eesa, M., O'Brien, B., . . . MONITOR and VISION study groups. (2010). Acute corticospinal tract Wallerian degeneration is associated with stroke outcome. *Stroke*, 41(4), 751–756.
- Di Lazzaro, V., Profice, P., Pilato, F., Capone, F., Ranieri, F., Pasqualetti, P., . . . Dileone, M. (2010). Motor cortex plasticity predicts recovery in acute stroke. *Cerebral Cortex*, 20(7), 1523–1528.
- Dong, Y., Dobkin, B. H., Cen, S. Y., Wu, A. D., & Winstein, C. J. (2006). Motor cortex activation during treatment may predict therapeutic gains in paretic hand function after stroke. *Stroke*, 37(6), 1552–1555.
- Doyle, K. P., Simon, R. P., & Stenzel-Poore, M. P. (2008). Mechanisms of ischemic brain damage. *Neuropharmacology*, 55(3), 310–318.
- Draganski, B., & May, A. (2008). Training-induced structural changes in the adult human brain. *Behavioural Brain Research*, 192(1), 137–142.
- Duckworth, J. L., & Stevens, R. D. (2010). Imaging brain trauma. *Current Opinion in Critical Care*, 16(2), 92–97.
- Eliassen, J. C., Boespflug, E. L., Lamy, M., Allendorfer, J., Chu, W. J., & Szaflarski, J. P. (2008). Brain-mapping techniques for evaluating poststroke recovery and rehabilitation: A review. *Topics in Stroke Rehabilitation*, 15(5), 427–450.
- Fabricius, M., Fuhr, S., Bhatia, R., Boutelle, M., Hashemi, P., Strong, A. J., & Lauritzen, M. (2006). Cortical spreading depression and peri-infarct depolarization in acutely injured human cerebral cortex. *Brain*, 129(Pt 3), 778–790.
- Farmer, J. E., Donders, J., & Warschausky, S. (Eds.). (2005). *Treating neurodevelopmental disabilities: Clinical research and practice*. New York, NY: Guilford Press.
- Festa, J., & Lazar, R. M. (2009). *Neurovascular neuropsychology*. New York, NY: Springer Science and Business Media.
- Fierstra, J., Maclean, D. B., Fisher, J. A., Han, J. S., Mandell, D. M., Conklin, J., . . . Tymianski, M. (2011). Surgical revascularization reverses cerebral cortical thinning in patients with severe cerebrovascular steno-occlusive disease. *Stroke*, 42(6), 1631–1637.
- Fitzgerald, A., Aditya, H., Prior, A., McNeill, E., & Pentland, B. (2010). Anoxic brain injury: Clinical patterns and functional outcomes. A study of 93 cases. *Brain Injury*, 24(11), 1311–1323.
- Flanagan, S. R., Cantor, J. B., & Ashman, T. A. (2008). Traumatic brain injury: Future assessment tools and treatment prospects. *Neuropsychiatric Disease and Treatment*, 4(5), 877–892.
- Fong, T. G., Bogardus, S. T., Jr., Daftary, A., Auerbach, E., Blumenfeld, H., Modur, S., . . . Inouye, SK. (2006). Cerebral perfusion changes in older delirious patients using 99mTc HMPAO SPECT. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 61(12), 1294–1299.
- Gale, S. D., Baxter, L., Roundy, N., & Johnson, S. C. (2005). Traumatic brain injury and grey matter concentration: A preliminary voxel based morphometry study. *Journal of Neurology, Neurosurgery & Psychiatry*, 76(7), 984–988.
- Gale, S. D., Burr, R. B., Bigler, E. D., & Blatter, D. (1993). Fornix degeneration and memory in traumatic brain injury. *Brain Research Bulletin*, 32(4), 345–349.

- Gale, S. D., & Hopkins, R. O. (2004). Effects of hypoxia on the brain: Neuroimaging and neuropsychological findings following carbon monoxide poisoning and obstructive sleep apnea. *Journal of the International Neuropsychological Society*, 10(1), 60–71.
- Gale, S. D., Johnson, S. C., Bigler, E. D., & Blatter, D. D. (1994). Traumatic brain injury and temporal horn enlargement: Correlates with tests of intelligence and memory. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 7, 160–165.
- Gale, S. D., Johnson, S. C., Bigler, E. D., & Blatter, D. D. (1995). Nonspecific white matter degeneration following traumatic brain injury. *Journal of the International Neuropsychological Society*, 1(1), 17–28.
- Gale, S. D., & Prigatano, G. P. (2010). Deep white matter volume loss and social reintegration after traumatic brain injury in children. *Journal of Head Trauma Rehabilitation*, 25(1), 15–22.
- Girard, T. D., Jackson, J. C., Pandharipande, P. P., Pun, B. T., Thompson, J. L., Shintani, A. K., . . . Ely, E. W. (2010). Delirium as a predictor of long-term cognitive impairment in survivors of critical illness. *Critical Care Medicine*, 38(7), 1513–1520.
- Goodrich-Hunsaker, N. J., & Hopkins, R. O. (2010). Spatial memory deficits in a virtual radial arm maze in amnesic participants with hippocampal damage. *Behavioral Neuroscience*, 124(3), 405–413.
- Groswasser, Z., Cohen, M., & Costeff, H. (1989). Rehabilitation outcome after anoxic brain damage. *Archives of Physical Medicine and Rehabilitation*, 70(3), 186–188.
- Gunther, M. L., Beck, C. J., Morandi, A., Girard, T. D., Pandharipande, P., Jackson, J. C., . . . Hopkins, R. O. (2010). Quantitative brain MRI findings in critically ill patients with delirium. *Journal of Respiratory and Critical Care Medicine*, 181(Meeting Abstracts), A5354.
- Heimer, L. (1995). *Human brain and spinal cord second edition* (2nd ed.). New York, NY: Springer-Verlag.
- Hoeft, F., McCandliss, B. D., Black, J. M., Gantman, A., Zakerani, N., Hulme, C., . . . Gabrieli, J. D. E. (2011). Neural systems predicting long-term outcome in dyslexia. *Proceedings of the National Academy of Sciences USA*, 108(1), 361–366.
- Hopkins, R. O., Fearing, M. A., Weaver, L. K., & Foley, J. F. (2006). Basal ganglia lesions following carbon monoxide poisoning. *Brain Injury*, 20(3), 273–281.
- Hopkins, R. O., Gale, S. D., & Weaver, L. K. (2006). Brain atrophy and cognitive impairment in survivors of acute respiratory distress syndrome. *Brain Injury*, 20(3), 263–271.
- Hopkins, R. O., McCourt, A., & Cleavinger, H. B. (2003). White matter hyperintensities and neuropsychological outcome following traumatic brain injury. *Journal of the International Neuropsychological Society*, 9, 234.
- Hopkins, R. O., Tate, D. F., & Bigler, E. D. (2005). Anoxic versus traumatic brain injury: Amount of tissue loss, not etiology, alters cognitive and emotional function. *Neuropsychology*, 19(2), 233–242.
- Hopkins, R. O., Weaver, L. K., Collingridge, D., Parkinson, R. B., Chan, K. J., & Orme, J. F., Jr. (2005). Two-year cognitive, emotional, and quality-of-life outcomes in acute respiratory distress syndrome. *American Journal of Respiratory and Critical Care Medicine*, 171(4), 340–347.
- Humm, J. L., Kozlowski, D. A., James, D. C., Gotts, J. E., & Schallert, T. (1998). Use-dependent exacerbation of brain damage occurs during an early postlesion vulnerable period. *Brain Research*, 783(2), 286–292.
- Incalzi, R. A., Corsonello, A., Trojano, L., Pedone, C., Acanfora, D., Spada, A., . . . Rengo, F. (2008). Cognitive training is ineffective in hypoxemic COPD: A six-month randomized controlled trial. *Rejuvenation Research*, 11(1), 239–250.
- Jackson, A. C., Gilbert, J. J., Young, G. B., & Bolton, C. F. (1985). The encephalopathy of sepsis. *Canadian Journal of Neurological Sciences*, 12(4), 303–307.
- Jackson, J. C., Clune, H., Hoenig, M., Morey, M., Anderson, V., Denne, L., . . . Ely, E. W. (2010). The returning to everyday tasks utilizing rehabilitation networks (RETURN) trial: A pilot, feasibility trial including in-home cognitive rehabilitation of ICU survivors. *American Journal of Respiratory and Critical Care Medicine*, 181, A5359.
- Jackson, J. C., Girard, T. D., Gordon, S. M., Thompson, J. L., Shintani, A. K., Thomason, J. W., . . . Ely, E.

- W. (2010). Long-term cognitive and psychological outcomes in the awakening and breathing controlled trial. *American Journal of Respiratory and Critical Care Medicine*, 182(2), 183–191.
- Jackson, J. C., Gordon, S. M., Burger, C., Ely, E. W., Thomason, J. W., & Hopkins, R. O. (2003). Acute respiratory distress syndrome and long-term cognitive impairment: A case study. *Archives of Clinical Neuropsychology*, 18(7), 688.
- Jantzen, K. J., Anderson, B., Steinberg, F. L., & Kelso, J. A. (2004). A prospective functional MR imaging study of mild traumatic brain injury in college football players. *American Journal of Neuroradiology*, 25(5), 738–745.
- Jezzard, P., Matthews, P. M., & Smith, S. M. (2001). *Functional MRI: An introduction to methods*. New York, NY: Oxford University Press.
- Johnson, S. C., Bigler, E. D., Burr, R. B., & Blatter, D. D. (1994). White matter atrophy, ventricular dilation, and intellectual functioning following traumatic brain injury. *Neuropsychology*, 8(3), 307–315.
- Johnson, S. C., Pinkston, J. B., Bigler, E. D., & Blatter, D. D. (1996). Corpus callosum morphology in normal controls and traumatic brain injury: Sex differences, mechanisms of injury, and neuropsychological correlates. *Neuropsychology*, 10(3), 408–415.
- Kaplan, C. P. (1999). Anoxic-hypotensive brain injury: Neuropsychological performance at 1 month as an indicator of recovery. *Brain Injury*, 13(4), 305–310.
- Keller, T. A., & Just, M. A. (2009). Altering cortical connectivity: Remediation-induced changes in the white matter of poor readers. *Neuron*, 64(5), 624–631.
- Kesler, S. R., Hopkins, R. O., Blatter, D. D., Edge-Booth, H., & Bigler, E. D. (2001). Verbal memory deficits associated with fornix atrophy in carbon monoxide poisoning. *Journal of the International Neuropsychological Society*, 7(5), 640–646.
- Ketonen, L. M., & Berg, M. J. (1997). *Clinical neuroradiology: 100 maxims*. New York, NY: Oxford University Press.
- Kondo, K., Maruishi, M., Ueno, H., Sawada, K., Hashimoto, Y., Ohshita, T., . . . Matsumoto, M. (2010). The pathophysiology of prospective memory failure after diffuse axonal injury—Lesion-symptom analysis using diffusion tensor imaging. *BMC Neuroscience*, 11, 147.
- Kraus, M. F., Susmaras, T., Caughlin, B. P., Walker, C. J., Sweeney, J. A., & Little, D. M. (2007). White matter integrity and cognition in chronic traumatic brain injury: A diffusion tensor imaging study. *Brain*, 130(Pt 10), 2508–2519.
- Kress, J. P., Pohlman, A. S., O'Connor, M. F., & Hall, J. B. (2000). Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. *New England Journal of Medicine*, 342(20), 1471–1477.
- Lauritzen, M., Dreier, J. P., Fabricius, M., Hartings, J. A., Graf, R., & Strong, A. J. (2011). Clinical relevance of cortical spreading depression in neurological disorders: migraine, malignant stroke, subarachnoid and intracranial hemorrhage, and traumatic brain injury. *Journal of Cerebral Blood Flow & Metabolism*, 31(1), 17–35.
- Levin, H. S., Benavidez, D. A., Verger-Maestre, K., Perachio, N., Song, J., Mendelsohn, D. B., & Fletcher, J. M. (2000). Reduction of corpus callosum growth after severe traumatic brain injury in children. *Neurology*, 54(3), 647–653.
- Levin, H. S., Hanten, G., Roberson, G., Li, X., Ewing-Cobbs, L., Dennis, M., . . . Swank, P. (2008). Prediction of cognitive sequelae based on abnormal computed tomography findings in children following mild traumatic brain injury. *Journal of Neurosurgery: Pediatrics*, 1(6), 461–470.
- Levin, H. S., Mendelsohn, D., Lilly, M. A., Yeakley, J., Song, J., Scheibel, R. S., . . . Bruce, D. (1997). Magnetic resonance imaging in relation to functional outcome of pediatric closed head injury: A test of the Ommaya-Gennarelli model. *Neurosurgery*, 40(3), 432–440; discussion 440–431.
- Little, D. M., Kraus, M. F., Jiam, C., Moynihan, M., Siroko, M., Schulze, E., & Geary, E. K. (2010). Neuroimaging of hypoxicischemic brain injury. *NeuroRehabilitation*, 26(1), 15–25.
- Little, D. M., Kraus, M. F., Joseph, J., Geary, E. K., Susmaras, T., Zhou, X. J., . . . Gorelick, P. B. (2010). Thalamic integrity underlies executive dysfunction in traumatic brain injury. *Neurology*, 74(7), 558–

- Maguire, E. A., Gadian, D. G., Johnsrude, I. S., Good, C. D., Ashburner, J., Frackowiak, R. S., & Frith, C. D. (2000). Navigation-related structural change in the hippocampi of taxi drivers. *Proceedings of the National Academy of Sciences USA*, *97*(8), 4398–4403.
- Manns, J. R., Hopkins, R. O., Reed, J. M., Kitchener, E. G., & Squire, L. R. (2003). Recognition memory and the human hippocampus. *Neuron*, *37*(1), 171–180.
- Marshall, R. S., Zarah, E., Alon, L., Minzer, B., Lazar, R. M., & Krakauer, J. W. (2009). Early imaging correlates of subsequent motor recovery after stroke. *Annals of Neurology*, *65*(5), 596–602.
- Mascalchi, M., Petrucci, P., & Zampa, V. (1996). MRI of cerebellar white matter damage due to carbon monoxide poisoning: Case report. *Neuroradiology*, *38*(Suppl. 1), S73–74.
- Matthews, P. M. (2001). An introduction to functional magnetic resonance of the brain. In P. Jezzard, P. M. Matthews, & S. M. Smith (Eds.), *Functional MRI: An introduction to methods* (pp. 3–34). New York, NY: Oxford University Press.
- Morandi, A., Gunther, M. L., Vasilevskis, E. E., Girard, T. D., Hopkins, R. O., Jackson, J. C., . . . Ely, E. W. (2010). Neuroimaging in delirious intensive care unit patients: A preliminary case series report. *Psychiatry (Edgmont)*, *7*(9), 28–33.
- Mori, S., & Barker, P. B. (1999). Diffusion magnetic resonance imaging: Its principle and applications. *Anatomical Record (New Anatomy)*, *257*, 102–109.
- Mukherjee, P., Berman, J. I., Chung, S. W., Hess, C. P., & Henry, R. G. (2008). Diffusion tensor MR imaging and fiber tractography: theoretic underpinnings. *American Journal of Neuroradiology*, *29*(4), 632–641.
- Nishio, Y., Hashimoto, M., Ishii, K., & Mori, E. (2011). Neuroanatomy of a neurobehavioral disturbance in the left anterior thalamic infarction. *Journal of Neurology, Neurosurgery & Psychiatry*, Epub ahead of print.
- O'Donnell, P., Buxton, P. J., Pitkin, A., & Jarvis, L. J. (2000). The magnetic resonance imaging appearances of the brain in acute carbon monoxide poisoning. *Clinical Radiology*, *55*(4), 273–280.
- Ogawa, S., Lee, T. M., Kay, A. R., & Tank, D. W. (1990). Brain magnetic resonance imaging with contrast dependent on blood oxygenation. *Proceedings of the National Academy of Sciences USA*, *87*(24), 9868–9872.
- Parkin, A. J., Miller, J., & Vincent, R. (1987). Multiple neuropsychological deficits due to anoxic encephalopathy: A case study. *Cortex*, *23*(4), 655–665.
- Parkinson, R. B., Hopkins, R. O., Cleavinger, H. B., Weaver, L. K., Victoroff, J., Foley, J. F., & Bigler, E. D. (2002). White matter hyperintensities and neuropsychological outcome following carbon monoxide poisoning. *Neurology*, *58*(10), 1525–1532.
- Pascual-Leone, A., Cammarota, A., Wassermann, E. M., Brasil-Neto, J. P., Cohen, L. G., & Hallett, M. (1993). Modulation of motor cortical outputs to the reading hand of braille readers. *Annals of Neurology*, *34*(1), 33–37.
- Peskine, A., Picq, C., & Pradat-Diehl, P. (2004). Cerebral anoxia and disability. *Brain Injury*, *18*(12), 1243–1254.
- Pierallini, A., Pantano, P., Fantozzi, L. M., Bonamini, M., Vichi, R., Zylberman, R., . . . Bozzao, L. (2000). Correlation between MRI findings and long-term outcome in patients with severe brain trauma. *Neuroradiology*, *42*(12), 860–867.
- Porter, S. S., Hopkins, R. O., Weaver, L. K., Bigler, E. D., & Blatter, D. D. (2002). Corpus callosum atrophy and neuropsychological outcome following carbon monoxide poisoning. *Archives of Clinical Neuropsychology*, *17*(2), 195–204.
- Povlishock, J. T., & Katz, D. I. (2005). Update of neuropathology and neurological recovery after traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *20*(1), 76–94.
- Prigatano, G. P., & Gale, S. D. (2011). The current status of postconcussion syndrome. *Current Opinion in Psychiatry*, *24*(3), 243–250.
- Puig, J., Pedraza, S., Blasco, G., Daunis, I. E. J., Prats, A., Prados, F., . . . Serena, J. (2010). Wallerian

- degeneration in the corticospinal tract evaluated by diffusion tensor imaging correlates with motor deficit 30 days after middle cerebral artery ischemic stroke. *American Journal of Neuroradiology*, 31(7), 1324–1330.
- Pusswald, G., Fertl, E., Falzl, M., & Auff, E. (2000). Neurological rehabilitation of severely disabled cardiac arrest survivors. Part II. Life situation of patients and families after treatment. *Resuscitation*, 47(3), 241–248.
- Quallo, M. M., Price, C. J., Ueno, K., Asamizuya, T., Cheng, K., Lemon, R. N., & Iriki, A. (2009). Gray and white matter changes associated with tool-use learning in macaque monkeys. *Proceedings of the National Academy of Science USA*, 106(43), 18379–18384.
- Reider, G., II, Groswasser, Z., Ommaya, A. K., Schwab, K., Pridgen, A., Brown, H. R., . . . Salazar, AM. (2002). Quantitative imaging in late traumatic brain injury. Part I: Late imaging parameters in closed and penetrating head injuries. *Brain Injury*, 16(6), 517–525.
- Scheid, R., Preul, C., Gruber, O., Wiggins, C., & von Cramon, D. Y. (2003). Diffuse axonal injury associated with chronic traumatic brain injury: evidence from T2\*-weighted gradient-echo imaging at 3 T. *American Journal of Neuroradiology*, 24(6), 1049–1056.
- Schlaug, G., Marchina, S., & Norton, A. (2009). Evidence for plasticity in white-matter tracts of patients with chronic Broca's aphasia undergoing intense intonation-based speech therapy. *Annals of the New York Academy Sciences*, 1169, 385–394.
- Schmahmann, J. D., & Pandya, D. N. (1997). The cerebrocerebellar system. *International Review of Neurobiology*, 41, 31–60.
- Schmidt, J. G., Drew-Cates, J., & Dombovy, M. L. (1997). Anoxic encephalopathy: Outcome after inpatient rehabilitation. *Journal of Neurological Rehabilitation*, 11, 189–205.
- Scholz, J., Klein, M. C., Behrens, T. E., & Johansen-Berg, H. (2009). Training induces changes in white-matter architecture. *Nature Neuroscience*, 12(11), 1370–1371.
- Seitz, R. J. (2010). How imaging will guide rehabilitation. *Current Opinion in Neurology*, 23(1), 79–86.
- Seitz, R. J., & Donnan, G. A. (2010). Role of neuroimaging in promoting long-term recovery from ischemic stroke. *Journal of Magnetic Resonance Imaging*, 32, 756–772.
- Sharshar, T., Carlier, R., Bernard, F., Guidoux, C., Brouland, J. P., Nardi, O., . . . Annane, D. (2007). Brain lesions in septic shock: A magnetic resonance imaging study. *Intensive Care Medicine*, 33(5), 798–806.
- Shioiri, A., Kurumaji, A., Takeuchi, T., Matsuda, H., Arai, H., & Nishikawa, T. (2010). White matter abnormalities as a risk factor for postoperative delirium revealed by diffusion tensor imaging. *American Journal of Geriatric Psychiatry*, 18(8):743–753.
- Smith, D. H., Meaney, D. F., & Shull, W. H. (2003). Diffuse axonal injury in head trauma. *Journal of Head Trauma Rehabilitation*, 18(4), 307–316.
- Stinear, C. (2010). Prediction of recovery of motor function after stroke. *Lancet Neurology*, 9(12), 1228–1232.
- Stone, J. R., Okonkwo, D. O., Singleton, R. H., Mutlu, L. K., Helm, G. A., & Povlishock, J. T. (2002). Caspase-3-mediated cleavage of amyloid precursor protein and formation of amyloid beta peptide in traumatic axonal injury. *Journal of Neurotrauma*, 19(5), 601–614.
- Suchyta, M. R., Jephson, A., & Hopkins, R. O. (2010). Neurologic changes during critical illness: Brain imaging findings and neurobehavioral outcomes. *Brain Imaging and Behavior*, 4(1), 22–34.
- Taylor, D. G., & Bushell, M. C. (1985). The spatial mapping of translational diffusion coefficients by the NMR imaging technique. *Physics in Medicine and Biology*, 30(4), 345–349.
- Turkstra, L. S., Holland, A. L., & Bays, G. A. (2003). The neuroscience of recovery and rehabilitation: What have we learned from animal research? *Archives of Physical Medicine and Rehabilitation*, 84(4), 604–612.
- Ueda, T., Yuh, W. T., Maley, J. E., Quets, J. P., Hahn, P. Y., & Magnotta, V. A. (1999). Outcome of acute ischemic lesions evaluated by diffusion and perfusion MR imaging. *American Journal of Neuroradiology*, 20(6), 983–989.
- Ulug, A. M., Moore, D. F., Bojko, A. S., & Zimmerman, R. D. (1999). Clinical use of diffusion-tensor

- imaging for diseases causing neuronal and axonal damage. *American Journal of Neuroradiology*, 20(6), 1044–1048.
- Vasa, R. A., Grados, M., Slomine, B., Herskovits, E. H., Thompson, R. E., Salorio, C., . . . Gerring, J. P. (2004). Neuroimaging correlates of anxiety after pediatric traumatic brain injury. *Biological Psychiatry*, 55(3), 208–216.
- Wardlaw, J. M., & Statham, P. F. (2000). How often is haemosiderin not visible on routine MRI following traumatic intracerebral haemorrhage? *Neuroradiology*, 42(2), 81–84.
- Welsh, J. P., Yuen, G., Placantonakis, D. G., Vu, T. Q., Haiss, F., O’Hearn, E., . . . Aicher, S. A. (2002). Why do Purkinje cells die so easily after global brain ischemia? Aldolase C, EAAT4, and the cerebellar contribution to posthypoxic myoclonus. *Advances in Neurology*, 89, 331–359.
- Wilde, E. A., Hunter, J. V., Newsome, M. R., Scheibel, R. S., Bigler, E. D., Johnson, J. L., . . . Levin, H. S. (2005). Frontal and temporal morphometric findings on MRI in children after moderate to severe traumatic brain injury. *Journal of Neurotrauma*, 22(3), 333–344.
- Williams, T. A., Dobb, G. J., Finn, J. C., Knuiman, M. W., Geelhoed, E., Lee, K. Y., & Webb, S. A. (2008). Determinants of long-term survival after intensive care. *Critical Care Medicine*, 36(5), 1523–1530.
- Yen, P. S., Teo, B. T., Chiu, C. H., Chen, S. C., Chiu, T. L., & Su, C. F. (2009). White matter tract involvement in brain tumors: A diffusion tensor imaging analysis. *Surgical Neurology*, 72(5), 464–469; discussion 469.
- Yeo, S. S., & Jang, S. H. (2011). The effect of thalamic hemorrhage on the fornix. *International Journal of Neuroscience*, 121(7), 379–383.
- Yokota, H., Ogawa, S., Kurokawa, A., & Yamamoto, Y. (2003). Regional cerebral blood flow in delirium patients. *Psychiatry and Clinical Neurosciences*, 57(3), 337–339.
- Young, G. B. (1995). Neurologic complications of systemic critical illness. *Neurologic Clinics*, 13(3), 645–658.

## System and Family Support

*Margaret Semrud-Clikeman, Jesse Bledsoe, and Lisa Vroman*

Children and adults with severe illness or neurological impairment require support throughout the recovery process. Such therapeutic support requires coordination of medical, educational, and family resources and services. For some disorders (i.e., seizure disorders, severe traumatic brain injury [TBI], and chronic illnesses), support will be needed throughout the life span. For others (mild to moderate TBI, localized stroke), care may be required for a few months to a few years. In each case, coordination of care within the systems involved for that person is important for optimal recovery.

Rehabilitation is a specific branch of medicine and education that has a major aim of assisting patients to maximize their recovery from physical and mental disorders (Russman, 1990). The rehabilitation plan is generally developed by a team of professionals, which are initially in the medical setting, and then moves into the school and home settings. In the medical field, rehabilitation is generally managed by physicians, nurses, and specialists (physical therapist, occupational therapist, speech/language therapist). In the school setting, rehabilitation is required to follow federal and state mandates for programming.

The relation between systems has been described as a mesosystem (overlap of two or more microsystems) that connects microsystems (medical, family, school; Farmer & Drewel, 2006). Linkages among these microsystems are the result of communication among the systems or the influence on other microsystems by actions within one care setting. These linkages include

communications, such as transition to school from a hospital or clinic rehabilitation center (Semrud-Clikeman, 2001). Such linkages are crucial for success in working with children and adults, and particularly when dealing with a large number of individual providers who may be fragmented and unhelpful to the child or adult and his or her family (Seid, Sobo, Gelhard, & Varni, 2004).

An aspect of rehabilitation that has not often been discussed in the literature is the ethical implications of selected aspects of rehabilitation. There are several areas that can pose an ethical dilemma (Flett & Stoffell, 2003). Similar to a later discussion in this chapter on treatments, the use of evidence-based practice is an important ethical issue. There are many treatments for those with severe illness or neurocognitive impairment that have poor evidence of efficacy. In addition, an ethical aspect that will be discussed throughout this chapter is the emphasis on functional improvement and quality-of-life issues rather than solely on neurological/neuropsychological deficit or medical diagnosis. Providing appropriate information to the adult or the caregiver of the child is very important, and the delivery of this information needs to be culturally sensitive as well as informative. Tied to providing this information is the need to assist parents and caregivers in understanding what the realistic prognosis of the child or adult is without destroying hope. If the child is intellectually or physically disabled following the injury and these injuries are not likely to dissipate, it is important for the neuropsychologist and/or therapist to communicate this information in a helpful manner.

The purpose of this chapter is to explore the different systems and case management that have been empirically found to be of most value to recovery. For the most part, this chapter will primarily focus on children and adolescents. The chapter is divided into the three main systems involved with children and adolescents; medical, school, and family. Each section will discuss issues that arise in the selected system, as they pertain to success in neurorehabilitation.

## MEDICAL SETTINGS

Initial diagnosis and prognosis of the child is provided by the physician, for children with neurological disorders it is generally the neurologist (Swaiman, Ashwal, & Ferriero, 2006), for children recovering from cancer it is the oncologist (Neglia et al., 2006), and for other types of disorders the specific specialist. Prior to the past 20 years, the role of the physician ended with diagnosis but with increased access to rehabilitation and appropriate interventions, the role of the neurologist and other specialists has increased to



include what is termed “restorative neurology” (Russman, 1990). Another more recent trend, particularly in pediatric rehabilitation and more recently with adults, is the view that developmental issues are key to understanding the child’s response to rehabilitation and that the inclusion of the family and school in the process is primary (Molnar, 1985).

Studies have found that children with disabilities compared to those without have four times the number of hospitalizations, twice the number of visits to the emergency room, twice the number of physician visits, five times the number of visits to ancillary specialists (occupational therapy, physical therapy, etc.), and three times the number of prescription medicines (Newacheck, Inkelas, & Kim, 2004). The level of medical support required for these children is often borne by private insurance companies as well as out-of-pocket funding from the families, adding to an already high level of stress (McMenamy & Perrin, 2004). As such, the burden on the family is immense.

### Care Teams

One program that has been developed is called the Missouri Partnership for Enhanced Delivery of Services (MO-PEDS; Farmer, Marien, Clark, Sherman, & Selva, 2004). This program was developed to serve children with significant health concerns. An initial survey found that the main issues faced in dealing with medical settings were a need for information, social support, and community services. This program involves a care team that generally consists of a nurse practitioner, physician, and family members. A needs assessment is first conducted to identify unmet needs and then an Individual Care Plan is developed. This plan includes short-term goals, assistance in seeking out additional needed services, and funding to obtain these services.

Similar to the MO-PEDS program was another one developed at the University of Minnesota called U Special Kids (USK). This program was developed to provide transition care from the hospital to a primary care home within the child’s community (Kelly, Golnik, & Cady, 2008). Working with children with complex and severe medical needs indicates these children require additional support requiring complex care coordination that can not only assist with rehabilitation, but also reduce additional utilization of medical facilities. Similarly, work in Michigan (Naar-King, Siegel, Smyth, & Simpson, 2003), Illinois (Rosenberg et al., 2005), South Carolina (Martin et al., 2007), and California (Smith, Layne, & Garrell, 1994) has found that care coordination not only improved the outcome for these children, but also indicated that for

children with serious and debilitating illness or injuries, complex care coordination was crucial for success following release from the hospital. One aspect that was found to be particularly important was the provision of information to the family, school, and community medical professionals as to the level of functioning of the child and the impact on his or her recovery.

The conclusion from these studies, particularly for children with severe needs, is that neuropsychologists can be particularly helpful in identifying those children who need in-depth and interdisciplinary care not currently in place. One professional who can assist in the translation of findings to other nonmedical professionals, as well as in supporting the school and family, is the neuropsychologist. The neuropsychologist is in a unique position to assist in communication of treatment plans across disciplines (microsystems) and to facilitate the development of collaborative teams. Subsequent to discharge from a rehabilitation facility, a neuropsychologist can play a key role in interpreting the findings from the assessment, as well as involving the community care coordinators in developing and identifying appropriate goals and treatment strategies across microsystems. One of the needs that may not be adequately addressed by medical personnel may be mental health functioning, particularly because it has been found that family psychosocial risk factors are strongly predictive of response to rehabilitation (Kazak et al., 2003). The neuropsychologist with knowledge of rehabilitation needs as well as mental health needs is in an excellent position to provide guidance and support to determine these needs.

In addition to providing clinical support and guidance, the neuropsychologists in a medical setting can be instrumental in implementing service delivery programs that are particularly helpful to families and schools. One method for delivering service to rural families and schools may be through telehealth technology (Farmer & Muhlenbruck, 2001). These methods can assist in providing the needed information to communities and for patients, where such skills are not readily present.

### **Cultural Concerns**

Appropriate services for the rehabilitation of children with severe disabilities or injury requires a team approach, as discussed above. Providing these services to racial and ethnic minorities, as well as to families from varying socioeconomic strata is also challenging. Although ethnic and minority differences may be more obvious, there are major differences within the White American population with

regard to geographic regions, rural or urban, religious beliefs, education level, and socioeconomic status (Echemendia & Westerveld, 2006). Neuropsychological assessment frequently is a crucial element in planning and implementing rehabilitation. Cultural and ethnic issues are an area of concern for assessment, as well as for rehabilitation. If the assessment reflects cultural or ethnic biases, then the rehabilitation will be founded on faulty information.

Aspects in the assessment that require attention by the neuropsychologist include the primary language of the patient, the level of acculturation if the patient is an immigrant, the comfort with the assessment process, and the motivation of the patient to complete the tasks (Brown, McCauley, Levin, Contant, & Boake, 2004). Differences have been found in how various minorities cope following an illness or injury to a family member. African American families have generally been found to use avoidant coping strategies and/or religion, whereas Whites generally used acceptance (Yeates et al., 2002). It appears crucial for the neuropsychologist and care team to develop appropriate rehabilitation programs that take into account the differences in acceptance of mental health and medical services that may exist in differing cultural perspectives. It has been strongly suggested that for a rehabilitation program to be successful, the practitioner must understand the view and response to the injury, and the coping strategies used by that particular family and work to develop appropriate rapport in order for the rehabilitation to be successful (Armengol, 1999).

### **Rehabilitation Strategies Within the Medical Facility**

Although it is beyond the scope of this chapter to discuss in detail the various types of rehabilitation, rehabilitative medical management has found certain treatments to be empirically validated. The use of long-term prophylactic medication with anticonvulsants is frequently used with severe TBI and for some patients, 10 days of such treatment is used (Ylvisaker et al., 2005). Medications generally used that do not impair cognition include carbamazepine, valproic acid, or clobazam/topiramate. Aggression that can occur with certain types of head injury or disorders may be treated with propranolol, resperidone, or selective serotonin reuptake inhibitors (SSRIs), (Prozac, etc.) (Semrud-Clikeman, 2001). At times, depression can be present and may need supportive counseling or medication depending on the severity. Fatigue is another common difficulty found following TBI, as well as following treatment for cancer and other central nervous system (CNS) disorders. Some have found improved sleep

routines, physical activity, and conditioning to be helpful (Bateman et al., 2001). Ineffective treatments include hyperbaric oxygen therapy, craniosacral therapy, chiropractic therapy, or functional electrical stimulation to improve spasticity (Ylvisaker et al., 2005).

A group of techniques included under cognitive rehabilitation have generally been found to be helpful for adults, particularly in remediation of attention, memory, functional communication, and executive functioning (Cicerone, Faust, Beverly, & Demakis, 2009), with less research for children at present (Sohlberg & Mateer, 2001). Teaching of metacognitive strategies, use of behavioral management techniques, rehearsed practice, cognitive-behavioral (CB) therapy, and parent training have the strongest empirical support for rehabilitation (Butler et al., 2008; Butler & Haser, 2006). Particular attention has been suggested for developing appropriate cognitive control over other skills (i.e., attention, memory, organization), as a major emphasis for rehabilitation (Ylvisaker et al., 2005).

There has been a major movement away from the traditional manner of providing rehabilitation through the emphasis on underlying neuropsychological impairment and restoration, or skills through cognitive exercises and computer training programs (discrete method) to providing rehabilitation that is embedded within the child's everyday life and delivered within clinical settings (context-sensitive and functional method) (Ylvisaker et al., 2005). Studies of the discrete method have not found improvement in the individual functions or generalizing to additional contexts (Park & Ingles, 2001). In contrast, contextualized interventions, particularly those that are embedded in the everyday routine of the child and supported by his or her family, have been empirically supported (Feeney, Ylvisaker, Rosen, & Green 2005). This approach has also been found to be helpful for adults, as well as being cost-effective (Feeney, Ylvisaker, Rosen, & Green, 2001). Many of these interventions can be provided either in the school or in the family.

## **FAMILY SYSTEM**

### **Family-Centered Service**

The family plays a pivotal role throughout the pediatric rehabilitation process. Although the child and family must travel to the rehabilitation center, often a hospital, care for the child does not end there. Specialized care must be provided outside the hospital setting in order for the rehabilitation to be as successful as

possible. The rehabilitation approach termed *family-centered service* (FCS) is a movement away from the traditional, professional-centered service (King, Teplicky, King, & Rosenbaum, 2004). FCS can be defined as a method of treatment that incorporates the family into each phase of the rehabilitation process (King, King, Rosenbaum, & Goffin, 1999; King et al., 2004).

Pediatric FCS was developed in the mid-1980s as a reaction to family's desire to play a more central role in the treatment process (Naar-King & Donders, 2006). In contrast to the traditional, professional-centered service, FCS involves the family in each aspect of the rehabilitation process, from treatment planning to the implementation of the treatment plan. Family-centered rehabilitation was founded on the ideology that the family should be empowered in order to best care for their child. The parents, or primary caregivers, are provided with complete information regarding treatment plans, control in all treatment decisions, and are treated respectfully by the FCS team. Thus, this approach to pediatric rehabilitation encompasses three characteristics of caregiving: (a) parents best understand their children, (b) each family is unique, and (c) children thrive in supportive environments (King et al., 2004; Rosenbaum, King, Law, King, & Evans, 1998). Under this model, it should also be noted that as the child ages, it may become appropriate to include the child in decisions made about his or her care. In time, the child may be able to weigh-in on the treatment decisions that were initially made on his or her behalf (Naar-King & Donders, 2006).

Incorporating the needs of the family in the design of the rehabilitation program will help ensure that the treatment is feasible. It will also aid in treatment compliance and follow-through on completion of treatment recommendation (Beaulieu, 2002). Some evidence exists for the effectiveness of family-supported care, as compared to traditional, clinician-delivered care in the treatment of children with TBI (Braga et al., 2005). Findings from this study suggest parents in the family-supported intervention were able to provide both physical and cognitive interventions in the family's home, and only the family-supported intervention group demonstrated clinically significant improvement in physical and cognitive outcome measures. The remainder of this section will discuss family's and professionals' satisfaction with FCS, the burdens families face when a child must receive rehabilitation services, siblings of children with disabilities, and the home-school collaboration. See Table 14.1 for a summary of articles on FCS.

**TABLE 14.1 Published Articles on FCS**

AUTHORS	PARTICIPANTS	OUTCOME
Farmer et al. (2004)	175 children, ages 0 to 17 years Dx chronic health condition	Families with minority-group status, low SES, little social support, and high perception of burden may need additional support.
King et al. (1999)	Parents of children w/ special needs 3 samples: (1) 330 parents, (2) 151 parents, (3) 164 parents	Parents made more comments about process (e.g., support, competence) not structure (e.g., access, cost). Parents made more <i>like</i> than <i>dislike</i> comments. Satisfied parents made more <i>like</i> comments and dissatisfied parents made more <i>dislike</i> comments.
King et al. (2001)	164 parents of children w/ neurodev. dis.	FCS predicted parents' satisfaction with services, emotional welfare, & stress. Child behavior problems predicted parental welfare. Family functioning/ social support predicted parent's welfare.
Naar-King et al. (2002)	Parents ( <i>n</i> = 345), staff ( <i>n</i> = 67), children > 8 years ( <i>n</i> = 63)	On the general satisfaction scale, 94% of parents were satisfied and 68% of staff were satisfied. The children's scores did not meet the 80% criterion.
Osberg et al. (1996)	120 children, ages 0 to 18 yrs. Hospital stay ≥ 1 day	At 1-month follow-up, 17.5% of families had one injury-related financial problem. At 6-month follow-up, 20% of families had one injury-related financial problem.
Sperry et al. (1999)	30 parents in ASD focus group w/ child dx w/ ASD, aged 2 to 30 yrs. 22 professionals	Parents and professionals agreed on topics of finance, family support, training, collaboration, early identification, advocacy, equity, and inclusion. Areas of diversion were primarily on degree of importance placed on inclusion.
Wade et al. (1996)	Families of children w/ severe TBI ( <i>n</i> = 44), moderate TBI ( <i>n</i> = 52), or orthopedic injury ( <i>n</i> = 69)	Families of children with severe TBI reported more stress. Both TBI groups reported more psychological distress than other two groups. High levels of parental psychological distress were related to maladaptive coping strategies (e.g., venting).

Abbreviations ASD = Autism spectrum disorder; Dx = Diagnosis; FCS = Family-centered service; Neurodev. Dis. = Neurodevelopmental disorder; SES = Socioeconomic status; TBI = Traumatic brain injury.

*Satisfaction with FCS.* The evaluation of rehabilitation programs is essential, as it is critical to determine whether the program is being satisfactorily and successfully implemented (Naar-King & Donders, 2006). In a study evaluating parent, child, and staff satisfaction with a collaborative approach to treatment for children with special health care needs, 94% of parents were satisfied with the care their child received (Naar-King, Siegel, & Smyth, 2002).

Additionally, 88% of parents felt the time involved in the treatment was worthwhile. Close to 80% of children were satisfied with the collaborative treatment approach, but satisfaction did not reach the prescribed 80% satisfaction mark. Sixty-eight percent of staff members were satisfied with the treatment approach, and 76% of staff members were satisfied with the team environment. These findings indicate the primary caregivers were pleased with the additional time spent on the rehabilitation process. The children, however, were less satisfied with their treatment, suggesting that they may need to be more involved in the treatment-planning process. Additionally, staff members were less satisfied with the approach to treatment, which is a barrier to treatment delivery (Naar-King & Donders, 2006).

Similarly, an investigation into the main elements of parents' satisfaction and dissatisfaction with their child's rehabilitation services indicated that satisfied parents commonly cited respectful and supportive care, skilled staff, availability of services, and access to information as reasons for their satisfaction (King, Cathers, King, & Rosenbaum, 2001). Dissatisfied parents commonly noted the reasons for their dissatisfaction were disrespectful and unsupportive care, inability to access services, and uncoordinated care. These results indicate the interpersonal skills of the staff play a critical role in the parents' satisfaction with their child's care. The medical knowledge the health care providers offer is of importance to the family; however, it is also evident the family values compassionate and collaborative care.

Research on the outcomes of parent and provider focus groups regarding services for children with autism spectrum disorders (ASDs) reveals that parents discussed the need for collaboration, equity, family support, and financial guidance (Sperry, Whaley, Shaw, & Brame, 1999). Specifically, parents cited the desire to be involved in the treatment process and expressed their need to be engaged in a relationship with the care providers in which they are respected and recognized as valuable members of the treatment team. It is critical that both the care providers and parents maintain open communication with one another in order to participate in a respectful and beneficial relationship. Parents also expressed the need to be more completely involved in the decisions made regarding services their children receive. Such treatment decisions greatly impact not only the child, but also the family unit as a whole. Therefore, the parents desired more control over these decisions. Relatedly, parents requested that service providers recognize the strengths and potential in their children and approach services for their child from a strength-based, not a deficit-based,

perspective. Finally, financial concerns were noted among parents. Support regarding funding options for services such as speech and occupational therapy was raised from parents.

These results are similar to those discovered in a survey of caregivers of children with a severe emotional disorder on family and professional collaboration (DeChillo, Koren, & Schultze, 1994). The results of the survey revealed four main elements of collaboration: supportive relationships, practical service arrangements, open exchange of information, and a flexible approach to measuring the success or failure of the treatment. Specifically, parents reported that they would like to be included in the treatment process and valued as a member of the treatment team. Parents also desired information and assistance with funding and coordinating services. Adequate information on service options and flexibility on the part of the professional in changing treatment due to parental feedback were also desired features of collaboration with professionals.

### **Family Burden**

It is also important to assess psychological distress in parents of children with severe illness and neurocognitive disabilities. Specifically, parents of children with moderate to severe TBI reported significant psychological distress 1 month after their child experienced TBI (Wade, Taylor, Drotar, Stancin, & Yeates, 1996). In this study, few parents or children were receiving any form of counseling. The authors conjecture that the failure to seek counseling may be attributed to the parents' inability to express their emotional needs. Or perhaps, psychological distress is viewed by the parents as normative and therefore they do not consider seeking psychological assistance. Others have found that 41% of parents were under clinically significant stress, but most were not receiving counseling (Hawley, Ward, Magnay, & Long, 2003). These results indicate that parents may benefit from receiving information on the emotional difficulties that frequently arise following pediatric TBI. Professionals must inquire about the parents' emotional well-being and not rely on the parents to discuss their emotional distress.

The pediatric rehabilitation process not only takes a toll on the child, but also on the family unit as a whole. Having a child who requires services can create both an emotional and financial burden on the family. In particular, caregivers of children with chronic health conditions (e.g., cancer, mental disorders, nervous system disorders) frequently report the need for more information on the child's condition and available services (Farmer et al., 2004).



Additionally, assistance with coordinated care was desired by 52% of caregivers, and more than one third of families receiving coordinated care were dissatisfied with this service. Identified predictors of unmet family needs include low socioeconomic status (SES), ethnic minority status, poor child functioning, perceptions of limited social support and high family burden, and dissatisfaction with health care services (Farmer et al., 2004). Health care providers must be cognizant of these risk factors and be prepared to counsel families on how to ensure their needs are met.

The coping strategies families favor may not be most appropriate, given the condition the child is suffering from. For example, in an investigation of coping strategies utilized by families with a child with TBI, it was determined that emotion-focused strategies were most beneficial for the caregivers and family units as a whole (Wade et al., 2001). The emotion-focused coping strategies are those that utilize social support networks to receive support. The use of these strategies and also the use of humor were associated with reduced distress following TBI. However, active coping strategies, or those in which one attempts to remedy the situation, resulted in more distress following the TBI.

### **Financial Burden**

Financial difficulties are also common among families with a disabled child. Preserving a stable work routine often becomes difficult following pediatric trauma (Osberg, Kahn, Rowe, & Brooke, 1996). Middle-class families, not low-income families, are often the most financially burdened (Osberg et al., 1996). This burden may be attributed to their ineligibility for many services low-income families can receive, as well as limited financial means compared to high-income families. Consequently, it should not be assumed that only low-SES families are in need of counseling on the array of financial and work problems that typically arise with pediatric trauma. Also, there is some evidence to suggest that the degree of financial difficulty may be related to severity of injury (Hawley et al., 2003). For example, 69% of families had a loss of income. This is in contrast to 21% of families with a child with mild injury.

### **Cultural Considerations**

As the family plays a central role in the rehabilitation process, it is important to incorporate the family's culture into the rehabilitation program. Health care providers must be culturally sensitive and must work closely with the child's family to understand any potential barriers to treatment, as well as opportunities

to adapt the rehabilitation to the family's culture in such a way as to increase the success of the treatment. This will become of increasing importance as the United States continues to become ethnically diverse, as most neuropsychologists are Caucasian, English-speaking, and utilize a Eurocentric model of treatment (Echemendia, 2004). Eurocentric models emphasize the role of the individual, whereas collective models emphasize the needs of the group (e.g., family). As different ethnic and cultural groups vary in the degree to which extended family is involved in the child's life, and now rehabilitation, health care providers must make a point to inquire about the role of the family. It is critical that the health care professionals not make assumptions about the family's cultural background, but instead, speak directly to the family (Naar-King & Donders, 2006).

Cultural sensitivity is key to ensuring the comfort of the child and the family, but it is also a matter of treatment efficacy (Echemendia & Westerveld, 2006). The vast majority of the treatment regimen ensues in the home. It is therefore critical that any treatment program be developed with the family needs, cultural requirements, and lifestyle in mind. A thoughtfully designed, culturally sensitive rehabilitation program is more likely to be implemented in the home and is thus more likely to result in positive outcomes.

### Siblings

The mental health and well-being of siblings of children who require rehabilitation is also important for successful treatment. Siblings are also often under stress; however, their needs may be pushed aside to care for the child in treatment. In a study investigating adjustment of siblings with developmental disabilities, it was determined that the family environment was predictive of siblings' behavior problems (Dyson, Edgar, & Crnic, 1989). Supportive family environments that allowed for open communication and ability to discuss feelings were predictive of fewer behavior problems. Also, it has been noted that siblings' self-concept is predicted by parental stress. In families where the parents are highly stressed, siblings are more likely to have a low self-concept (Semrud-Clikeman, 2001).

Siblings of children with disabilities may have unique needs but lack the ability to convey their needs. Young children, in particular, may not possess the appropriate language necessary to make their needs known. Siblings may not have the emotional development to understand or express their emotions. Additionally, siblings may engage in maladaptive coping strategies, such as self-

blame, wishful thinking, and avoidance, in response to the increased attention required by the patient (Orsillo, McCaffrey, & Fisher, 1993). Consequently, the responsibility largely falls to the family to ensure that siblings receive the support they need. Care providers should make an effort to inform parents that although some siblings may adjust well to changes in family functioning, others may need additional support and guidance. Caring for the physical and emotional needs of the child with the disability can be emotionally exhausting for caregivers and caring for the needs of other children may be an additional stressor for some families. Support groups for siblings exist (e.g., Sib-shop) and may be a resource for some families. The following section will discuss appropriate interventions and issues within the school setting for children with significant medical needs.

## **SCHOOL REINTEGRATION AFTER BRAIN INJURY**

Most of the research on reentry to school has been conducted with children with TBI. Over 1 million school-aged children in the United States experience TBI or brain-related injuries each year, with estimates of 130,000 requiring special education needs (Batchelor & Dean, 1996; D'Amato & Rothlisberg, 1996; Glang, Tyler, Pearson, Todis, & Morvant, 2004). According to the United States Department of Education, fewer than 5% of children aged 6 to 21 with acquired brain injury received special education or related services (United States Department of Education, 2006). It is also estimated that 61.9% of adolescents with TBI will graduate from high school and nearly 23% will drop out. These negative outcomes are likely due to disruptions in skills important for academic success and social or emotional relationships, including memory, movement, expressive and receptive language, attention, and behavior (Semrud-Clikeman, 2001). Others suggest insufficient training and education for teachers and school psychologists, as well as poorly designed standardized assessments for children with cognitive impairments, are related to negative outcomes for children who experience TBI (Walker, Boling, & Cobb, 1994; Ylvisaker et al., 2005). For these reasons, it is essential for well-coordinated and preemptive planning on the part of the family, health care professionals, and school personnel during the child's transition from hospital to school. The purpose of the following section is to discuss important issues related to successful school reintegration for children with TBI, including school services, assessment strategies, and educational interventions. For this chapter, we will define children who experience head injuries or those with traumatic head injuries relating to treatment for cancer,

drowning, or coma as TBI.

## School Services

*IDEA 2004.* The Individuals with Disabilities Education Act of 1990 required that children with disabilities, including TBI, receive special education services. There are 13 categories under the most recent report (e.g., IDEA 2004) used to classify students with disabilities: autism, deaf–blindness, developmental delays, emotional disturbance, hearing impairments, mental retardation, multiple disabilities, orthopedic impairments, other health impairment, speech or language impairment, visual impairment, and TBI. According to IDEA 2004, children with disabilities should receive educational services in a “least restrictive environment” meaning they should be educated among their nonaffected peers as much as possible and should have access to the same services and educational programs. The IDEA also allows for the correct classification of children with disabilities; in the past, it was not uncommon for children with TBI to be misclassified as mentally retarded, emotionally disturbed, or learning disabled. States are required to follow the federal definition of TBI according to IDEA, which states TBI is caused by “an acquired injury to the brain caused by an external physical force.” Children with *internal* damage caused by tumors, metabolic disorders, anoxia, stroke, or brain infections, however, may not qualify for special education resources according to some states’ definitions but should qualify under the category of Other Health Impaired ([Katsiyannis & Conderman, 1994](#)). In other cases, these children are classified under the category of TBI. Thus, it is important for parents, educators, medical personnel, and psychologists working with the child to be familiar with their states’ definition of TBI, according to IDEA 2004.

*504 Plan.* The 504 Plan, part of the Rehabilitation Act of 1973, mandates federally funded schools to provide proper accommodations to students with disabilities. In order for a student to qualify for 504 Plan services, the student must have a physical or mental impairment that “substantially limits one or more major activities, has a record of the impairment, or is regarded as having an impairment.” Some academic accommodations that could be included in a student’s 504 Plan include extended time on tests, preferential classroom seating, electronic reading resources, individualized testing environments (e.g., student may be allowed to take tests in an environment other than the classroom like an office, where it is quiet and less distracting), or the use of audio taped lectures (see [Blosser & DePompei, 1994](#); [Semrud-Clikeman 2001](#) for recommendations).

Table 14.2 provides a comparison among IDEA, 504, and IEP.

**TABLE 14.2 Federal and State Services for Students With Disabilities**

SERVICE	PROVIDER	PURPOSE
IDEA 2004	Federal Law	Identification and classification of students with disabilities in order to ensure services.
504 Plan	Federal Law	Requires federally funded schools to provide reasonable accommodations for students with disabilities.
IEP	School System and Family	The IEP includes a comprehensive assessment of student needs that will inform treatment and academic planning. It also serves as a contract between the school system and family outlining the services that the student needs and services the student will be provided. IDEA 2004 states that the IEP should be in place by age 16.
ITP	School System and Family	The ITP is required for all students receiving an IEP. The purpose of the ITP is to prepare the student for the posthigh-school transition and is based on the student's individual needs, skills, and interests.

IDEA 2004 = Individuals with Disabilities Education Act; 504 Plan = Section 504 of the Rehabilitation Act of 1973;

IEP = Individualized Education Program; ITP = Individualized Transition Plan.

*IEP and ITP.* The Individualized Education Program (IEP) is developed once the school has determined that the student meets IDEA 2004 criteria for TBI or other disability. The IEP should include an assessment of the student's present level of academic and cognitive functioning (see below); measurable academic and functional goals; how progress toward goals will be measured; all available services; schedule of the available services, including frequency, duration, and location of services; and modifications for school staff that work with the student (see IDEA, 2004).

Planning and development of individualized goals for the IEP should occur, however, while the child is still hospitalized. Communication among the medical, school system, and family of the child's current functioning and prognosis of functioning should begin long before hospital discharge in order to facilitate a clear communication of resources, goals, and objectives for the child's transition to school (Janus, 1994). It is suggested that this network of medical, school, family, and professionals be informed of the IEP and of any changes to the IEP after the student has started school (Blosser & DePompei, 1993). In addition, as the child is reintegrated to school, her or his network should meet regularly to discuss any changes to the IEP that may reflect either

improvements or new/unaddressed difficulties the student may be experiencing. Some recommend that the team meet at least every 3 to 6 months during the first 2 years post-TBI (Farmer & Peterson, 1995).

As the child comes closer to completing high school, he or she will begin the sometimes difficult transition to adulthood and may need additional support systems (Bergland, 1996). Successful planning and development of the Individualized Transition Plan (ITP) should therefore begin as soon as possible. The ITP is required by law (e.g., Public Law 101–476, IDEA) and should be developed with enough time to facilitate comprehensive planning for vocational, community, or educational goals. The ITP assesses current levels of functioning, employment options, community resources, and vocational evaluation and instructional needs (Grigal, Test, Beattie, & Wood, 1997). Some suggest connections with community services should begin around age 14 and continue through age 16, depending on individual needs and severity (Semrud-Clikeman, 2001). It is also important that the student be involved in transition planning in order to engender student empowerment, determination, student-identified postschool goals, and self-determination (Halpern, 1994). Research has identified 10 characteristics of successful transition planning for students with disabilities: (a) early planning, (b) collaboration and communication between agencies, (c) individualized transition planning, (d) a focus on integration, (e) community-relevant curriculum, (f) community-based training and planning, (g) business linkages, (h) job placement, (i) ongoing staff development, and (j) program evaluation (Rusch & DeStefano, 1989). It is critical that agencies working with students with disabilities attempt to integrate as many of these characteristics as possible when developing the ITP to ensure long-term success of the student after school.

The involvement of the school system, family, medical or psychological community, and individuals working together requires tremendous planning and organization and should begin as soon as possible after TBI. This network should begin planning for the child's return to school before hospital discharge and should continue meeting to discuss and monitor progress during the school years. The IEP should include comprehensive cognitive and academic assessments and should include a detailed account of the services available to the student and plan to help the student reintegrate into school. The ITP should be developed by the network as well as the student and provide linkages to community resources, job opportunities, volunteer opportunities, continued educational planning, and personal goals of the student, prior to finishing school

(Ylvisaker et al., 2007). Now that we have discussed important services, we will discuss important intervention strategies targeted at successful school reintegration.

## Intervention Strategies

Once a comprehensive neuropsychological assessment is conducted and intellectual, behavioral, cognitive, environmental, social, and psychological recommendations are made for the student, interventions are discussed. One of the first goals is to determine how often the student will be at school. For this reason, interventions may need to start off slower and be less demanding at the beginning of recovery. Students with less severe TBI may transition back to school full time, whereas others may attend half-days at school, residential treatment, or home-school. Some suggest that a slow transition to school starting with the student attending half-days and working up to full days with increased recovery is best (Cohen, Joyce, Rhoades, & Welks, 1985; Rosen & Gerring, 1986). This stepwise progression may be easier for the student than overwhelming him or her with the social and academic demands of full-time school.

## The School Environment

School reintegration is often an overwhelming process for caregivers. Families must meet with teachers and other school officials and school psychologists in order to implement a curriculum tailored to the child's needs. Often an IEP is developed at this point. A conjoint consultation is recommended in developing an appropriate treatment plan outlined by Sheridan, Kratochwill, and Bergan (1996; Conoley & Sheridan, 1996). In a conjoint consultation model, parents and school officials are equally invested in identifying and addressing the child's areas of difficulty. This model progresses in four stages: (a) problem identification, (b) problem analysis, (c) treatment implementation, and (d) treatment evaluation. In the first stage, the child's problem areas are identified. These may include academic concerns or behavioral or psychological concerns. In the next stage, an intervention plan is developed in consideration of the information gathered in stage one. The intervention is then implemented. In the fourth and final stage, the treatment is evaluated to determine whether the child is improving in the areas identified in stage one. Changes may need to be made to the treatment plan in order to meet the needs of the child.

Changes to the school environment may also promote academic success for

children with neurocognitive disorders. Braswell and Bloomquist (1991) suggest students may benefit from being seated in the front and center of the classroom in order to decrease distractibility. It is also suggested that eye contact and occasional physical contact from the teacher can help keep the child focused. [Semrud-Clikeman \(2001\)](#) suggests that teachers ask older students to develop vocabulary lists and outline class material that is then shared with younger students. This material allows the child to become familiar with vocabulary and topics of the lesson prior to class. Reviewing vocabulary and having outlines helps children prepare for class, keep up with the pace of the lesson, prepares them to ask questions during the lesson, and listen instead of becoming lost in unfamiliar vocabulary. Categorizing or “chunking” information is also thought to help in retention and recall of information and may be a good strategy for children with TBI. It may also be helpful to teach and test students on parts of a subject, rather than trying to cover too much at once (see Tyler, Blosser, & DePompei, 1999, for a list of strategies to help students organize, follow directions, study, and attend better in the classroom). Children may also benefit from cooperative group-learning activities that involve multiple students and tasks ([Semrud-Clikeman, 2001](#)). Working in groups helps divide up the work and may also facilitate social relationships and interpersonal communication skills.

## Classroom Interventions

Classroom interventions are targeted at reducing negative social and psychological symptoms associated with TBI. Teachers who are educated about the symptoms of TBI and who are aware of common teaching and management strategies for those with disabilities may have more success teaching children with TBI ([Ylvisaker et al., 2005](#)). Continued education and training programs for teachers are imperative for successful and effective teaching, whereas single-day workshops are not ([Sparks, 1983](#)). Schools and support staff should take advantage of any opportunity to participate in effective support programs for children with brain injuries. One program that has been successful is The Kid’s Team in New South Wales Australia. The Kid’s Team, offered by the South West Brain Injury Rehabilitation Service, offers many interdisciplinary programs for children with brain injuries. Services include comprehensive intellectual and neuropsychological assessment, training support and resources for families and school staff (including competency training), case management strategies, sibling support resources, and help with the development of local



support networks.

There are many evidence-based educational strategies that have been found to improve classroom behavior, self-concept, social functioning, and academic performance in children with TBI. Ylvisaker *et al.* (2001) describes many intervention strategies specifically designed for students with TBI, including: (a) metacognitive strategies, which assess organized and strategic learning needs; (b) self-awareness training targeted at validation and understanding of the student's role in education; (c) cognitive-behavior modification, which promotes self-control and behavior regulation; (d) positive antecedent-focused behavior, which focuses on antecedents of behavior problems; and (e) circle of friends, which is a program designed to decrease social isolation and increase interpersonal relationships. Teachers and school staff should seek continued educational opportunities to learn these techniques in order to increase their effectiveness in the classroom.

## Psychological/Behavioral Interventions

Students with TBI may experience diverse behavioral symptoms that make it difficult for them to succeed academically and socially. Disruptions in attention, memory, intelligence, and social or emotional functioning are common in those with TBI (Ewing-Cobbs, Fletcher, & Levin, 1998). Because social and psychological symptoms can profoundly impact school performance, well-validated interventions that are effective for treating social and psychological symptoms may also promote academic success.

Students with TBI may benefit from empirically supported intervention techniques that have been developed to treat specific symptoms or behaviors. CB interventions are a common treatment for patients as well as family members. CB techniques include using variable reinforcement of positive behaviors, modeling appropriate behavior, shaping wanted behaviors, developing behavioral contracts, and role playing to increase self-efficacy. CB interventions often include behavioral questionnaires (e.g., *Behavioral Assessment Scale for Children, 2nd Edition, BASC-2*) that are filled out each month by teachers and caregivers and provide a way to measure progress. Social skills training may also be a useful component of CB interventions for children with social and interpersonal difficulties following injury.

CB interventions that target specific psychological disorders and behaviors (e.g., depression, anxiety, attention deficit hyperactivity disorder) might also be a primary intervention in the school. School-based CB modification was

reported to be an effective treatment for symptoms of aggression and hyperactive–impulsive behaviors in a large meta-analysis (Robinson, Smith, Miller, & Brownell, 1999). The authors also reported symptom reductions after 3 years posttreatment. This suggests that CB interventions, administered in school settings, may have long-term positive effects, even after therapy is discontinued. Thus, CB treatments for psychopathology or behavior that is functionally similar to that of TBI may be a useful intervention approach and should be discussed with teachers, family, and the child’s therapist or counselor.

## Pharmaceutical Interventions

The benefits of medications for cognitive and behavioral functioning in children and adolescents with psychological disorders are well documented. One of the most commonly prescribed stimulants is methylphenidate, which is a dopaminergic and noradrenergic agonist, which has been found to enhance functioning in areas of the frontal lobe and connected striatum (Vaidya et al., 1998). There is a large body of research that documents the efficacy of stimulant medication for symptoms of inattention (and improving sustained attention), vigilance, reaction time, hyperactivity, externalizing behavior, as well as academic achievement (Brown, 1998; Jensen et al., 2001; Rappoport, Denny, DuPaul, & Gardner, 1994; Semrud-Clikeman, Pliszka, & Liotti, 2008; Swanson, Cantwell, Lerner, McBurnett, & Hanna, 1991). Given that children with neurodevelopmental disabilities and TBI also experience many of these symptoms, stimulant medication may also be an effective treatment for them as well.

Recent work has investigated the effects of antidepressant medications on cognitive and mood symptoms in adults with TBI. The antidepressant fluoxetine (i.e., Prozac) was associated with improved working memory performance and fewer symptoms of depression in a small sample of five male outpatients (Horsfield et al., 2002). Others have reported reduction in depressive symptoms in adult patients with depression and TBI using the tricyclic antidepressant desipramine (Wroblewski, Joseph, & Cornblatt, 1996). The findings are limited by small sample sizes, and the effectiveness in treating symptoms of depression in children with TBI has yet to be studied.

## SUMMARY

Children with neurocognitive disabilities following brain trauma often experience profound difficulties reintegrating into the home and school after

injury. Planning for the child's transition and developing a network of school, medical, and psychological professionals before hospital discharge is the best way to ensure social, psychological, and emotional success. Parents, medical, school, and psychological providers should set up meetings as soon as possible to discuss the child's 504 Plan and IEP. The family should be made aware of all possible services for the child based on the 504 Plan and IEP, and discuss goals and objectives and how they will be measured and maintained. Planning for a child's transition out of school should also be discussed years before he or she is ready to leave school. The ITP should include plans for community, volunteer, educational, and employment opportunities.

There are several intervention strategies that have been found to improve academic, psychological, and social impairments in children with neurocognitive disabilities. It is suggested that teachers seek continued education and research on teaching strategies for children with disabilities, rather than single-day workshops that are less effective. In addition, changes to the school environment may also facilitate academic success, including seating the child in the front and center of the classroom, providing the child with lesson outlines before class, and allowing children to work in group-learning experiences (see [Semrud-Clikeman, 2001](#)). CB strategies both in therapy and in the classroom have been found to improve social and psychological behaviors in children with neurocognitive disabilities. These treatments should be implemented as soon as possible after injury and discussed with teachers and educators in order to facilitate translational skills in the classroom. Psychopharmacological treatments may also be helpful for symptoms of depression, anxiety, aggression, and hyperactivity or impulsivity in children with neurocognitive disorders.

Thus, studies and clinical practice support the need for the medical, family, and school teams working together to provide the best support for children who experience brain injuries. The treatment plan not only needs to conform to state and federal guidelines, it also needs to be viewed as a "work in progress." Children with brain injury not only require additional support academically, but also socially. The families of children with brain injury also require support to deal with myriad financial issues, as well as the psychological impact of the injury on the family. Siblings need particular attention as they attempt to work through what has happened to their brother or sister. In addition, support is required for the siblings who may feel pushed aside as the parents deal with all of the requirements for the injured family member.

## REFERENCES

## REFERENCES

- Armengol, C. G. (1999). A multimodal support group with Hispanic traumatic brain injury survivors. *Journal of Head Trauma Rehabilitation, 14*(3), 233–246.
- Batchelor, E. S., & Dean, R. S. (1996). *Pediatric neuropsychology: Interfacing assessment and treatment for rehabilitation*. Boston, MA: Allyn & Bacon.
- Bateman, A., Culpan, F. J., Pickering, A. D., Powell, J. H., Scott, O. M., & Greenwood, R. J. (2001). The effect of aerobic training on rehabilitation outcomes after recent severe brain injury: A randomized controlled evaluation. *Archives of Physical Medicine and Rehabilitation, 82*(2), 174–182.
- Beaulieu, C. L. (2002). Rehabilitation and outcome following pediatric traumatic brain injury. *Surgical Clinics of North America, 82*(2), 393–408.
- Bergland, M. M. (1996). Transition from school to adult life: Key to the future. In A. L. Goldberg (Ed.), *Acquired brain injury in childhood and adolescence* (pp. 171–194). Springfield, IL: Charles C Thomas.
- Blosser, J. L., & DePompei, R. (1993). Professional training and development for pediatric rehabilitation. In C. J. Durgin, N. D. Schmidt, & L. J. Fryer (Eds.), *Staff development and clinical intervention in brain injury rehabilitation* (pp. 229–253). Gaithersburg, MD: Aspen Publications.
- Blosser, J. L., & DePompei, R. (1994). *Pediatric traumatic brain injury: Proactive intervention*. San Diego, CA: Singular.
- Braga, L. W., Da Paz, A. C., & Ylvisaker, M. (2005). Direct clinician-delivered versus indirect family-supported rehabilitation of children with traumatic brain injury: A randomized controlled trial. *Brain Injury, 19*(10), 819–831.
- Brown, R. T. (1998). Short-term cognitive and behavioral effects of psychotropic medications. In R. T. Brown & M. Sawyer (Eds.), *Medications for school age children: Effects on Learning and Behavior* (pp. 29–61). New York, NY: Guilford Press.
- Brown, S. A., McCauley, S. R., Levin, H. S., Contant, C., & Boake, C. (2004). Perception of health and quality of life in minorities after mild-to-moderate traumatic brain injury. *Applied Neuropsychology, 11*(1), 54–64.
- Butler, R. W., Copeland, D. R., Fairclough, D. L., Mulhern, R. K., Katz, E. R., Kazak, A. E., . . . Sahler, O. J. (2008). A multicenter, randomized clinical trial of a cognitive remediation program for childhood survivors of a pediatric malignancy. *Journal of Consulting and Clinical Psychology, 76*(3), 367–378.
- Butler, R. W., & Haser, J. K. (2006). Neurocognitive effects of treatment for childhood cancer. *Mental Retardation and Developmental Disabilities Research Reviews, 12*(3), 184–191.
- Cohen, S. B., Joyce, C. M., Rhoades, K. W., & Welks, D. M. (1985). Educational programming for head injured students. In M. Ylvisaker (Ed.), *Head injury rehabilitation children and adolescents* (pp. 383–409). San Diego, CA: College-Hill Press.
- Conoley, J. C., & Sheridan, S. M. (1996). Pediatric traumatic brain injury: Challenges and interventions for families. *Journal of Learning Disabilities, 29*(6), 662–669.
- D'Amato, R. C., & Rothlisberg, B. A. (1996). How education should respond to students with traumatic brain injury. *Journal of Learning Disabilities, 29*(6), 670–683.
- DeChillo, N., Koren, P. E., & Schultze, K. H. (1994). From paternalism to partnership: Family and professional collaboration in children's mental health. *American Journal of Orthopsychiatry, 64*(4), 564–576.
- Dyson, L., Edgar, E., & Crnic, K. (1989). Psychological predictors of adjustment by siblings of developmentally disabled children. *American Journal of Mental Retardation, 94*(3), 292–302.
- Echemendia, R. J. (2004). Cultural diversity and neuropsychology: An uneasy relationship in a time of change. *Applied Neuropsychology, 11*(1), 1–3.
- Echemendia, R. J., & Westerveld, M. (2006). Cultural perspectives in pediatric rehabilitation. In J. E. Farmer, J. Donders, & S. Warschusky (Eds.), *Treating neurodevelopmental disabilities* (pp. 289–307). New York, NY: Guilford Press.
- Ewing-Cobbs, L., Fletcher, J. M., & Levin, H. S. (1986). Neurobehavioral sequelae following head injury in children: Educational implications. *Journal of Head Trauma Rehabilitation, 1*, 57–65.

- Farmer, J. E., & Drewel, E. H. (2006). Systems interventions for comprehensive care. In J. E. Farmer, J. Donders, & S. Warschusky (Eds.), *Treating neurodevelopmental Disabilities* (pp. 269–288). New York, NY: Guilford Press.
- Farmer, J. E., & Muhlenbruck, L. (2001). Telehealth for children with special health care needs: Promoting comprehensive systems of care. *Clinical Pediatrics*, *40*(2), 93–98.
- Farmer, J. E., & Peterson, L. (1995). Pediatric traumatic brain injury: Promoting successful school reentry. *School Psychology Review*, *24*(2), 230–243.
- Farmer, J. E., Marien, W. E., Clark, M. J., Sherman, A., & Selva, T. J. (2004). Primary care supports for children with chronic health conditions: Identifying and predicting unmet family needs. *Journal of Pediatric Psychology*, *29*(5), 355–367.
- Feeney, T. J., Ylvisaker, M., Rosen, B. H., & Greene, P. (2001). Community supports for individuals with challenging behavior after brain injury: An analysis of the New York state behavioral resource project. *The Journal of Head Trauma Rehabilitation*, *16*(1), 61–75.
- Flett, P. J., & Stoffell, B. F. (2003). Ethical issues in paediatric rehabilitation. *Journal of Paediatrics and Child Health*, *39*(3), 219–223.
- Glang, A., Tyler, J., Pearson, S., Todis, B., & Morvant, M. (2004). Improving educational services for students with TBI through statewide consulting teams. *Neuro Rehabilitation*, *19*(3), 219–231.
- Halpern, A. S. (1994). The transition of youth with disabilities to adult life: A position statement of the division on career development and transition, the council of exceptional children. *Career Development for Exceptional Individuals*, *17*(2), 115–124.
- Hawley, C. A., Ward, A. B., Magnay, A. R., & Long, J. (2003). Parental stress and burden following traumatic brain injury amongst children and adolescents. *Brain Injury*, *17*(1), 1–23.
- Horsfield, S. A., Rosse, R. B., Tomasino, V., Schwartz, B. L., Mastropaolo, J., & Deutsch, S. I. (2002). Fluoxetine's effects on cognitive performance in patients with traumatic brain injury. *International Journal of Psychiatry in Medicine*, *32*(4), 337–344.
- Janus, P. L. (1994). The role of school administration. In R. C. Savage & G. F. Wolcott (Eds.), *Educational dimensions of acquired brain injury* (pp. 345–365). Austin, TX: Pro-Ed.
- Jensen, P. S., Hinshaw, S. P., Swanson, J. M., Greenhill, L. L., Conners, C. K., Arnold, L. E., . . . Wigal, T. (2001). Findings from the NIMH Multimodal Treatment Study of ADHD (MTA): Implications and applications for primary care providers. *Journal of Developmental and Behavioral Pediatrics*, *22*(1), 60–73.
- Katsiyannis, A., & Conderman, G. (1994). Serving individuals with traumatic brain injury. *Remedial and Special Education*, *15*, 319–325.
- Kazak, A. E., Cant, C., Jenson, M. M., McSherry, M., Rourke, M. T., Hwang, W., . . . Lange, B. J. (2003). Identifying psychosocial risk indicative of subsequent resource use in families of newly diagnosed pediatric oncology patients. *Journal of Clinical Oncology*, *21*, 3220–3225.
- Kelly, A., Golnik, A., & Cady, R. (2008). A medical home center: Specializing in the care of children with special health care needs of high intensity. *Maternal and Child Health Journal*, *12*(5), 633–640.
- King, G., King, S., Rosenbaum, P., & Goffin, R. (1999). Family-centered caregiving and well-being of parents of children with disabilities: Linking process with outcome. *Journal of Pediatric Psychology*, *24*, 41–53.
- King, G., Cathers, T., King, S., & Rosenbaum, P. (2001). Major elements of parents' satisfaction and dissatisfaction with pediatric rehabilitation services. *Children's Health Care*, *30*, 11–134.
- King, S., Teplicky, R., King, G., & Rosenbaum, P. (2004). Family-centered service for children with cerebral palsy and their families: A review of the literature. *Seminars in Pediatric Neurology*, *11*(1), 78–86.
- Martin, A. B., Crawford, S., Probst, J. C., Smith, G., Saunders, R. P., Watkins, K. W., & Luchok, K. (2007). Medical homes for children with special health care needs: A program evaluation. *Journal of Health Care for the Poor and Underserved*, *18*(4), 916–930.
- McMenamy, J. M., & Perrin, E. C. (2004). Integrating psychology into pediatrics: The past, the present, and

- the potential. *Family Systems and Health*, 20, 153–160.
- Molnar, G. E. (1985). *Pediatric rehabilitation*. Baltimore, MD: Williams & Wilkins.
- Naar-King, S., & Donders, J. (2006). Pediatric family-centered rehabilitation. In J. E. Farmer, J., Donders, & S. Warchausky (Eds.), *Treating neurodevelopmental disabilities* (pp. 149–169). New York, NY: Guilford Press.
- Naar-King, S., Siegel, P. T., & Smyth, M. (2002). Consumer satisfaction with a collaborative, interdisciplinary health care program for children with special needs. *Children's Services: Social Policy, Research, and Practice*, 5, 189–200.
- Naar-King, S., Siegel, P. T., Smyth, M., & Simpson, P. (2003). An evaluation of an integrated health care program for children with special needs. *Children's Health Care*, 32, 233–243.
- Neglia, J. P., Robison, L. L., Stovall, M., Liu, Y., Packer, R. J., Hammond, S., . . . Inskip, P. D. (2006). New primary neoplasms of the central nervous system in survivors of childhood cancer: A report from the Childhood Cancer Survivor Study. *Journal of the National Cancer Institute*, 98(21), 1528–1537.
- Newacheck, P. W., Inkelas, M., & Kim, S. E. (2004). Health services use and health care expenditures for children with disabilities. *Pediatrics*, 114(1), 79–85.
- Orsillo, S. M., McCaffrey, R. J., & Fisher, J. M. (1993). Siblings of head-injured individuals: A population at risk. *Journal of Head Trauma Rehabilitation*, 8, 102–115.
- Osberg, J. S., Kahn, P., Rowe, K., & Brooke, M. M. (1996). Pediatric trauma: Impact on work and family finances. *Pediatrics*, 98(5), 890–897.
- Park, N. W., & Ingles, J. L. (2001). Effectiveness of attention rehabilitation after an acquired brain injury: A meta-analysis. *Neuropsychology*, 15(2), 199–210.
- Robinson, R. T., Smith, S. W., Miller, D., & Brownell, M. T. (1999). Cognitive behavior modification of hyperactivity-impulsivity and aggression: A meta-analysis of school-based studies. *Journal of Education Psychology*, 91(2), 195–203.
- Rosen, C. D., & Gerring, J. P. (1986). *Head trauma: Educational reintegration*. San Diego, CA: College-Hill Press.
- Rosenbaum, P., King, S., Law, M., King, G., & Evans, J. (1998). Family-centered service: A conceptual framework and research review. *Physical & Occupational Therapy in Pediatrics*, 18, 1–20.
- Rosenberg, D., Onufer, C., Clark, G., Wilkin, T., Rankin, K., & Gupta, K. (2005). The need for care coordination among children with special health care needs in Illinois. *Maternal and Child Health Journal*, 9(Suppl. 2), S41–S47.
- Rusch, F. R., & DeStefano, L. (1989). Transition from school to work: Strategies for young adults with disabilities. *Interchange*, 9, 1–2.
- Russman, B. S. (1990). Rehabilitation of the pediatric patient with a neuromuscular disease. *Neurologic Clinics*, 8(3), 727–740.
- Seid, M., Sobo, E. J., Gelhard, L. R., & Varni, J. W. (2004). Parents' reports of barriers to care for children with special health care needs: Development and validation of the barriers to care questionnaire. *Ambulatory Pediatrics*, 4(4), 323–331.
- Semrud-Clikeman, M. (2001). *Traumatic brain injury in children and adolescents*. New York, NY: Guilford Press.
- Semrud-Clikeman, M., Pliszka, S., & Liotti, M. (2008). Executive functioning in children with attention-deficit/hyperactivity disorder: Combined type with and without a stimulant medication history. *Neuropsychology*, 22(3), 329–340.
- Smith, K., Layne, M., & Garell, D. (1994). The impact of care coordination on children with special health care needs. *Children's Health Care: Journal of the Association for the Care of Children's Health*, 23(4), 251–266.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach* (2nd ed.). New York, NY: Guilford Press.
- Sparks, G. M. (1983). Synthesis of research of staff development for effective teaching. *Educational Leadership*, 41(3), 65–72.

- Sperry, L. A., Whaley, K. T., Shaw, E., & Brame, K. (1999). Services for young children with autism spectrum disorders: Voices of parents and providers. *Infants and Young Children, 11*, 17–33.
- Swaiman, K. F., Ashwal, S., & Ferriero, D. M. (2006). *Pediatric neurology* (4th ed.). San Diego, CA: Mosby.
- Swanson, J. M., Cantwell, D., Lerner, M., McBurnett, K., & Hanna, G. (1991). Effects of stimulant medication on learning in children with ADHD. *Journal of Learning Disabilities, 24*(4), 219–230.
- Tyler, J., Blosser, J., & DePompei, R. (1999). *Teaching strategies for students with brain injuries*. Wake Forest, NC: Lash and Associates Publishing/Training Inc.
- United States Department of Education, Office of Special Education Programs. (2006). *Implementation of the individuals with disabilities education Act: 28th Annual report to Congress*. Washington, DC: US Department of Education, Office of Special Education Programs.
- Vaidya, C. J., Austin, G., Kirkorian, G., Ridlehuber, H. W., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. (1998). Selective effects of methylphenidate in attention deficit hyperactivity disorder: A functional magnetic resonance study. *Proceedings of the National Academy of Sciences of the United States of America, 95*(24), 14494–14499.
- Wade, S. L., Borawski, E. A., Taylor, H. G., Drotar, D., Yeates, K. O., & Stancin, T. (2001). The relationship of caregiver coping to family outcomes during the initial year following pediatric traumatic injury. *Journal of Consulting and Clinical Psychology, 69*(3), 406–415.
- Wade, S. L., Taylor, H. G., Drotar, D., Stancin, T., & Yeates, K. O. (1996). Childhood traumatic brain injury: Initial impact on the family. *Journal of Learning Disabilities, 29*(6), 652–661.
- Walker, N. W., Boling, M. S., & Cobb, H. (1999). Training of school psychologists in neuropsychology and brain injury: Results of a nationally survey of training programs. *Child Neuropsychologist, 5*, 137–142.
- Wroblewski, B. A., Joseph, A. B., & Cornblatt, R. R. (1996). Antidepressant pharmacotherapy and the treatment of depression in patients with severe traumatic brain injury: A controlled, prospective study. *Journal of Clinical Psychiatry, 57*(12), 582–587.
- Yeates, K. O., Taylor, H. G., Woodrome, S. E., Wade, S. L., Stancin, T., & Drotar, D. (2002). Race as a moderator of parent and family outcomes following pediatric traumatic brain injury. *Journal of Pediatric Psychology, 27*(4), 393–403.
- Ylvisaker, M., Adelson, P. D., Braga, L. W., Burnett, S. M., Glang, A., Feeney, T., Todis, B. (2005). Rehabilitation and ongoing support after pediatric TBI: Twenty years of progress. *Journal of Head Trauma Rehabilitation, 20*(1), 95–109.
- Ylvisaker, M., Turkstra, L., Coelho, C. A., Yorkston, K., Kennedy, M., Sohlberg, M. M., & Avery, J. (2007). Behavioural interventions for children and adults with behaviour disorders after TBI: A systematic review of the evidence. *Brain Injury, 21*, 769–805.

## Practitioner Traits in Neurorehabilitation

*Pamela S. Klonoff*

Patients with brain injuries often rely on their psychotherapists to help them with their long, painstaking climb toward rebuilding their lives. The fruitfulness of psychotherapy after brain injury is dependent on the collaborative relationship between the patient and psychotherapist (Klonoff, 2010). For the process to unfold and flourish, the psychotherapist must not only encompass a deep understanding of brain–behavior relationships, but also embody perceptiveness and empathy. The psychotherapist must also be invested in reflective listening, and together with the patient undertake the worthy search for self-discovery and personal growth (see Klonoff, 2010, for a review). The psychotherapist’s helpfulness is also based on his or her ability to create a safe and hospitable environment, where the patient can think creatively and freely express his or her emotions, ideas, and needs.

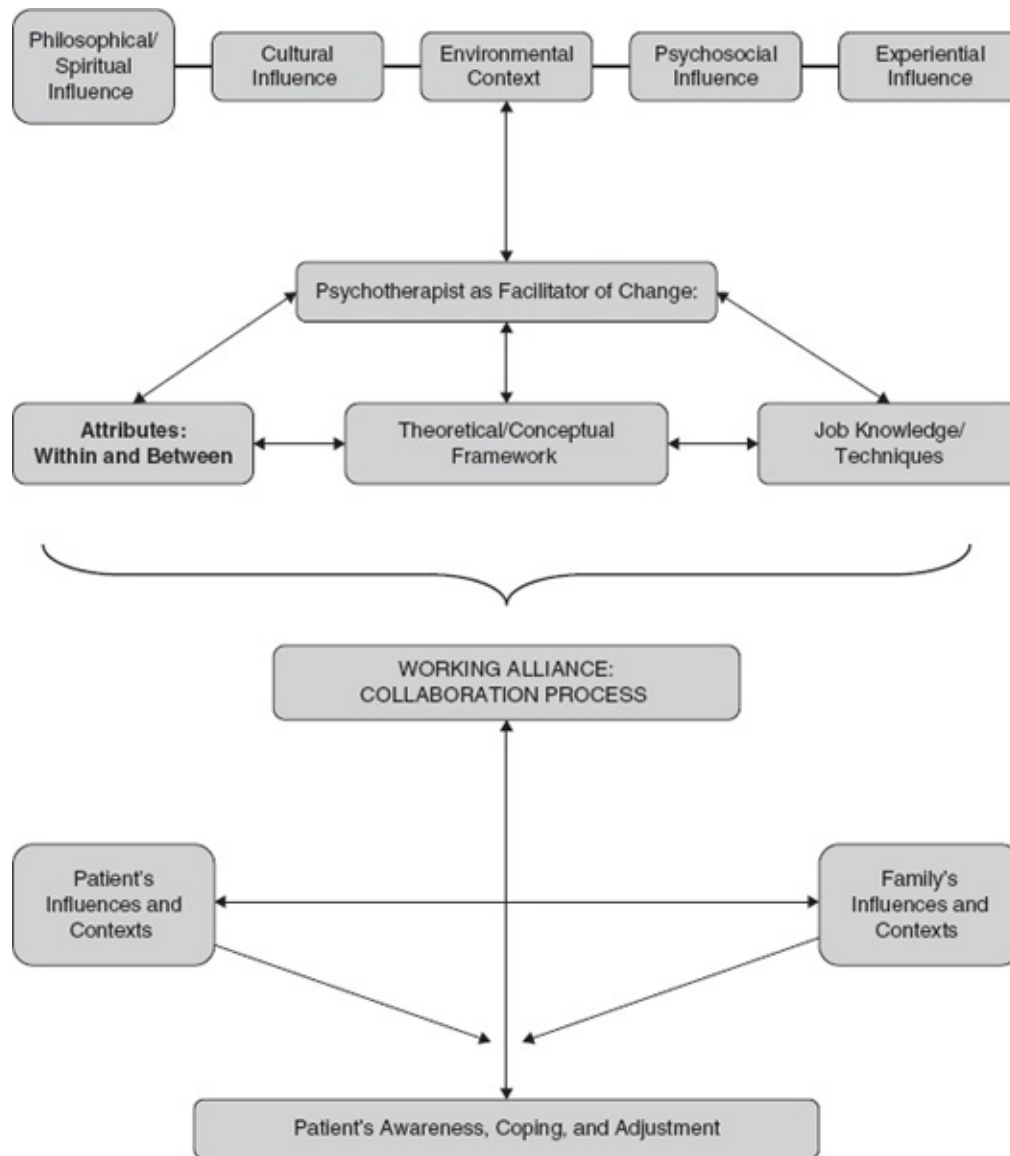
Historically, there is a dearth of psychotherapy models, which examine the optimal qualities of the psychotherapist who treats patients with brain injuries. This includes not only the psychotherapist’s personal influences, attributes, and job knowledge, but just as importantly, his or her specific capabilities to function as part of a treatment team. This chapter will briefly overview an integrative multifactorial model for psychotherapists treating patients with brain injuries and then discusses in detail the psychotherapist’s internal attributes, which guide and articulate the psychotherapeutic process. Of note, the personality characteristics described are considered all-purpose and pertinent to treating patients with varied brain injury etiologies, chronicities, severities, and ages, including their developmental status. In addition, the internal qualities that enhance the



psychotherapist's success are germane in any setting: private practice, clinic, hospital-based practice where the clinician liaisons with other specialists, or as part of an outpatient or holistic milieu treatment setting.

### **MODEL DESCRIPTION**

[Figure 15.1](#) depicts an integrated multifactorial model that incorporates both the patient and psychotherapist engaged in the collaborative approach to change. The psychological transformation process depends on a multiplicity of factors, which are both internal and external to the therapist and the patient. The patient's personal growth depends on two components: (a) the psychotherapist's ability to act as a meaningful facilitator of change and (b) the patient's willingness and capability to improve his or her awareness, coping skills, and adjustment. Within the context of recovery after a significant brain injury, the patient also requires investment from an external support system, for example, the family. The conduit for patients' growth and adjustment within this collaborative process is the working alliance ([Klonoff, 2010](#)).



**FIGURE 15.1** Collaborative model of psychotherapy after brain injury (adapted from [Klonoff, 2010](#)).

Components of this model have been described elsewhere, including theoretical conceptual frameworks, elements of the working alliance, and aspects of job knowledge (e.g., “tools of the trade;” [Klonoff, 2010](#)). This chapter will focus on a heuristic exploration of the ideal characteristics and influences, which shape the psychotherapist providing neurorehabilitation treatment on an individual basis and in a team setting.

### The Psychotherapist

The qualities, skills, and life experiences of the psychotherapist are fundamental

to the course and success of the psychotherapeutic process after brain injury. Contributing factors include external influences, the psychotherapist's internal attributes (e.g., *within* characteristics), and attributes that facilitate synchrony with other treating therapists (*between* characteristics).

### External Influences

There are a number of external influences that shape and articulate the psychotherapist's persona. These include his or her broader philosophical, spiritual, and cultural underpinnings (Aponte, 1996; Hoshmand, 2006). These interact and amalgamate with the psychotherapist's specific psychosocial background, environmental context, and personal life experiences (within and outside of educational experiences; Reupert, 2008). When integrated, they manifest as a life perspective, embedded in a worldview (Farber, Manevich, Metzger, & Saypol, 2005). Ultimately, the psychotherapist's demographics (e.g., gender and sexual orientation), values, morality, political orientations, ideology, race, ethnicity, social class, cultural beliefs, role models, and traditions undoubtedly mold the psychotherapist's identity, attitudes, behaviors, assumptions, communication style, unintentional biases, and even possibly personal prejudices toward patients (Aponte, 1996; Comas-Díaz, 2005; Davar, 2008; Farber et al., 2005; Haldeman, 2010; Loewenthal, 2006; Reupert, 2008; Vasquez, 2007; White, 2007). Also, some authors have suggested that the experience of personal loss, illness or disability, suffering, and psychic pain predisposes the psychotherapist to become a "wounded healer" (Comas-Díaz, 2005; Farber et al., 2005; Jung, 1951). Sometimes, psychologists choose to specialize in the field of neuropsychology, related to their own personal experience with an individual who suffered a brain injury, whether he or she is a friend or relative.

All of these factors have particular precursor influences on the psychotherapist's viewpoints of the meaning and implications of brain injury. These influences then define the psychotherapist's treatment population preferences, the nature and style of therapeutic interventions, and countertransference reactions. Recognition of these influences promotes mutual understanding and tolerance of the complex and sometimes vexing injury-related challenges.

### *Within* Attributes of the Psychotherapist

Table 15.1 depicts the *within* component of the model, which focuses on the

integral, internal attributes *within* the treating psychotherapist. This list is not intended to be exhaustive; instead, it serves to represent our personal quest for internal attributes, standards, and virtues, not only as psychotherapists, but also as human beings. The process also mirrors the travels of our patients, as at times their acquisition of life principles and goals postinjury seem disconcerting, elusive, or overly lofty. The *within* component has been subdivided into five categories to assist in its conceptualization: personality traits, cognitive capabilities, narcissistic/ego structures, relational behaviors, and existential qualities.

### Personality Traits

The personality traits of the psychotherapist are instrumental in providing the “secure base” in the therapeutic environment (Bowlby, 1988) that patients need to undertake the perilous journey of accepting and adapting to the challenges faced after brain injury. The subset of traits elucidates the primacy of the psychotherapist’s personality and demeanor in facilitating and guiding this process; they can be considered the “therapeutic disposition” (Knobel, 1990). In this author’s clinical experience, regardless of the severity—even severe brain injuries—or chronicity of the damage, patients are adept at “reading” the personality style of the psychotherapist and responding to the presence or absence of these internal traits.

Central to developing the working alliance is the psychotherapist’s intuition. This includes his or her vigilance, receptivity, and attunement (Bove & Rizzi, 2009) manifested as the experience of hunches, sudden insights, and uncanny feelings, which shape the therapeutic process (Welling, 2005). Given the limitations patients often experience in their ability to access and describe emotions after brain injury, the psychotherapist’s intuition becomes a salient compass for therapeutic dialogue.

The therapeutic connection is contingent on the therapist’s humaneness manifested by compassion and respect toward patients and their plights (Dell Orto & Power, 2000). This quality of humaneness tangibly communicates feelings of value and security to the patients after brain injury, as they recognize their emotional needs will be addressed in a kindhearted manner in the midst of a sudden and frightening reality.

**TABLE 15.1** *Within* Attributes of the Psychotherapist

---

<p><i>Personality Traits</i></p> <ul style="list-style-type: none"> <li>intuitive</li> <li>humane</li> <li>has integrity</li> <li>humble</li> <li>sincere</li> <li>authentic</li> <li>dedicated/advocate</li> <li>dependable</li> <li>enthusiastic</li> <li>affable</li> <li>sense of humor</li> </ul>	<p><i>Relational Behaviors</i></p> <ul style="list-style-type: none"> <li>warm/kind</li> <li>patience</li> <li>courteous</li> <li>empathic</li> <li>soothing</li> <li>validating</li> <li>trusting</li> <li>attuned</li> <li>healthy self-disclosure</li> <li>"productive worry"</li> <li>nonjudgmental</li> </ul>
<p><i>Cognitive Capabilities</i></p> <ul style="list-style-type: none"> <li>executive functions</li> <li>metacognitive skills</li> <li>efficient</li> <li>curious</li> <li>capacity to learn and acquire wisdom</li> </ul>	<p><i>Existential Qualities</i></p> <ul style="list-style-type: none"> <li>individuality</li> <li>resolve</li> <li>self-actualizing</li> <li>process-oriented</li> <li>creative</li> </ul>
<p><i>Narcissistic/Ego Structures</i></p> <ul style="list-style-type: none"> <li>introspection and self-scrutiny</li> <li>tolerance for painful and negative emotions</li> <li>tolerance for imperfections resiliency</li> <li>resiliency</li> <li>a cohesive self</li> <li>self-respect/self-worth</li> <li>self-preservation</li> <li>adaptability</li> </ul>	<ul style="list-style-type: none"> <li>spiritual</li> <li>make sense of suffering</li> <li>has vision/strives for meaning in life</li> <li>altruistic</li> <li>hopeful</li> </ul>

Other optimal personality traits of the psychotherapist are his or her level of integrity, humility, honesty, sincerity, and authenticity (Gross, 2001; Kitron, 2007; Newman, 2002; Strupp, 1973). Integrity is the foundation of acting in the best interest of the patient (Gross, 2001). This is paramount as the individual often feels helpless and vulnerable and is so reliant on the therapist for guidance. In addition, as part of dedication to patients' needs, "taking a stand" and demonstrating patient advocacy emboldens patients in their own often daunting undertakings at community reintegration.

Humility, characterized by the psychotherapist acknowledging his or her ineptitudes and shortcomings, invigorates the patient in his or her quest to integrate postinjury limitations. Patients need to sense the core inner strength and stability in the psychotherapist juxtaposed with his or her personal feelings of humility in order to submit themselves to sharing their innermost feelings of fragility and disillusionment. The psychotherapist's humility enables him or her to reflect internally, question approaches, and formulate new and better

hypotheses (Newman, 2002) to maximize the patient's progress and recovery. The psychotherapist also needs to display modesty about his or her skill set, also based on recognizing and admitting the limits of one's knowledge, for example, "knowing what you know." This ownership provides the foundation for the patient to emulate and confront his or her grandiose fantasies and develop a realistic sense of self, albeit modified after the injury.

Intrinsic to effective psychotherapy is the patient's perception of the psychotherapist as sincere and authentic. This includes demonstrating "personal modeling" (Klonoff, 1997; Yalom, 2002) as the patient senses whether the psychotherapist embodies the very characteristics that he or she is encouraging and developing within the patient. In our treatment milieu, patients are held highly accountable for their rehabilitation endeavors, (e.g., punctuality and completing assignments). Likewise, the psychotherapist must be fully committed and genuinely embrace his or her responsibilities and fulfill "assignments," (e.g., follow up on promised phone calls and complete paperwork in a timely fashion). This dependability avoids "double standards" and feelings of hypocrisy in the patients and their support systems.

The psychotherapist's enthusiasm or "perkiness," and passion about his or her vocational pursuits imbue patients with the impression that they matter and are likeable; this offsets their dour self-image. Affability in the psychotherapist is contagious to patients, especially as they struggle with devastating postinjury losses. Positive emotion, a sense of humor, and laughter aid in coping with adversity and reduce distress levels and feelings of inferiority, as well as give patients the courage to be imperfect (see Scholl & Ragan, 2003 for a review). Joking and friendly humor are powerful healing tools after brain injury; they promote an atmosphere of joy, breed bonding, and create lightheartedness when so much of the patient's preoccupations center on dejection and grief.

## Cognitive Capabilities

The second category of *within* characteristics relates to the psychotherapist's cognitive abilities. The psychotherapist represents an "auxiliary ego" or "alter ego," and utilizes superior executive functions to ameliorate patients' struggles with dysexecutive symptoms, specifically, abstract reasoning, preplanning, problem solving, flexible and "big picture" thinking, as well as decision making (Freed, 2002; Klonoff, 1997, 2010; Klonoff & Lage, 1991). The psychotherapist's "frontal lobe" capabilities are used to retrain patients' fundamental executive system skills to complete concrete daily activities (e.g.,

ordering medications, making doctors' appointments, and registering for school). In addition, the psychotherapist's metacognitive skills in the areas of analysis, prioritization, "case management," and task execution are imperative to create the necessary structure and momentum for their patients' postinjury goal attainment. Often, the psychotherapist helps to "blaze a trail" as patients grapple to identify with realistic objectives (e.g., living situation, driving, school, and work; [Klonoff, 2010](#)). Assisting patients to realize their visions for the future in an efficient manner also requires strong skills in common sense, conceptualization, and organization in the psychotherapist ([Klonoff, 1997](#)).

Remaining curious about the interrelationships between patients' neurological and psychological recovery elucidates the direction of therapeutic interventions. An important byproduct of the collaborative process is that the therapist can also learn and grow personally and professionally from the patient, as long as he or she is open to interactions and experiences ([Rosenblatt, 2009](#)).

### Narcissistic/Ego Structures

Patients with brain injuries struggle with heightened states of anxiety and emotional turmoil ([Gunther, 1971](#); [Lewis & Rosenberg, 1990](#)) and are thrust into the process of grieving the loss of their former existence and identity ([Klonoff, 2010](#); [Lewis & Rosenberg, 1990](#)). Guiding patients into the perilous emotional territory of exploration and coping with intense feelings and an altered state of being is often foreshadowed by the psychotherapist's aptitude and motivation to self-reflect. The psychotherapist best models this process of introspection and self-scrutiny by being attuned to and watchful of his or her limitations, vulnerabilities, inner disappointments, and imperfections ([Bettelheim, 1975](#); [Klonoff, 2011](#); [Lewis, 1999](#); [Yalom, 2002](#)).

The hearty psychotherapist must be able to tolerate personal feelings of isolation, anxiety, and frustration, which are inevitable in their work ([Klonoff, 2011](#); [Yalom, 2002](#)). To the extent the psychotherapist can experience, endure, and even embrace his or her own feelings of emotional pain, sorrow, and loss, he or she is better prepared to address and cope with the often intensified emotionality in his or her patients.

The psychotherapist's tolerance for mistakes and imperfections mirrors the same (but exaggerated) process patients experience after brain injury; this normalizes their feelings ([Klonoff, 2011](#)). For example, the psychotherapist is bound to make human errors (e.g., occasionally forgets a patient promise) and therefore must be able to acknowledge these foibles. Similarly, the

psychotherapist's internal comfort with his or her shameful experiences, feelings of helplessness, and personal insecurities feeds the reservoir of empathic responsiveness and attunement with the patient's similar emotional experiences postinjury (Gunther, 1977; Yalom, 2002). For example, the psychotherapist must at times confront and cope with realizations about the limits of his or her neurorehabilitation interventions and then accept and cope with clinical shortfalls and setbacks (Klonoff, 2011).

The intensity and pace of the neurorehabilitation process is often fraught with pressures, emotional crises, as well as medical challenges and complications coming "fast and furious." The psychotherapist's resiliency and hardiness allows him or her to adapt and rebound from stressful situations (Dell Orto & Power, 2000). Psychotherapists will also encounter many negative emotions displayed by the patients through their transference and resistance responses. Therefore, the psychotherapist requires the attributes of personal cohesiveness and resiliency to "ride the emotional storm" of the therapeutic process.

The psychotherapist's internal sense of self-respect and self-worth inculcated by healthy techniques of self-preservation should transmit similar behaviors and beliefs to his or her patients. That is, via transmuted internalization, the patient is undergoing a reciprocal process of developing attributes of the self, including self-initiative and cohesion (Kohut, 1984). Ultimately, it is the psychotherapist's commitment to his or her own process of lifelong self-investment and adaptability that replicates the parallel process for his or her patients.

## Relational Behaviors

Relational characteristics encompass the psychotherapist's interpersonal abilities for relating to and communicating properly with patients. Warmth, kindness, patience, and courtesy are the foundation for a bona fide working alliance with the patient and family (Ackerman & Hilsenroth, 2003; Block, 1987; Leber & Jenkins, 1996; Pinkerton, 2008). Conversely, a distanced, disconnected, or indifferent treatment style negatively impacts the working relationship (Hersoug, Høglend, Havik, von der Lippe, & Monsen, 2009).

Pivotal to every psychotherapeutic relationship, including after brain injury, is empathy. Empathy means the psychotherapist is able to relate to the patient with "deeply reverberating understanding and resonant emotionality" (Kohut, 1984, p. 82). This allows the psychotherapist to relate to the plight of the



patients and imagine their struggles and worries in an observant and tolerant way (Bettelheim, 1975; Newman, 2002). Bettelheim (1975) describes a process whereby the therapist must “join the patient where he dwells emotionally” (p. 277) and patiently construct a ladder built from the psychotherapist’s past, knowledge, understanding, and empathy. Gans (1983) suggests that the empathic stance helps the psychotherapist recognize the underlying feelings of powerlessness and hopelessness in patients after brain injury who may display external manifestations of hatred and resentment toward their psychotherapists. Gunther (1971) suggests that the empathic acceptance by rehabilitation staff and the admiration of patients’ efforts sustains their hopes during the crises caused by their injuries. Thus, empathic responsiveness creates the relational climate necessary for connectedness and active collaboration within the therapeutic process and reconstitution of the patient’s identity (Flaskas, 2009; Leber & Jenkins, 1996; Lewis & Rosenberg, 1990).

An outcropping of empathy is the psychotherapist’s knack for providing a soothing or pacifying function for the patient (Basch, 1980), as well as validating his or her psychic pain (Newman, 2002). This becomes critical when patients sustaining brain injuries collapse emotionally due to catastrophic reactions (Klonoff, Lage, & Chiapello, 1993) or disintegration of their sense of self (Klonoff & Myles, 2010).

Precursors to empathy are the abilities to attach to and trust others, reciprocity, and mutuality (Hojat, 2007). In addition, through emotional engagement, attunement, and shared understanding, the therapist imagines himself or herself in relation to the patient (Flaskas, 2009). This has also been referred to as mindfulness, as the therapist is receptive and has a presence within the therapeutic space (Siegel, 2010).

Therapists in our holistic treatment setting are emotionally expressive; this includes tears of sadness or joy, exhilaration, consternation, and (controlled) frustration. The psychotherapist’s self-disclosures (with careful application in the therapy process) also encourage the patient to communicate and share openly. Personal vignettes should be chosen only if helpful to the patient’s needs and ambitions, so as to preserve healthy boundaries.

The tendency on the part of the psychotherapist to “worry productively” reflects his or her personal commitment and investment in the psychological well-being of the patient and the strength not to “turn a blind eye” (Lage, personal communication) in the face of dangerous or unhealthy situations the patient may gravitate toward. For example, sometimes patients return to

substance abuse in the latter phases of their recovery. Carefully monitoring these relapses using random urinalyses and substance abuse counseling communicates a sense of earnest unease, given the risk of harmful medical complications (Klonoff, 2010).

The search for awareness, acceptance, and realism prompts patients after brain injury to meander psychologically, sometimes becoming side-tracked by pursuing unattainable objectives. Nonetheless, truly listening to the patient without imposing prejudgments, intolerance, and personal biases creates an ambience of unconditional support for the patients' thoughts and feelings (Hojat, 2007), thereby providing the external structure and emotional sustenance for patients to recalibrate their aspirations.

### Existential Qualities

The psychotherapist's existential being relates to what is self-defining and transcendent. This translates to the psychotherapist's individuality, resolve, and ultimately the pursuit of self-actualization. Like patients with acquired brain injuries, the psychotherapist is focusing on improving self-awareness and self-development (Maslow, 1971). This evolving life perspective also matches the patients' ultimate transition to finding quality of life and meaning, often through exploration of postinjury key life values (Klonoff, 2010). Like the patient's recovery process, it behooves the therapist to focus on enjoying the journey of his or her toils, rather than being preoccupied with rapid transit to the usually elusive destination. The psychotherapist's composure allows his or her therapeutic endeavors and destiny to unfold, rather than trying to orchestrate and coerce the outcome (Klonoff, 2011), which only serves to frustrate his or her patients. In this way, the psychotherapist must be comfortable with ambiguity and/or paradox (Bien, 2004). This self-view also encourages patients to adapt to many postinjury uncertainties, also with permission to heal gradually rather than with undue self-pressure and self-criticism.

The psychotherapist's creativity enriches the psychotherapeutic process and invites the patient to immerse himself or herself in a similar process of self-discovery. As the psychotherapist quests for attainable goals in functional, spiritual, and existential domains, he or she sets a foundation for a personally and professionally fulfilled existence. The psychotherapist's appreciation of these higher order ideals sets a context for inspiring patients to set their postinjury sights for genuine personal and collective purposes.

The psychotherapist in the neurorehabilitation setting will be surrounded

by sickness, loss, and sometimes death. Assisting patients in transforming their losses and life tragedies into a new existence is predicated in the belief that one can make sense of and even transcend suffering. This fosters the clinician's altruism and personal quest for new hope, vision, and life meaning both in himself or herself and in the patients (Klonoff, 2010).

### ***Between Attributes of the Treatment Team***

When the psychotherapist liaisons with his or her colleagues as part of an interdisciplinary team and unifies treatment efforts, the patient's therapeutic gains are maximized (Klonoff, 2011). Crucial to the success of traditional outpatient therapy settings and holistic milieu programs are the treatment team's composition and dynamics. Staff members strive to effect therapeutic change by promoting a healing ambience for patients. The therapists need to be able to utilize their specialized skills within their trained disciplines, and at the same time, have the opportunity and flexibility to team treat.

In our holistic milieu model, the therapists operate in both multidisciplinary and interdisciplinary capacities. This team is made up of neuropsychologists; speech–language pathologists; occupational, physical, and recreational therapists; a neurorehabilitation technician; psychiatrist; and a dietician. The team interfaces closely with the medical director, who is a psychiatrist. All therapists in the milieu “wear multiple hats.” For example, the speech–language pathologists and occupational therapists colead a group to teach patients the techniques of using a datebook system. A neuropsychologist and speech–language pathologist colead a psychoeducational group about the brain and the recovery process. Most therapists administer daily cognitive retraining activities. The neuropsychologists attend consultations with the psychiatrist, patients, and their family members. Multiple therapists participate in supervising work experiences and an occupational therapist, speech–language pathologist, and neuropsychologist conduct a vocational group that prepares patients with the essential skills to succeed in the work environment, including developing small work projects (inside garden, fish tank; Klonoff, 2010).

Managing and providing recommendations for the complex process of recovery requires a team that functions competently and collaboratively. Treating therapists need to recognize the “big picture” of the patients' personal goals and at the same time employ practicality and efficiency in the context of often limited treatment timelines.

Table 15.2 describes the *between* characteristics of the team. It contains the

inherent skills, qualities, and talents that occur and influence the relationships *between* team members, which ultimately impact the quality of patient care. Like the *within* characteristics, the list is not exhaustive and represents ideals to strive for in team settings. The *between* characteristics can be conceptualized along the cognitive, interpersonal and communication, and existential dimensions.

Of note, the patient's progress is mostly interdependent with a team that is healthy, integrated, and dedicated to the best services for and interest of their patients. The cohesiveness of the team is the most important precursor to the integrity and integration of the treatment process. When tensions, dissension, and disharmony dominate the team dynamics, valuable therapeutic energies are diverted and depleted from the treatment process. Therefore, patients' therapeutic gains and eventual outcomes are predicated on the energies, synergy, ethics, and vision of the team that is mentoring the process (Klonoff, 2010).

### Cognitive Capabilities

Team members require keen observational, analytical, and interpretative cognitive skills in order to accurately perceive and understand team dynamics (distinct from patients' clinical needs, per se). This includes knowledge and objectivity about the potential influence of the patients' psychodynamics on the treatment team's interactions, including transference reactions (Harty, 1979; Klonoff, 2010). Ultimately, therapists must also have the capabilities to set priorities and problem solve about the team's needs and challenges in order to optimize the efficacy of their treatment techniques. The wherewithal to think creatively or, "outside the box," enables the team to mobilize their energies toward inventive, flexible, and productive problem solving about their own personal strengths, capabilities, and pitfalls, as well as relevant patient interventions. For example, to maximize the patients' recovery and self-exploration, the treatment team should be able to conceptualize and promote ingenious techniques. To this end, in our setting, novel group treatment approaches have been proposed and implemented over time by various therapists, including adaptive tai chi and yoga groups, a newsletter group, and an adaptive book club for aphasic patients.

### Interpersonal and Communication Skills

The team members' social interaction skills with one another set the tone of the treatment setting and substantially influence the nature of social behaviors in the patients themselves. As with their interactions with patients, the team members'

listening capacity and perspective, degree of respect, empathy, and thoughtfulness toward one another defines the treatment ambience (Bettelheim, 1975; Harty, 1979; Kemper & von Wild, 2001; Klonoff, 2010).

Team members need to recognize and value how they both differ and complement one another in the treatment setting. Finding compromise or “middle ground” and openness to change are essential to maintaining positive working relationships within the team. Crucial to the viability, integrity, and longevity of the team are its members’ competence in communicating openly and appropriately to resolve conflict between one another (Butterill, O’Hanlon, & Book, 1992; Harty, 1979). This requires the sensitivity and willingness to find the suitable venue, time, and pace for feedback to colleagues about areas of dissension. This also translates to a collaborative style versus a judgmental, competitive, territorial, egotistical, dogmatic, or defensive stance.

**TABLE 15.2 *Between* Attributes of the Treatment Team**

<p><b>Therapist–Therapist Dynamics</b></p> <p><i>Cognitive Capabilities</i></p> <ul style="list-style-type: none"> <li>observe, analyze, and interpret team behaviors</li> <li>prioritize and flexibly problem solve team needs and challenges</li> <li>think creatively and “outside the box”</li> </ul> <p><i>Interpersonal and Communication Skills</i></p> <ul style="list-style-type: none"> <li>listening capacity and perspective</li> <li>respect, empathy, and thoughtfulness</li> <li>compromise or modify opinions</li> <li>conflict resolution</li> <li>collaboration and a team mentality</li> <li>give and take</li> <li>guide versus dictate</li> <li>camaraderie and collegiality</li> <li>build new relationships and work incrementally</li> <li>accountability/reliability toward the team</li> <li>investment in the team</li> <li>mutual trust and loyalty to the team</li> <li>show and share feelings and vulnerabilities</li> <li>team resiliency, cohesiveness, and stability</li> <li>create a “holding environment” and safe place</li> <li>prioritize team needs over self-gratifying personal needs</li> <li>cope with transference/countertransference to leadership and coworkers</li> </ul> <p><i>Existential Qualities</i></p> <ul style="list-style-type: none"> <li>collective responsibility, resolve, goal-setting, and team growth</li> <li>experiment and take chances</li> <li>experience inspiration/share work passions</li> <li>enthusiastic inquiry</li> <li>realism about collective limitations and vulnerabilities</li> <li>shared vision and meaning</li> <li>hopeful</li> </ul>
<p><b>Supervisor–Therapist Dynamics</b></p> <ul style="list-style-type: none"> <li>cosmic view</li> <li>pragmatist</li> <li>mediator</li> <li>mentor</li> <li>educator</li> <li>team advocate</li> <li>lead by example</li> </ul>

Team dynamics of uniting toward common goals must supersede the tendency to dominate or control other therapists. Treating staff must find the balance between their professional autonomy and their interdisciplinary treatment responsibilities. With a give-and-take attitude, each team member brings his or her wealth of experience and expertise, counterbalanced with the humility and openness to learn from others. This maximizes dialogue and innovation. The openness to guide (versus dictate) requires self-insight, sensitivity, and self-monitoring. The demonstration of this professionalism in a treatment milieu characterized by feelings of camaraderie and collegiality serves to model similar integral behaviors in our patients. As new team members join the treatment setting, the “old guard” needs to embrace them and demonstrate an

eagerness to build new relationships and work incrementally toward goals.

Team members are interrelated and interdependent in performing their duties; the entirety of the treatment process depends on a maximum degree of accountability, investment, and commitment toward one another, as well as the patients' needs. The team members' mutual trust ([Butterill et al., 1992](#)) and loyalty to one another and their mission are foundational for propelling the team's maturation and treatment process forward.

Therapists' capacity to express emotion to each other, including disclosing personal feelings of loss, pain, and sorrow create a foundation of honest dialogue and authenticity (which patients perceive and eventually emulate). Life events that create pain, disappointment, and trauma are inevitable in a treatment team, which has history and longevity. This can include the death or suicide of patients, or personal loss or illness in team members ([Klonoff, 2011](#)). How the team shares, addresses, and recovers from these adverse events foretells the process for the patients, who will bear witness to the coping strategies (or lack thereof) of the team. Similarly, a high degree of solidarity and psychological resiliency of the team in times of work-related distress or crisis is imperative in order to empathically support and nurture one another, as well as convey a sense of stability to the patients. The team's facility in creating and providing an emotionally and professionally safe and healthy working environment for one another allows for a sense of security and well-being within the treatment milieu. A hierarchy of needs to maintain the focus and evolution of the treatment process is critical. This requires the predominance of patient/family needs followed by the overall needs of the team to perform their best work, with the self-gratifying needs of the individual clinician relegated to a tertiary position.

Inherent in any therapeutic environment are feelings of transference and countertransference between the coworkers and team leader. Self-exploration and mature interaction by every therapist over perceived areas of disagreement, tensions, or "blind spots" are intrinsic to the survival of the therapeutic process.

## Existential Qualities

One of the chief missions of the team is to envision a future for the patients, which they generally cannot fathom for themselves, secondary to the nature and extent of their brain injuries, in combination with their reeling emotions in the injury aftermath. It also requires a collective, team-inspired sense of professional responsibility, resolve, and wish fulfillment on behalf of their patients. For this to emerge, the team itself must have the talents to set "lofty," yet attainable goals

and “stretch” in creative and novel directions. Sometimes, this relies on the team’s zest for experimenting with new ideas and taking chances. In our setting, this has involved incorporating younger and/or sometimes more acutely ill patients into the treatment setting, while simultaneously attending to their individual and developmental needs. This expansionistic thinking promulgates new horizons for the team, overall program growth and development, and by extension the accomplishments of the patients. Conversely, the mentality of indifference or pessimism (i.e., the negative self-fulfilling prophecy) contaminates the environment as well as deflates and depletes the collective energies of the team. Ultimately, this translates into discouragement and misery in the patients. The team’s capacity for enthusiastic inquiry, thereby sharing inspirations, vision, and meaningful values enhances the team’s commitment and dedication toward providing the best therapeutic interventions possible for their patients. Realistic embracement of the team’s collective limitations and vulnerabilities, while at the same time striving to realize their hopes on behalf of their professional growth and patient care keeps the team noncomplacent and vibrant in their life work (Klonoff & Lage, 1995).

### **The Psychotherapist as a Team Leader and Supervisor**

The psychotherapist who functions as a team leader and supervisor must encompass specialized qualities in order to gain the trust and respect of the team and supervisees. The previously described *within* attributes are all applicable. In addition to these, the team leader must have a “cosmic view” of a “game plan” that is goal-oriented and attainable (Klonoff, 2010). The team leader also requires the emotional and energy investment to “move the process forward” with, insight, clarity, and momentum (Klonoff, 2010). As a pragmatist, he or she must adeptly navigate complex clinical and administrative issues, also including resolving reimbursement strains. This entails being proactive, rather than reactive (Dell Orto & Power, 2000). While mediating, he or she also must be able to weigh and counterbalance varying perspectives and even biases in formulating optimum interventions and objectives.

The team leader should function as a mentor, with a commitment to cultivating the best qualities in the team members. As an educator, he or she can clarify, and when necessary, intercede about the complex and sometimes troublesome underlying psychodynamics between the patient and therapists.

The work of neurorehabilitation is intensive and inevitably becomes periodically emotionally draining for treating therapists (Klonoff, 2011). The



team leader can bolster the therapists' commitment and energy reserves through demonstrating a strong sense of loyalty and advocacy (Klonoff, 2010). Small representations of this include finding ways to verbally and financially reward staff for their contributions, demonstrating kindness and a receptivity to adapt schedules when the need arises, and constant accessibility to those individuals who need a helping hand or psychological boost.

Leading by example is vital for the respect and commitment of the team; the team leader needs to be actively immersed in the treatment milieu, not just a by-stander proposing esoteric solutions without the first-hand knowledge and experiential data. This is especially paramount when considering complex psychiatric and compliance issues, which can be particularly stressful and unnerving for the team. Minimizing a hierarchical arrangement in which the team views the leader as distant or uninvolved lends solidity to the efforts of the team. Overall, the team leader's role is to coalesce the team, patient, and support network so as to realize attainable goals in an atmosphere of nurturance and harmony.

## CASE STUDY

Dr. Maggie Retlaw is a 50-year-old female Caucasian psychotherapist raised in a liberal and loving home environment. She had a younger sister by 2 years, who at age 6 was diagnosed with poorly controlled epileptic seizures, following a high fever. Because Dr. Retlaw's parents owned a grocery store and worked long hours, she found herself the "protector" of her younger sister making sure she stayed out of harm's way. After school, Dr. Retlaw would entertain her sister by reading stories and playing card games; this was a great joy to her sister, who because of her seizure disorder was limited in her social and physical activities with her peers. After graduating from university with a PhD in neuropsychology, Dr. Retlaw found herself drawn to the field of psychotherapy for patients with acquired brain injuries. She pursued an eclectic education, which included mentoring with a psychiatrist who specialized in psychodynamics and self-psychology. Through her own life experiences and studies, she had developed a very patient and nurturing demeanor.

Initially, Dr. Retlaw worked independently as a private practitioner. However, she yearned for more collegial interactions, and so she joined a small multidisciplinary treatment team comprised of a rehabilitation physician, physical therapist, occupational therapist, and speech-language pathologist.

After about 6 months, Dr. Retlaw was referred a 30-year-old male patient,

Edward. After suffering a grand mal seizure, he had recently been diagnosed with a low grade brain tumor in the left frontal convexity, for which he underwent brain surgery. Postsurgery, he suffered from intermittent seizures, for which he took medication. Overall, Edward made a very good recovery, although he was left with mild to moderately impaired memory and attentional difficulties, as well as higher order executive function deficits in the areas of organization, flexible problem solving, judgment, multitasking, strategy generation, seeing the “big picture,” impulse control, planning, and decision making. Edward had been married to Mia for 5 years; they had a 6-year-old daughter, Rebecca.

A primary goal of neurorehabilitation was to provide psychoeducation and supportive psychotherapy to Edward and his wife, as his cognitive deficits were negatively impacting their relationship and overall family life. Mia was also very anxious and vigilant about Edward’s safety, as he tended to be somewhat impulsive and therefore reckless doing community activities.

Dr. Retlaw had a keen sense of intuition, practicality, and proactivity about other potential dangers for Edward. Her vigilance about his seizure risk promoted her to recommend that Edward not swim alone and always wear a life vest. Soon after this, he and his family were in his swimming pool when he suffered a grand mal seizure. Thankfully, because of these precautions, his wife was able to rescue him and then notify the paramedics. Given the decline in his seizure control, Dr. Retlaw promised to help Edward and his wife, Mia, locate another neurologist in a neighboring city. She interfaced with her colleague, the rehabilitation physician, and then facilitated an appointment, calling personally to expedite the process. Later, when loss of appetite and decreased weight became problematic, Dr. Retlaw advocated for nutrition services and assisted him in finding a dietician who specialized in neurological diagnoses.

Edward and Mia saw Dr. Retlaw weekly for 4 months and then bimonthly for 2 months. They were also furnished with Dr. Retlaw’s pager, in case of emergencies. They were respectful of this privilege, even though Dr. Retlaw assured them that it was acceptable to contact her if needed. Three months into their treatment, Edward demonstrated noticeable cognitive decline at home, and after being admitted to the hospital, Mia notified Dr. Retlaw by pager. Given Dr. Retlaw’s busy schedule, she made sure to visit them in his hospital room at the end of her workday. It turned out the decline was only temporary, related to mixing up his seizure medications, rather than tumor regrowth. Nonetheless, Dr. Retlaw’s compassion and dependability cemented their working relationship.

Through “productive worry,” Dr. Retlaw also arranged for a follow-up home visit with his occupational therapist to revisit how he was managing his medications in the home.

Related to Edward’s dysexecutive symptoms, he would sometimes have difficulty initiating and following through on home commitments, (e.g., cleaning and organizing his office). Dr. Retlaw’s gentle demeanor and kind interaction style helped mentor Mia, so she could react with less vexation. Dr. Retlaw’s attunement skills enabled her to respond in a nonjudgmental manner and validate Edward’s intermittent exasperation and panic, secondary to his newly discovered ineptitudes. Edward witnessed that Dr. Retlaw was trusting of both his and Mia’s feedback and was sincerely dedicated in helping to foster a more loving relationship. In a conjoint session, they developed the mantra, “ask, initiate, and anticipate” to cue Edward to be attentive to his family’s needs. With guidance, Edward also developed a list of the “10 things I could do to help make our relationship better” (e.g., take time to connect with Mia one on one) and 10 ways to implement them (e.g., write her a poem or love letter).

Dr. Retlaw’s colleagues, Joyce, a physical therapist; Fiona, an occupational therapist; and Alice, a speech-language pathologist worked collaboratively with Dr. Retlaw, Edward, and Mia. Joyce addressed Edward’s higher level balance and endurance difficulties and helped him develop a gym exercise program. Alice focused her therapies on improving Edward’s attentional, memory, and reasoning skills using cognitive retraining activities. She also assisted Edward in composing more concise and error-free work-related e-mails. Fiona conducted weekly home visits to coordinate home responsibilities among family members using a written daily checklist, as well as to help Edward organize his office work space so he could resume his home-based Internet sales business. She actively assisted Edward in organizing his iPhone to accommodate appointments and his “to do” list. Joyce, Fiona, and Alice were very thoughtful and invested in Edward’s rehabilitation. They eagerly arranged team meetings with Dr. Retlaw and developed inventive treatment techniques. Alice was a relatively new team member; Fiona willingly provided mentorship and invited Alice to cotreat at a home visit so that all parties could collaborate. Dr. Retlaw, Joyce, Alice, and Fiona also held bimonthly “debriefings” for mutual emotional support related to Edward’s fragile health. These sessions also served to galvanize personalized treatment techniques to maximize the family’s quality of life.

Related to Edward’s ongoing seizures, Dr. Retlaw found herself reflecting upon her feelings of helplessness and sorrow about her own sister’s lifelong

seizure-related challenges. However, she was careful through external supervision and self-scrutiny to contain these feelings, so as not to undermine the current treatment process. Instead, she found that her life experience helped to feed her empathic responsiveness. She found she could more easily put herself in Mia's predicament, as well as draw on her own wisdom and life lessons of "one step at a time," with respect to future planning. She drew on her inner resiliency and cohesiveness, recognizing that her primary function was to support and guide Edward and his family.

One of the areas Dr. Retlaw was most helpful in was providing direction for Edward regarding parenting skills. Dr. Retlaw's training did involve parenting skills after brain injury. However, she was curious and devoted to acquiring more knowledge about up-to-date treatment techniques, and so she undertook additional professional reading, as well as attending continuing education conferences. Treatment interventions included how to be flexible with his young daughter and to rely on the partnership with his wife for decision making and discipline. Dr. Retlaw's strong executive function skills (e.g., planning and problem solving) were also very beneficial in providing useful hints when specific home scenarios arose. She instituted parenting sessions, also including finding relevant parenting books, for example, *20 Teachable Virtues* (Unell & Wyckoff, 1995) and *The Last Lecture* (Pausch & Zaslow, 2008). Dr. Retlaw, Edward, and Mia also greatly enjoyed reminiscing in sessions about some of the humorous escapades with Rebecca, through pictures from Halloween and birthday celebrations.

Dr. Retlaw's life philosophy emphasized the "here and now," and letting the future unfold. She helped Edward and Mia realize that with his tumor diagnosis, every day counted. Through psychotherapy, Edward became adamant about living by his key life values. He strove to set apt schedules and timelines, including getting up early to make his wife and daughter breakfast, and limiting work activities to the hours his daughter was in school. He arranged local jaunts with his daughter and wife, to enjoy the environs. He volunteered to be a troop leader for Girl Scouts, and he planned regular family vacations, based on the preferences of Mia and Rebecca. His psychotherapist also enthusiastically helped him plan and implement innovative and eclectic date nights.

Dr. Retlaw had a strong existential outlook about her patients' futures, especially those with terminal illnesses. She pondered about creative and constructive ways that Edward could develop a personal and community-based legacy, should his health deteriorate. Through discussion with Edward and Mia,

they decided to dedicate themselves 100% to their relationship and parenting responsibilities with a goal of “no regrets.” They developed a videographic library of family experiences and events. Edward and Mia spent countless hours creating and cataloguing pictures with special captions, which they often shared during “family nights.” Toward the end of therapy, each family member developed a “1-year plan” which they shared in a conjoint session with Dr. Retlaw. This culminated in a family vacation to Australia.

Dr. Retlaw’s altruism and hopefulness, inspired by her history as a “wounded healer,” as well as nuggets of personal growth from this case catalyzed her involvement in community awareness projects. She volunteered her time to lead a support group for patients with epilepsy. She also encouraged Edward and Mia to find similar inspirational outlets. The couple decided to participate in a local fundraiser for brain tumor research. Through therapeutic dialogue, all parties were making sense out of Edward’s suffering.

After completing his rehabilitation, Edward wrote this letter to his therapists:

Never in my life did I think I was going to get a brain tumor or have brain surgery. Never in my life did I think I would be in a program like this; but I am glad that when my life needed it, you were here. I look back on fond memories—like when Mia and I would do our weekly schedule with our datebooks, and Rebecca would chime in with, “Okay what else do we need at the store?”—also making sure to jot that down in her miniaturized datebook . . . The most difficult challenge that anyone can ever cope with is when your own identity is in question. I cannot change something that has happened in the past; the only thing that matters is what I do about it. It is amazing how all of you have such patience and love for your work—it shows in everything you do. Thank you for creating such a healing environment.

## ACKNOWLEDGMENT

To Mr. Edward Koberstein for technical assistance and to Mr. Edward Koberstein, Dr. Brian Kelley, Dr. Susan Rumble, and Dr. Irwin Altman for reviewing earlier drafts of this chapter.

## REFERENCES

- Ackerman, S. J., & Hilsenroth, M. J. (2003). A review of therapist characteristics and techniques positively impacting the therapeutic alliance. *Clinical Psychology Review, 23*(1), 1–33.
- Aponte, H. J. (1996). Political bias, moral values, and spirituality in the training of psychotherapists. *Bulletin of the Menninger Clinic, 60*(4), 488–502.
- Basch, M. F. (1980). *Doing psychotherapy*. New York, NY: Basic Books.
- Bettelheim, B. (1975). The love that is enough: Countertransference and the ego processes of staff members in a therapeutic milieu. In P. Giovacchini (Ed.), *Tactics & techniques in psychoanalytic therapy* (Vol. 2, pp. 251–278). New York, NY: Aronson.
- Bien, T. H. (2004). Quantum change and psychotherapy. *Journal of Clinical Psychology, 60*(5), 493–501.

- Block, S. H. (1987). Psychotherapy of the individual with brain injury. *Brain Injury*, 1(2), 203–206.
- Bove, S., & Rizzi, M. (2009). Listening to intuition: Reflections on unconscious processes in the therapeutic relationship. *Transactional Analysis Journal*, 39(1), 39–45.
- Bowlby, J. (1988). *A secure base*. New York, NY: Basic Books.
- Butterill, D., O'Hanlon, J., & Book, H. (1992). When the system is the problem, don't blame the patient: Problems inherent in the interdisciplinary inpatient team. *Canadian Journal of Psychiatry*, 37(3), 168–172.
- Comas-Díaz, L. (2005). Becoming a multicultural psychotherapist: The confluence of culture, ethnicity, and gender. *Journal of Clinical Psychology*, 61(8), 973–981.
- Davar, E. (2008). To whom does the unconscious belong? The interface between the private and the social dimension in psychotherapy practice. *Psychodynamic Practice*, 14(2), 137–153.
- Dell Orto, A. E., & Power, P. W. (2000). *Brain injury and the family: A life and living perspective* (2nd ed.). Boca Raton, FL: CRC Press.
- Farber, B. A., Manevich, I., Metzger, J., & Saypol, E. (2005). Choosing psychotherapy as a career: Why did we cross that road? *Journal of Clinical Psychology*, 61(8), 1009–1031.
- Flaskas, C. (2009). The therapist's imagination of self in relation to clients: Beginning ideas on the flexibility of empathic imagination. *Australian & New Zealand Journal of Family Therapy*, 30(3), 147–159.
- Freed, P. (2002). Meeting of the minds: Ego reintegration after traumatic brain injury. *Bulletin of the Menninger Clinic*, 66(1), 61–78.
- Gans, J. S. (1983). Hate in the rehabilitation setting. *Archives of Physical Medicine and Rehabilitation*, 64(4), 176–179.
- Gross, S. (2001). On integrity. *Psychodynamic Counselling*, 7(2), 207–216.
- Gunther, M. S. (1971). Psychiatric consultation in a rehabilitation hospital: A regress hypothesis. *Comprehensive Psychiatry*, 12(6), 572–585.
- Gunther, M. S. (1977). The threatened staff: A psychoanalytic contribution to medical psychology. *Comprehensive Psychiatry*, 18(4), 385–397.
- Haldeman, D. C. (2010). Reflections of a gay male psychotherapist. *Psychotherapy*, 47(2), 177–185.
- Harty, M. K. (1979). Countertransference patterns in the psychiatric treatment team. *Bulletin of the Menninger Clinic*, 43(2), 105–122.
- Hersoug, A. G., Høglend, P., Havik, O., von der Lippe, A., & Monsen, J. (2009). Therapist characteristics influencing the quality of alliance in long-term psychotherapy. *Clinical Psychology & Psychotherapy*, 16(2), 100–110.
- Hojat, M. (2007). *Empathy in patient care: Antecedents, development, measurement, and outcomes*. New York, NY: Springer Science & Business Media, LLC.
- Hoshmand, L. T. (2006). Culture and the field of psychotherapy and counselling. In L. T. Hoshmand (Ed.), *Culture, psychotherapy and counselling* (pp. 25–46). Thousand Oaks, CA: Sage.
- Jung, C. G. (1951). *Fundamental questions of psychotherapy*. Princeton, NJ: Princeton University Press.
- Kemper, B., & von Wild, K. (2002). Requirements of team effectiveness in neurosurgical rehabilitation. *Acta Neurochirurgica*, 79, 37–39.
- Kitron, D. G. (2007). Psychoanalytic psychotherapy: The unmeasurable profession. *Psychoanalytic Review*, 94(3), 463–473.
- Klonoff, P. S. (1997). Individual and group psychotherapy in milieu-oriented neurorehabilitation. *Applied Neuropsychology*, 4(2), 107–118.
- Klonoff, P. S. (2010). *Psychotherapy after brain injury: Principles and techniques*. New York, NY: Guilford Press.
- Klonoff, P. S. (2011). A therapist experiential model of treatment for brain injury. *Bulletin of the Menninger Clinic*, 75(1), 21–45.
- Klonoff, P. S., & Lage, G. A. (1991). Narcissistic injury in patients with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 6(4), 11–21.

- Klonoff, P. S., & Lage, G. A. (1995). Suicide in patients with traumatic brain injury: Risk and prevention. *Journal of Head Trauma Rehabilitation, 10*(6), 16–24.
- Klonoff, P. S., Lage, G. A., & Chiapello, D. A. (1993). Varieties of the catastrophic reaction to brain injury: A self psychology perspective. *Bulletin of the Menninger Clinic, 57*(2), 227–241.
- Klonoff, P. S., & Myles, S. M. (2010). Sense of self and identity. In *Psychotherapy after brain injury: Principles and techniques* (pp. 75–99). New York, NY: Guilford Press.
- Knobel, M. (1990). Significance and importance of the psychotherapist's personality and experience. *Psychotherapy and Psychosomatics, 53*(1–4), 58–63.
- Kohut, H. (1984). The role of empathy in psychoanalytic cure. In A. Goldberg & P. Stepansky (Eds.), Chicago, IL: University of Chicago Press.
- Leber, W. R., & Jenkins, M. R. (1996). Psychotherapy with clients who have brain injuries and their families. In R. L. Adams, O. A. Parsons, J. L. Culbertson, & S. J. Nixon (Eds.), *Neuropsychology for clinical practice: Etiology, assessment, and treatment of common neurological disorders* (pp. 489–506). Washington, DC: American Psychological Association.
- Lewis, L. (1999). Transference and countertransference in psychotherapy with adults having traumatic brain injury. In K. G. Langer, L. Laatsch, & L. Lewis (Eds.), *Psychotherapeutic interventions for adults with brain injury or stroke: A clinician's treatment resource* (pp. 113–127). Madison, CT: Psychosocial Press.
- Lewis, L., & Rosenberg, S. J. (1990). Psychoanalytic psychotherapy with brain-injured adult psychiatric patients. *Journal of Nervous and Mental Disease, 178*(2), 69–77.
- Loewenthal, D. (2006). Cultural conflict, values, and relational learning in psychotherapy. In L. T. Hoshmand (Ed.), *Culture, psychotherapy and counseling* (pp. 205–225). Thousand Oaks, CA: Sage.
- Maslow, A. H. (1971). *The farther reaches of human nature*. New York, NY: Viking Press.
- Newman, C. F. (2002). A cognitive perspective on resistance in psychotherapy. *Journal of Clinical Psychology, 58*(2), 165–174.
- Pausch, R., & Zaslow, J. (2008). *The last lecture*. New York, NY: Hyperion.
- Pinkerton, R. (2008). "I apologize for being late": The courteous psychotherapist. *Psychotherapy, 45*(2), 273–277.
- Reupert, A. (2008). A trans-disciplinary study of the therapist's self. *European Journal of Psychotherapy and Counselling, 10*(4), 369–383.
- Rosenblatt, P. C. (2009). Providing therapy can be therapeutic for a therapist. *American Journal of Psychotherapy, 63*(2), 169–181.
- Scholl, J. C., & Ragan, S. L. (2003). The use of humor in promoting positive provider-patient interactions in a hospital rehabilitation unit. *Health Communication, 15*(3), 319–330.
- Siegel, D. J. (2010). *The mindful therapist: A clinician's guide to mindfulness and neural integration*. New York, NY: W. W. Norton.
- Strupp, H. H. (1973). *Psychotherapy: Clinical, research, and theoretical issues*. Lanham, MD: Jason Aronson.
- Unell, B. C., & Wyckoff, J. L. (1995). *20 teachable virtues: Practical ways to pass on lessons of virtue and character to your children*. New York, NY: Perigee Trade.
- Vasquez, M. J. T. (2007). Cultural difference and the therapeutic alliance: An evidence-based analysis. *American Psychologist, 62*(8), 878–885.
- Welling, H. (2005). The intuitive process. *Journal of Psychotherapy Integration, 15*(1), 19–47.
- White, C. (2007). Fertile ground at the edge of difference: Self, other and potential space: Commentary on paper by Gillian Straker. *Psychoanalytic Dialogues, 17*(2), 171–187.
- Yalom, I. D. (2002). *The gift of therapy: An open letter to a new generation of therapists and their patients*. New York, NY: HarperCollins.

# Transitioning in Neurorehabilitation

*Susan M. Rumble*

The period after acquiring a brain injury through the rebuilding of one's life afterward involves a great deal of change. Transition is defined in the literature as a passage of one life phase, condition, or status to another (Schlossberg, 1984). With regard to neurorehabilitation following acquired brain injury (ABI), there are several key periods of transition. Each period brings with it challenges for the patient and family, as well as for the professionals providing care. The transition period in neurorehabilitation has been identified as a time that little is known about, evidenced by limited literature on the transition period from hospital to home after ABI (Rittman, Boylstein, Hinojosa, Hinojosa, & Haun, 2007; Turner, Fleming, Ownsworth, & Cornwell, 2008). Most of the current literature focuses on this transition period from hospital to home and recommends increasing the level of support for the patient and his or her family during the transition phases following an ABI. This chapter will focus on the critical issues for patients and families during these transition periods, including the role of neuropsychologists in the transition process and proposed ways to improve support for patients and families in the transition process.

Qualitative studies find that patients recognize and experience distinct transition periods following ABI, and they report a need for better transition between rehabilitation programs and the community (Turner et al., 2007). In examining the need for services to help facilitate transition periods, three key transition phases can be considered in the neurorehabilitation process. The first major transition occurs when the patient is transferred from acute hospitalization to inpatient neurorehabilitation. The second major transition for patients and



families is the transition home and into outpatient therapies. The third key transition is the return to productivity, including paid or volunteer work, school, and other life roles. This final transition may be an evolving transition, particularly for children who may later require assistance with the transition from school to paid employment.

According to available literature and clinical experience, there are emotional, behavioral, and cognitive issues that need to be addressed for patients and families during the transition phases in neurorehabilitation. Depression and high stress levels are found in both patients and family members following hospital discharge after ABI (Turner, Fleming, Cornwell, Haines, & Ownsworth, 2009). Behavioral challenges are often a hindrance to successful return to productivity, as are injury-related cognitive deficits (Winkler, Unsworth, & Sloan, 2006; Ylvisaker et al., 2007). Given that patients have a variety of needs following ABI, multidisciplinary rehabilitation is often required. With regard to the needs of patients and families in the transition period, neuropsychologists can play a key role. Neuropsychologists possess both advanced knowledge of the brain and behavior, as well as the ability to carry out psychological intervention. These skills equip the neuropsychologist to provide support to patients and families as they navigate the transitions in neurorehabilitation following ABI.

In neurorehabilitation settings, as in other settings, neuropsychologists may play a number of different roles, including evaluative, education, intervention, or a combination of these. The structure in rehabilitation settings varies, such that a neuropsychologist may be the head of a rehabilitation team, a team member, or act as a consultant to the physician or treatment team. Regardless of the neuropsychologist's positioning in the neurorehabilitation process, there are opportunities to improve the transition experience by working with other professionals to improve continuity of care and through evaluation or intervention for emotional, behavioral, and cognitive needs of patients and families.

## **ACUTE HOSPITALIZATION TO INPATIENT REHABILITATION**

After the patient is medically stable in his or her acute hospitalization, the first transition is typically to an inpatient neurorehabilitation unit. Ideally, this transition should occur as soon as the physician managing the acute hospitalization care determines that the patient is stable enough to tolerate rehabilitation. Requirements for admission to an inpatient rehabilitation program may vary, but typically the patient must have the stamina for at least 3 hours of

therapies daily and have a medical need for rehabilitation of functions required for independence. A complicating factor at this transition phase is the possibility of delay in transfer to a rehabilitation unit, which has led to negative consequences when studied in a pediatric population (Tepas et al., 2009). In a retrospective study, Tepas et al. (2009) demonstrated that delays in starting comprehensive rehabilitation were correlated with reduced outcome and efficiency of rehabilitation. In this study, outcome was based on the change in scores of functional independence measurement (FIM) at the start and completion of inpatient rehabilitation and efficiency based upon the ratio of FIM score improvement to time in inpatient rehabilitation. The authors note that common causes for this delay in starting rehabilitation after being stabilized in critical care are often due either to bed availability or problems obtaining financial support for rehabilitation. They advocate for an ideal timeline of 24 hours between the transition from critical care to comprehensive inpatient rehabilitation.

At the time of this first transition, the patient may or may not be fully oriented. Needs for the patient may consist of helping him or her remain calm by providing orientation cues. Families at this time, in many cases, are filled with relief, happiness, or thankfulness that their loved one is alive. They often do not know about the possible outcomes or the length of therapy that will be required. The vast individual differences in patients and injury types make it difficult for professionals to predict the outcome as well. Anger, frustration, disbelief, shock, sadness, and grief are also common feelings, particularly for those families who have difficulty accessing appropriate rehabilitation (Turner et al., 2007). Family members report that this transition period is characterized by uncertainty, but that it is a shift from uncertainty about whether their loved one would die to uncertainty about the future and need for rehabilitation (Turner et al., 2007). The needs of the families and patients at this point may vary based on their level of understanding of ABI, as well as their level of coping and level of acceptance of the possible outcomes. Families report a variety of needs during the acute hospitalization stage that have been described as falling into four main themes: getting the news, uncertainty, making sense of the news, and moving on (Keenan & Joseph, 2010). With these needs in mind, some general intervention strategies and tools can be created to facilitate the transition for families.

Emotional support can be provided by assessing the family's level of coping and support. A single consultative session to evaluate the family's emotional needs can be helpful in this regard. One area to assess is the family's

support network. This includes whether they have emotional support, such as people they can talk to, as well as a support network for practical needs. Practical needs may include managing household affairs while they are with their loved one in the hospital, child care, whether they have a supportive workplace and availability to be away from work. If families are lacking in these areas, communicating with hospital social workers may be of help, as well as recommending that the family recruit available social resources as they prepare for what may be a very long road of recovery. The provision of educational material commensurate with the family's level of understanding and need can help in this new and uncertain time for the family. Explanation of, or introduction to the materials can help by allowing the family an opportunity to have their questions answered. This is important as a reduction in stress can be achieved when the meaning of brain injury and its effect on behavior is explored in therapy (Lezak, 1978). Recommending or providing additional supportive sessions may be helpful in this regard.

Although the uncertainty of the situation cannot be changed, providing education on the process of entering rehabilitation can offer some guidance or overview of what is to come. Provision of this information in the simplified form of a general road map may be of comfort for the families. Listing criteria for admission and discharge from rehabilitation, tailored to the specific programs in the local area, can help provide a framework of functional goals that the patient and family are working toward. For families struggling to cope, encouraging them to track their loved one's progress from day to day in a journal can be a helpful intervention. This is useful as it keeps the focus on successes, and during difficult times, one can review the journal to help keep in mind the big picture of how far they have come.

Behavioral intervention may be requested of a neuropsychologist in either the acute or inpatient neurorehabilitation settings. To ease the transition between these settings, a well-constructed behavior management plan may need to be initiated; however, the patient's cognitive limitations and level of orientation need to be considered in the construction of the plan. If a patient is in posttraumatic amnesia, frequent reminders of the parameters of the plan will be necessary. Vail beds or bed restraints may be required depending on the patient's level of cognitive functioning, and more often the behavior management plans can help to reduce the necessity of these restrictive interventions once the patient is stable and has transitioned into inpatient rehabilitation.

Cognitive evaluation at this early transition stage may consist of assessing

mental status, orientation, and reporting on clearance from posttraumatic amnesia. In cases of milder traumatic brain injury (TBI), a neuropsychological consultation may be requested to help provide recommendations for patients who may be functioning well enough to transition directly home without inpatient rehabilitation. In this case, commenting on the patient's ability to be safe and the level of supervision likely needed at home are important, as well as readiness to return to work, and timeline for follow-up evaluations to determine progress and update recommendations.

### **INPATIENT TO HOME TRANSITION**

The transition period from hospital to home is associated with challenges for patients with a variety of ailments (fractures, cardiac/vascular problems, spinal cord injuries, ABI (Gage, Cook, & Fryday-Field, 1997). Patients report feeling isolated, having difficulty accessing community resources, and feeling unprepared for the practical and emotional challenges associated with their return to the community. Following ABI, patients often experience physical, cognitive, and behavioral changes that persist long after they return home from the hospital, some of which may be lifelong changes (Winkler et al., 2006). Upon their initial transition to home, patients and families may experience feelings of loss and separation from the professionals who were a key part of their daily care and support for so long (Lefebvre, Pelchat, Swaine, Gélinas, & Levert, 2005). Rusconi and Turner-Stokes (2003) reported that many patients did not feel prepared for the reduction in amount of therapy upon their transition home, with 39% feeling that their therapy ended early or was inadequate. Lefebvre et al. (2005) found that when a small group of patients were interviewed, patients with TBI and their families all criticize the lack of available resources upon their transition home. For many patients, especially those who do not live near a metropolitan area, the ability to access outpatient rehabilitation services is likely limited.

The reported dissatisfaction with lack of services and support following inpatient rehabilitation for ABI is understandable, given that in a study of patients with TBI by Mellick, Gerhart, and Whiteneck (2003) almost two thirds received no services following acute hospitalization. Although most of those patients had mild to moderate injuries, they were found to have functional impairments, as measured by functional independence assessment, and a higher unemployment rate than the general public. Barnes, Frank, Montgomery, and Nichols (2005) studied factors that predict provision of rehabilitative services

following TBI and found that injury severity was the most significant predictor of receiving services, and of whether they were inpatient or outpatient services. Admission to an intensive care unit, being male, having comorbid orthopedic injury, and having medical insurance coverage were also predictive of more rehabilitation service provision. This dissatisfaction with services can be addressed by better preparation and planning with the families as they near the time of hospital discharge. Engaging support from social work or encouraging the families to contact resources before hospital discharge can be helpful. Helping families to understand the necessity of initiating the next phase of rehabilitation and the need to advocate for their loved ones could help them prepare. Given the aforementioned literature, this should particularly be emphasized for patients with less severe injuries, females, and those without comorbid orthopedic injuries.

Turner *et al.* (2007) found that some patients tend to isolate themselves from their community and social network following ABI and that these people were more likely to experience other negative events in their transition home (Turner *et al.*, 2007). In this group of patients, those with more difficult transition experiences reported feeling bored during these first months. The patients were more likely to report an overall successful transition experience when they were able to engage in meaningful activities during the main part of their day (hobbies, volunteer work, employment, etc.). Therefore, patients may benefit from recommendations to begin some type of activity if they are able. In recommending activities, the neuropsychologist will need to consider injury-related deficits that may require the patient to have assistance to compensate for deficits of initiation, organization, physical, or other impairments that will impede the patient's ability to engage in various activities.

In the initial transition home, patients should be considered at risk of experiencing emotional difficulties. Turner *et al.* (2009) found increasing levels of patient depression and stress during the transition phase from inpatient rehabilitation to home after ABI (Turner *et al.*, 2009). This transition time in ABI has been described by patients as a time when self-awareness of deficits increases, which some suggest may be associated with the increased emotional distress and self-reports of anxiety and depression that are also observed at this time (Turner *et al.*, 2007). However, these may or may not be directly related to the increased awareness (Cott, Wiles, & Devitt, 2007). Fleming, Winnington, McGillivray, Tatarevic, and Ownsworth (2006) found increases in self-awareness and depression following hospital discharge in TBI; however, no

consistent relationship was found among the level of self-awareness, emotional functioning, and community integration. The authors suggest that the transition phase immediately following hospital discharge is an important phase for interventions targeting adjustment to disability and compensation strategies (Fleming et al., 2006). Regardless of the etiology, anxiety, depression, and adjustment issues should be addressed to improve emotional functioning in the transition, clearing obstacles to completing therapy to address physical, cognitive, and behavioral changes.

A potential impediment to treating the emotional functioning may be a patient's lack of awareness of his or her injury-related deficits. For many reasons, the reality of postinjury deficits is often not apparent to the patient with ABI upon his or her discharge from the hospital. This impaired awareness of deficits, or anosognosia, is observed to be a residual impairment following ABI lasting months to years in some cases (Prigatano et al., 1986). The limited awareness is not typically global, and rather there is limited awareness for specific deficits (Cott et al., 2007). Although impaired self-awareness may be a direct result of the injury, it can also result from psychologically protective mechanisms such as denial (see Prigatano, 2010b for a comprehensive work on anosognosia).

The relevance of limited awareness of deficits to the transition from hospital to home is that poor self-awareness following TBI has been found to be related to poor treatment compliance and the ability to form a working alliance with therapists (Schönberger, Humle, & Teasdale, 2006; Trahan, Pépin, & Hopps, 2006). Patients may not comply with recommendations for rehabilitation therapies or with recommendations to delay their return to work or school. Many work settings and schools will require a physician's release to return after such a significant injury. However, some settings may be less prepared for these situations or may not enforce their standard procedures, particularly if the patient appears well, or is in a position of authority in the work setting. Returning to work with impairments and limited self-awareness can be dangerous and puts the patient at risk of termination or loss of needed disability benefits. Family support and involvement is crucial to engaging the patient in outpatient rehabilitative therapy in such situations. Psychotherapeutic intervention by a neuropsychologist to treat impaired self-awareness may be useful in this transition period to foster the ability to form a working alliance and address treatment compliance. (A description of psychotherapeutic techniques is beyond the scope of this chapter; please see Klonoff (2010) for psychotherapy principles

and techniques after brain injury).

Caregiver burden during the transition home cannot be overlooked. The practical burdens of financial support, facilitating transportation to appointments and community resources, and daily living care fall primarily on the caregiver (Turner *et al.*, 2007). Turner *et al.* (2010) found that the transition period from hospital to home was notable for prevalent emotional strain on caregivers with the rate of depressive symptoms in caregivers being higher than in the general population. Studies have found that the highest levels of depressive symptoms in caregivers are found just prior to discharge and decreasing thereafter (Turner *et al.*, 2009). There is some evidence that caregivers may benefit from treatment in this transition period (Perrin *et al.*, 2010). Turner *et al.* (2010) recommend providing more specific caregiver support and preparation prior to hospital discharge. They additionally recommend that ensuring adequate time in inpatient rehabilitation is beneficial for caregiver well-being.

Discharge planning programs for caregivers and patients have been implemented in recent years by health care providers to maximize discharge outcomes (Turner *et al.*, 2008). A recent study by Perrin *et al.* (2010) evaluated a Transition Assistance Program developed for caregivers of stroke patients. The program included skill development, education, and supportive problem solving, with 122 participants who were assigned to either the treatment group or a control group. The patients reported high program satisfaction, and the program was found to reduce caregiver strain at 3-month follow-up (Perrin *et al.*, 2010). A trend was noted for lower depression in the treatment group at the 3-month follow-up time. The authors suggest this may indicate a preventative effect on depression for caregivers who were not depressed at the time of discharge. Providing intervention for caregivers may also have benefit to the patients. Intensifying therapy during the transition home after stroke has been shown to have long-term effects in a study of stroke patients reducing institutionalization and mortality at 31-month follow-up (Gräsel, Schmidt, Biehler, & Schupp, 2006). This study compared a group whose families were able to watch therapy prior to discharge, with the intensive transition program. The intensive transition program added the following interventions: therapeutic weekend care at home before discharge, an individual training course for family carers, a psychoeducational seminar for family carers, and telephone counseling 3 months after discharge. These studies show promise for the benefit of increased intervention for both patients and caregivers in the transition home following ABI. Further evaluation and implementation of various discharge planning

interventions will be an important support that neuropsychologists can provide at a systems level, where study outcomes may be used to lobby for increased financial support of successful interventions.

In some cases, patients with ABI may not have family support to take on a caregiving role. For such patients, transitional living programs may be available. Transitional living programs are provided to foster independent living when patients are able to transition out of inpatient rehabilitation, but are not yet able to be independent in the community (Simpson et al., 2004). These residential facilities are often close to community resources. There is a focus on independent living, including domestic tasks, personal time management, accessing community resources, and building and maintaining of social relationships (Simpson et al., 2004). Therapy is provided by a multidisciplinary team to help patients achieve functional goals (Minnes, Harrick, Carlson, & Johnston, 1998). Patients see improvements in independent living, psychosocial outcomes, and in global functioning (Simpson et al., 2004). This concept is ideal for patients who are ready to transition out of inpatient rehabilitation but may not have viable options for supervision at home. To date, there are no randomized control trials for transitional living programs (Turner et al., 2008).

When considering ways to improve accessibility of support for patients and families through the transition from hospital to home, Internet and telephone may be viable options that deserve consideration. Distance therapy has been shown to have some success in providing support for children and their families following pediatric ABI. Wade et al. (2006) found less global distress, particularly in patient depression and anxiety symptoms, and improved problem-solving skills following a primarily web-based family problem-solving intervention. In the treatment group of this study, the investigators initially met face to face in the home with the child and his or her family to build rapport and administer a structured assessment. Weekly Internet-based videoconferencing sessions were then conducted with the families with self-guided instruction that the families completed prior to the sessions. Education was provided related to behavioral and cognitive changes that commonly follow TBI, as well as behavioral management techniques. The findings were significant in comparison to a control group, which was provided with Internet resources. This particular intervention was carried out with families several months following their TBI. However, the success of the intervention opens the potential to try such a program in the transition period. This could provide families with easily accessible education about their child's injury and ways to manage behavior as



they transition home from the hospital.

Another promising study regarding distance intervention in the transition period home from the hospital and during the first year following adult TBI was conducted by [Bell et al. \(2005\)](#). They found that patients who received regular telephone contact by researchers showed a higher functional status and quality of life when compared to a control group receiving the standard hospital discharge of referral to outpatient services with no further contact. The telephone intervention group received regular calls consisting of counseling, education, brief motivational interviewing, and facilitation of follow-up appointments. The patients were randomized to either the intervention group or control group; however, the study did not consider what other therapies the patients in either group were receiving. Although more research is needed to replicate the findings and identify the factors related to the improved functioning, this is a promising step as it may provide a viable adjunctive support option for people in rural communities, those with transportation issues, or a low-cost option for those with limited or no financial support for more intensive or holistic therapies.

Neuropsychological evaluations are conducted prior to discharge from inpatient neurorehabilitation and are reviewed elsewhere in this book. To provide greater support to the patients and families during the transition, the feedback session of a neuropsychological evaluation should include both the patient and family whenever possible. They should be encouraged to take notes or be provided with written recommendations and educational information on brain injury. Experience with families following discharge from inpatient neurorehabilitation proves that many families are overwhelmed and may not recall even very important details such as the need for 24-hour supervision. Providing information to families in written format can be helpful, including names and numbers of community resources such as the local brain injury association, psychotherapists who are experienced in working with brain injury, social work resources familiar with brain injury, outpatient rehabilitation resources, and caregiver services if 24-hour supervision is required. When possible, being available by telephone on a nonemergency basis to clarify recommendations can be comforting and helpful to families.

Behavioral and environmental control recommendations may be necessary as the patient transitions home from inpatient rehabilitation. These recommendations can be provided to families prior to the patient's discharge. It is often necessary to educate families about reducing stimulation in the environment after returning home. In addition to providing general

recommendations such as limiting visitors to one or two at a time, turning off television or radio, and so forth, it can be helpful to inquire about specific environmental stimuli in the home. For example, if there are many children or pets around, it may be helpful to designate a place where the patient can take a quiet break. If impulsivity or poor judgment is of concern, the family may need guidance about keeping sharp instruments or weapons out of the patient's access until these or other concerns are lessened. Information from the neuropsychological evaluation and from successful behavioral techniques used in the hospital can be integrated with information about the home environment provided by the family to help prepare them for the transition home.

## RETURN TO PRODUCTIVITY

The transition back to productivity, including work, school, and other preinjury roles, is an important step in a patient's recovery following ABI. Inability to successfully make this transition can impact both personal financial stability as well as place financial strain on systems that support disabled persons. Inability to return successfully to productivity also has an impact on level of satisfaction with life (Burleigh, Farber, & Gillard, 1998). Following ABI, unemployment and passive life styles have been associated with decreased life satisfaction. Supporting patients through the return to productivity and community/social integration is a vital step in rehabilitation.

Productivity rates and employment often decline following ABI. Dikmen *et al.* (1994) investigated the rate of return to productivity following TBI for patients without rehabilitation benefits for vocation. When including all severity levels, 66% of patients had returned to work 1 year after injury, and 72% had returned 2 years after injury. However, return-to-work rates were significantly lower for those who sustained severe injuries, with only 37% returning to work 2 years following the injury. In addition, of those patients following ABI who return to employment, only a small number are able to return to the same level as their preinjury employment (McCabe *et al.*, 2007).

When investigating barriers to community integration following ABI, several factors have been found to be significant. Severity of injury, age at injury, level of disability, and behavioral dysregulation issues have all been found to limit level of community integration (Winkler *et al.*, 2006). Of these, the challenging behavior (impulsivity, aggression, irritability, and restlessness) following TBI is the only one of these factors that may be altered by intervention. Patients have been found to have better integration when few of the

challenging behaviors are present (Winkler et al., 2006). In a review of outcomes from interventions following TBI (Ylvisaker et al., 2007), both children and adults tend to show more externalizing behaviors than internalizing behavior. Disinhibition, irritability, aggression, anger dyscontrol, social deficits and withdrawal, and depression often associated with poorer outcome (Ylvisaker et al., 2007). Interventions to minimize these challenging behaviors are important for a successful return to preinjury roles. Ylvisaker et al. (2007) reviewed behavioral interventions for adults and children following TBI and concluded that behavioral interventions are considered treatment guidelines, with both contingency management procedures and positive behavioral support procedures deemed evidence-based treatment options.

Studies have evaluated the role of intervention in successful return to work following ABI. Parente and Stapleton (1999) found that compared to a matched control group, patients who participated in groups that provided continuous coaching and cognitive strategies applicable to work were more likely to return to full-time employment. In one qualitative study in which almost all patients expressed a desire to be able to return to work, those who were participating in a supported return-to-work program were more likely to have maintained employment, with others having failed after attempting a return to employment (Turner et al., 2007). Prigatano et al. (1994) used a prospective control group design to study the level of productivity in a protected work trial finding higher productivity in the treatment group. In reviewing various studies of cognitive strategies such as those listed above, McCabe et al. (2007) report that there is limited evidence supporting that cognitive strategies increase the likelihood that patients will return to full-time employment. Limited evidence was also found for supported employment strategies following ABI to improve job placement and retention. These studies emphasize the importance of continued rehabilitation support through the return to gainful employment.

On preparing for the transition to school following a brain injury, a key support that can be offered to the family includes helping to develop a home-school partnership (Semrud-Clikeman, 2010). This will be necessary as the child's educational needs and supports will require adjustment as the child progresses in recovery and continues to mature and develop. The child's family and school will need to be flexible in relation to the child's changing needs and will need to reach a common understanding to work toward finding the most appropriate supports in the school and community (Semrud-Clikeman, Pliszka, Bledsoe, & Lancaster, 2012).

Children and adolescents who will return to school will benefit from provision of specific recommendations to help them capitalize on their strengths and procure accommodations and treatment for their injury-related deficits. For this reason, it is important for the neuropsychologist working with the pediatric or adolescent populations to know the local laws with regard to services the schools are required to provide. It is also important to know or enquire about the capability of the local school district to provide services, as this will vary from district to district. In some cases, a transfer to a district more equipped to manage the necessary accommodations may be of benefit to the patient. Communication with the school's administration, teachers, school psychologists, and speech therapists is important. This may be done through report recommendations; however, if the family provides a release of information for school personnel, communicating directly with the school personnel is preferred. Attending individual education plan (IEP) meetings can be useful as there are typically several educators involved in the child's care in the same room, where specific suggestions can be explained. Experience has found educators to be supportive of telephone conferencing for these meetings, which is useful for students who live a distance from rehabilitation services. However, further support may be needed, including observation of the child during the school day to evaluate his or her needs in the classroom and individualize recommendations.

In a review of empirical research on rehabilitation in pediatric TBI, [Semrud-Clikeman \(2010\)](#) identifies the importance of the pediatric neuropsychologist to provide support for schools and families through both therapy for the patient and in-services for school personnel. Providing this in-service education may involve general information about brain injury, although information about the specific struggles of the child they will be working with is invaluable. Cognitive therapy sessions provided by the neuropsychologist or other therapists may be conducted at the school if necessary to provide an opportunity for aides or others working with the patient so see the techniques that have been found successful for the child. The school personnel can then implement the same strategies with the child on a daily basis. Most important, the aforementioned home-school partnership is key in this process, as the patient's needs will change as he or she grows and develops in the context of his or her brain injury.

## **TRANSITION FROM SCHOOL TO WORK**

Return to school is merely the beginning of a series of transitions for a child.

Because the child with a brain injury will continue to grow and develop, his or her recommendations and accommodations at school will need regular modification. Without this very important follow-up, later transitions such as moving on to either work or postsecondary education can be impacted.

A consideration for patients and families in this transition period may be whether to be enrolled in school-based life-skills programs leading toward a modified diploma, or general academic track. A study of postsecondary transition outcomes by [Todis and Glang \(2008\)](#) found that students enrolled in life-skills programs were eligible for transition services through school until age 22. However, these students graduated with a modified diploma and lacked the skills and credits to enroll in most postsecondary training. Students in the study who participated in learning resource centers in school more often qualified to receive a regular diploma, but were then not entitled to receive transition services from the school. This study highlights the difficulty that students may face when entering the transition from high school to either secondary education or employment. In a study of the transition following high school for patients with TBI, 80% of parents reported that their children needed further services and life-skills training following high school ([Wagner, Newman, Cameto, Garza, & Levine, 2005](#)). From a systems perspective, continued efforts at improving the educational transition services available for children and adolescent with ABI is necessary.

Government-funded vocational rehabilitation services may be available to fund rehabilitation services to help the teenager transition to work after high school. However, some studies have found that the assumption that patients will use these resources has reduced the services offered through the schools ([Todis & Glang, 2008](#)). Students with TBI in this study were more likely to complete postsecondary programs if they were linked with disability services and support agencies outside of school. This transition period is a key opportunity for intervention both directly with the patient and through in-service training at schools. At this juncture a reevaluation of neuropsychological functioning is also important to help guide the necessary services to help the student transition to either postsecondary training or to a workplace.

## SUMMARY

This chapter highlights the key transition processes in neurorehabilitation and opportunities for neuropsychological intervention. The key transitions include the transition from acute care to inpatient rehabilitation, the transition home, and

the return to productivity. Family needs during the first transition phase are primarily for support of and reduction in the uncertainty surrounding their situation. During the transition period from hospital to home, available research suggests that both patients and caregivers experience emotional difficulties. Early, holistic, and intensive postdischarge rehabilitation services are recommended to support psychosocial, emotional, and global functioning during the transition phase (Turner et al., 2009). This is a sound recommendation given the need for multidisciplinary therapies following brain injuries to treat the physical, cognitive, and emotional/behavioral deficits. Patients may require psychotherapeutic intervention to increase awareness of deficits, which can impact ability to form a working alliance in rehabilitation therapies. Other interventions, including telephone and web-based interventions, show promise as potential adjunctive support for managing emotional, cognitive, and behavioral issues that may arise following ABI. This may help increase availability of support for the many patients who report limited support following their transition home. The transition back to productivity is important for patients both financially and for satisfaction with life. Patients appear to benefit from cognitive coaching and behavioral intervention during this transition. For patients returning to school, helping to develop a home-school partnership is recommended as a key intervention (Semrud-Clikeman, 2010). Communicating with and providing in-service education for school personnel is also an important function for the neuropsychologist. Finally, repeat evaluations are important for the student or professional whenever new transitions arise in life (change in work, birth of a child, loss of a caregiver, etc.).

Neuropsychologists provide services to patients through evaluation, education, and intervention addressing cognitive, behavioral, and emotional domains. Neuropsychology also has a role in conducting research and collaborating with other rehabilitation professionals to evaluate patient needs and effectiveness of interventions. The recognition of the need to improve support for patients during transitions in neurorehabilitation is a relatively recent revelation, with research growing in recent years. With continued focus on facilitating the transition process in neurorehabilitation, it is feasible that outcomes may be improved for patients with ABI and their families.

## REFERENCES

- Barnes, E. F., Frank, E. M., Montgomery, A., & Nichols, M. (2005). Factors predicting service provision in adults with traumatic brain injury. *Journal of Medical Speech-Language Pathology, 13*(1), 69–84.
- Bell, K. R., Temkin, N. R., Esselman, P. C., Doctor, J. N., Bombardier, C. H., Fraser, R. T., . . . Dikmen, S.

- (2005). The effect of a scheduled telephone intervention on outcome after moderate to severe traumatic brain injury: A randomized trial. *Archives of Physical Medicine and Rehabilitation*, 86(5), 851–856.
- Burleigh, S. A., Farber, R. S., & Gillard, M. (1998). Community integration and life satisfaction after traumatic brain injury: Long-term findings. *American Journal of Occupational Therapy*, 52(1), 45–52.
- Cott, C. A., Wiles, R., & Devitt, R. (2007). Continuity, transition and participation: Preparing clients for life in the community post-stroke. *Disability and Rehabilitation*, 29(20–21), 1566–1574.
- Dikmen, S. S., Temkin, N. R., Machamer, J. E., Holubkov, A. L., Fraser, R. T., & Winn, H. R. (1994). Employment following traumatic head injuries. *Archives of Neurology*, 51(2), 177–186.
- Fleming, J., Winnington, H., McGillivray, A., Tatarevic, B., & Ownsworth, T. (2006). The development of self-awareness and relationship to emotional functioning during early community reintegration after traumatic brain injury. *Brain Impairment*, 7(2), 83–94.
- Gage, M., Cook, J. V., & Fryday-Field, K. (1997). Understanding the transition to community living after discharge from an acute care hospital: An exploratory study. *American Journal of Occupational Therapy*, 51(2), 96–103.
- Gräsel, E., Schmidt, R., Biehler, J., & Schupp, W. (2006). Long-term effects of the intensification of the transition between inpatient neurological rehabilitation and home care of stroke patients. *Clinical Rehabilitation*, 20(7), 577–583.
- Keenan, A., & Joseph, L. (2010). The needs of family members of severe traumatic brain injured patients during critical and acute care: A qualitative study. *Canadian Journal of Neuroscience Nursing*, 32(3), 25–35.
- Klonoff, P. S. (2010). *Psychotherapy after brain injury: Principles and techniques*. New York, NY: Guilford Press.
- Lefebvre, H., Pelchat, D., Swaine, B., Gélinas, I., & Levert, M. J. (2005). The experiences of individuals with a traumatic brain injury, families, physicians and health professionals regarding care provided throughout the continuum. *Brain Injury*, 19(8), 585–597.
- Lezak, M. D. (1978). Living with the characterologically altered brain injured patient. *Journal of Clinical Psychiatry*, 39(7), 592–598.
- McCabe, P., Lippert, C., Weiser, M., Hilditch, M., Hartridge, C., & Villamere, J. (2007). Community reintegration following acquired brain injury. *Brain Injury*, 21(2), 231–257.
- Mellick, D., Gerhart, K. A., & Whiteneck, G. G. (2003). Understanding outcomes based on the post-acute hospitalization pathways followed by persons with traumatic brain injury. *Brain Injury*, 17(1), 55–71.
- Minnes, P., Harrick, L., Carlson, P., & Johnston, J. (1998). A transitional living environment for persons with brain injuries: Staff and client perceptions. *Brain Injury*, 12(11), 987–992.
- Parente, R., & Stapleton, M. (1999). Development of a cognitive strategies group for vocational training after traumatic brain injury. *Neuro Rehabilitation*, 13, 13–20.
- Perrin, P. B., Johnston, A., Vogel, B., Heesacker, M., Vega-Trujillo, M., Anderson, J., & Rittman, M. (2010). A culturally sensitive Transition Assistance Program for stroke caregivers: Examining caregiver mental health and stroke rehabilitation. *Journal of Rehabilitation Research and Development*, 47(7), 605–617.
- Prigatano, G. (2010a). Anosognosia after traumatic brain injury. In G. P. Prigatano (Ed.), *The study of anosognosia* (pp. 229–254). New York, NY: Oxford University Press.
- Prigatano, G. (Ed.). (2010b). *The study of anosognosia*. New York, NY: Oxford University Press.
- Prigatano, G. P., Fordyce, D. J., Zeiner, H. K., Roueche, J. R., Pepping, M., & Wood, B. (1986). *Neuropsychological rehabilitation after brain injury*. Baltimore, MD: Johns Hopkins University Press.
- Prigatano, G. P., Klonoff, P. S., O'Brien, K. P., Altman, I. M., Amin, K., Chiapello, D., et al. (1994). Productivity after neuropsychologically oriented milieu rehabilitation. *Journal of Head Trauma Rehabilitation*, 9, 91–102.
- Rittman, M., Boylstein, C., Hinojosa, R., Hinojosa, M. S., & Haun, J. (2007). Transition experiences of stroke survivors following discharge home. *Topics in Stroke Rehabilitation*, 14(2), 21–31.
- Rusconi, S., & Turner-Stokes, L. (2003). An evaluation of aftercare following discharge from a specialist

- inpatient rehabilitation service. *Disability and Rehabilitation*, 25(22), 1281–1288.
- Schlossberg, N. (1984). *Counseling adults in transition: Link practice with theory*. New York, NY: Springer Publishing Company.
- Schönberger, M., Humle, F., & Teasdale, T. W. (2006). Subjective outcome of brain injury rehabilitation in relation to the therapeutic working alliance, client compliance and awareness. *Brain Injury*, 20(12), 1271–1282.
- Semrud-Clikeman, M. (2001). *Traumatic brain injury in children and adolescents*. New York, NY: Guilford Press.
- Semrud-Clikeman, M. (2010). Pediatric traumatic brain injury: Rehabilitation and transition to home and school. *Applied Neuropsychology*, 17, 116–122.
- Simpson, G., Secheny, T., Lane-Brown, A., Strettles, B., Ferry, K., & Phillips, J. (2004). Post-acute rehabilitation for people with traumatic brain injury: A model description and evaluation of the Liverpool Hospital Transitional Living Program. *Brain Impairment*, 5(1), 67–80.
- Tepas, J. J., Leaphart, C. L., Pieper, P., Beaulieu, C. L., Spierre, L. R., Tuten, J. D., & Celso, B. G. (2009). The effect of delay in rehabilitation on outcome of severe traumatic brain injury. *Journal of Pediatric Surgery*, 44(2), 368–372.
- Todis, B., & Glang, A. (2008). Redefining success: Results of a qualitative study of postsecondary transition outcomes for youth with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 23(4), 252–263.
- Trahan, E., Pépin, M., & Hopps, S. (2006). Impaired awareness of deficits and treatment adherence among people with traumatic brain injury or spinal cord injury. *Journal of Head Trauma Rehabilitation*, 21(3), 226–235.
- Turner, B., Fleming, J., Cornwell, P., Worrall, L., Ownsworth, T., Haines, T., . . . Chenoweth, L. (2007). A qualitative study of the transition from hospital to home for individuals with acquired brain injury and their family caregivers. *Brain Injury*, 21(11), 1119–1130.
- Turner, B. J., Fleming, J. M., Ownsworth, T. L., & Cornwell, P. L. (2008). The transition from hospital to home for individuals with acquired brain injury: A literature review and research recommendations. *Disability and Rehabilitation*, 30(16), 1153–1176.
- Turner, B., Fleming, J., Cornwell, P., Haines, T., & Ownsworth, T. (2009). Profiling early outcomes during the transition from hospital to home after brain injury. *Brain Injury*, 23(1), 51–60.
- Turner, B., Fleming, J., Parry, J., Vromans, M., Cornwell, P., Gordon, C., & Ownsworth, T. (2010). Caregivers of adults with traumatic brain injury: The emotional impact of transition from hospital to home. *Brain Impairment*, 11(3), 281–292.
- Wade, S. L., Michaud, L., & Brown, T. M. (2006). Putting the pieces together: Preliminary efficacy of a family problem-solving intervention for children with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 21(1), 57–67.
- Wagner, M., Newman, L., Cameto, R., Garza, N., & Levine, P. (2005). *After high school: A first look at the post-school experiences of youth with disabilities: A report from the National Longitudinal Transition Study-2 (NLTS2)*. Menlo Park, CA: SRI International. Retrieved from [http://www.nlts2.org/nlts2\\_textonly/reports/reports\\_collapsed.html](http://www.nlts2.org/nlts2_textonly/reports/reports_collapsed.html)
- Winkler, D., Unsworth, C., & Sloan, S. (2006). Factors that lead to successful community integration following severe traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 21(1), 8–21.
- Ylvisaker, M., Turkstra, L., Coelho, C. A., Yorkston, K., Kennedy, M., Sohlberg, M. M., & Avery, J. (2007). Behavioral interventions for children and adults with behavior disorders after TBI: A systematic review of the evidence. *Brain Injury*, 3, 67–71.



# Cross-Specialty Collaboration in Neuropsychological Rehabilitation

*Therese Meyer-Cox and Christine V. Paradee*

The formalized practice of cross-specialty collaboration in rehabilitation has its origin in the aftermath of World War II. Due to improvements in medical care, medications, and surgical procedures, an increased number of soldiers were able to survive their injuries. This increase in survival rate, and the subsequent increase in wounded soldiers, led to the need for more effective, efficient, and focused rehabilitation efforts (Diller, 1990). The historical model of fragmented care provided by multiple health care disciplines would no longer meet the medical system's need for efficient rehabilitative services. According to Strasser, Uomoto, and Smits (2008), at that time in medical history, “innovative strategies were needed, and central to this response was the development of the interdisciplinary team to promote comprehensive and collaborative care” (p. 179). Howard Rusk, who is considered to be the father of comprehensive rehabilitation medicine, championed the interdisciplinary, collaborative care model in the post–World War II period (Butt & Caplan, 2010). Rusk's vision involved gathering professionals with expertise in a variety of rehabilitative domains into a one-stop-shopping treatment model with the hope that such integrated and collaborative care would be more efficient, cost-effective, and convenient to all those involved, most important the patients and their families (Diller, 1990).

This post–World War II movement led to the eventual emergence of essentially two team models: the multidisciplinary model and the interdisciplinary model of team functioning. Although both models are

predicated on the efforts of multiple disciplines, the degree of collaboration varies quantitatively and qualitatively. In multidisciplinary teamwork, clinicians function independently and information generated through assessment and treatment is typically communicated to an identified leader—often an attending physician or possibly a case manager. Information is not routinely shared or discussed, nor are treatment plans and goals established among team members. Individual providers are solely responsible for activities that are specific to their discipline (Butt & Caplan, 2010; Pecukonis, Doyle, & Bliss, 2008). Conversely, the interdisciplinary team model “places a premium on sharing assessment findings, development of communal goals, negotiation of responsibilities, and interdisciplinary collaboration while maintaining one’s unique discipline identity” (Butt & Caplan, 2010, p. 452). Regardless of the model employed, the clinical complexity of neuropsychological rehabilitation makes it crucial for treatment providers to be proficient in cross-specialty collaboration.

### **MAKING THE CASE FOR CROSS-SPECIALTY COLLABORATION**

The importance of effective collaboration among health care providers in neuropsychological rehabilitation cannot be, quite simply, overstated. Perhaps in no other medical arena is the need for collaboration among disciplines more beneficial to persons served. The field of neuropsychological rehabilitation focuses on individuals working to recover from impairments in their physical, emotional, social, and cognitive functioning as a result of acquired brain dysfunction—be it due to traumatic injury, cerebrovascular accident, tumor, or other acute/ chronic illness. The basic premise of comprehensive, neurological rehabilitation is that no clinician or professional working alone has the requisite skills to produce an outcome in which all affected domains (physical, emotional, social, and cognitive) can be adequately addressed. To ensure an optimal clinical and social outcome, comprehensive rehabilitation relies on the interworking of a large number of health care professionals representing numerous disciplines (Kemper & von Wild, 2001; Neumann et al., 2010; Wood, 2003). That optimal outcome requires a myriad of professionals (physical therapists, occupational therapists, physicians, nurses, speech-language pathologists, psychologists, social workers, dieticians, vocational counselors, chaplains, among others) working together in a concentrated and collaborative team effort. Such a team effort, be it multidisciplinary or interdisciplinary in nature, relies upon the effective cross-specialty collaborative abilities of all team members involved. Optimal neuropsychological rehabilitation is built upon the central and necessary

premise that the involved professionals can work together in an effective and efficient manner. An individual clinician working in isolation will lack certain knowledge, skills, and experiences that would be possessed by a team of multiple disciplines working together (Wood, 2003).

Effective cross-specialty collaboration involves extensive communication and joint decision making across disciplines in which knowledge and expertise are shared to promote an optimal outcome for a particular patient. According to Kemper and von Wild (2001), multiple disciplines working together to eliminate any redundancy, inconsistency, and contradictions in treatment, as well as to share the burden of care promotes efficiency in rehabilitation. The benefits of collaborative teamwork are based on the notion that working together will promote communication among treating professionals, allow for the sharing of knowledge across disciplines, promote a focused, goal-oriented approach toward patient care, and allow for more continuity in the course of rehabilitation (Wood, 2003).

There is no lack of empirical support for the importance of collaborative efforts among health care professionals working in a neurorehabilitation venue. Notably, the interdisciplinary team model has received the most attention and support from researchers (Halstead, 1976; Kemper & von Wild, 2001; Melvin, 1989). For example, Halstead (1976) reviewed the literature regarding team approaches in health care from 1955 to 1975. He concluded that collaborative strategies involving a coordinated team effort resulted in outcomes that were more effective relative to fragmented approaches in which disciplines acted independently of one another and relative to noncollaborative approaches, collaboration resulted in maintainable improvements in functional status across time. In another study, Neumann *et al.* (2010) concluded that clinical outcomes were superior for patients when they had been treated by an interdisciplinary team compared to settings where cross-specialty collaboration was not practiced. Finally, Sinclair, Lingard, and Mohabeer (2009) monitored the work of an interdisciplinary rehabilitation team over time and concluded that this collaborative model enabled the team to work synergistically to produce superior care than could have been provided by each clinician working alone.

As indicated, there is clear evidence for the benefit to patients for multiple disciplines working collaboratively in rehabilitation. Given the empirically supported evidence, it is the professional responsibility of all involved clinicians to ensure that effective collaboration occurs.

## **FACILITATING CROSS-SPECIALTY COLLABORATION**

## FACILITATING CROSS-DISCIPLINARY COLLABORATION

Regardless of professional identity, health care providers should ideally be adept at collaborating with other disciplines as they enter the work environment. Unfortunately, this is rarely the case, as it is unusual for clinical training centers to provide trainees with opportunities to develop interprofessional and collaborative competencies that include an appreciation for the education, training, and input of other health care disciplines. Instead, most training centers educate health care providers in isolation from other disciplines, creating profession-centric practitioners who have little appreciation for the educational background and professional development of their future team members. This may foster competition rather than collaboration among providers by minimizing an appreciation of what other disciplines bring to the table in the assessment and treatment of patients (Pecukonis, Doyle, & Bliss, 2008). Unfortunately, cross-specialty collaborative skills are often learned in the work environment and in a trial-and-error fashion, which may lead to practitioners continuing to operate from a discipline-centric belief system (Pecukonis, Doyle, & Bliss, 2008). Increasing educational efforts geared toward interdisciplinary activity and collaboration at the trainee level could significantly offset the adverse effects of educating health care professionals in isolation and would promote collaboration among professionals from the outset.

Given that collaboration occurs within an inherent social context, factors affecting group dynamics warrant consideration. The group dynamics of a treatment team should allow for open and honest dialogue to discuss potentially controversial issues involving the patient's care and/or the focus of rehabilitation. A team that collaborates effectively, even when conflict among team members is evident, must be able to respond to one another in a respectful and highly trusting manner. Working in a multidisciplinary or interdisciplinary team environment invariably sparks conflict and team members must be adept at managing that conflict by problem solving (negotiating, listening, deferring, etc.) in an effective means to allow the team to continue to function as a cohesive unit and promote the best outcome for the patient (Kemper & von Wild, 2001). Such rapport among team members comes slowly and after numerous positive interactions. As the saying goes "Rome was not built in a day," a treatment team able to deal with conflict adaptively requires facilitating conditions and extended time. In a review of the literature regarding team factors that contribute to effective cross-disciplinary collaboration, several themes were evident: (a) the importance of *consistency* and *stability* of team members (which allows for

trusting and respectful relationships to evolve among team members); (b) the importance of a *structure* for frequent communication among team members, be it formal (i.e., regularly scheduled team meetings) or informal (i.e., hallway conversations) (Sinclair, Lingard, & Mohabeer, 2009); and (c) the importance of consistency in *consensus decisions* (defined by the consideration of perspectives and expertise of all clinicians who will be expected to administer the treatment plans) (Sitley-Brown & Folen, 2005). The basic communication abilities that allow for an adaptive group dynamic include attentive listening, constructive criticism, and respectful interactions that demonstrate an appreciation for the personality and expertise of other members of the team. As the theme of “frequent communication” indicates, frequent contact among team members is critical. Such contact allows for rapport building, timely conversations that are anticipatory to problems, interactive problem solving, negotiation of goals, consensus development, and the translation of discipline-specific impressions into useful and applicable information for those individuals not trained in that discipline (Diller, 1990; Dunn, Sommer, & Gambina, 1992).

## BARRIERS TO AN EFFECTIVE COLLABORATIVE PROCESS

In a discussion regarding the social dynamics and social psychology involved in multidisciplinary or interdisciplinary teams, Mullins, Keller, and Chaney (1994) did not have a favorable view for the potential effectiveness of such team functioning and concluded that

such cohesive teams are largely mythical. What is seen more often are dysfunctional representations of the interdisciplinary and transdisciplinary team. Coordinated care is often compromised as team members lapse into a highly unstructured interdisciplinary model, or even multidisciplinary model. Individual team members may make independent decisions, or form coalitions and/or small collaborative teams (subsystems) that independently make treatment decisions. Such team fragmentation leads to poor communication with other members of the institutional staff . . . Why would such a scenario occur? First, we would argue that the interdisciplinary and transdisciplinary teams, although ostensibly representing ideal models, violate principles of both systems and social cognitive approaches to human behavior. (p. 166)

As most clinicians would likely agree, true cross-specialty collaboration is not necessarily an easy goal to attain. Even the most experienced health care professionals will at times struggle with the balance between professional autonomy and the team agenda. Conflicts between team demands and expectations of a professional discipline are common (Dunn et al., 1992). If team members strictly adhere to the demands of their individual profession, the team will become *discipline*-oriented and the comprehensive needs of the patient will

be lost. In order to be effective, each clinician must make a commitment toward a “team-focused” as opposed to a “discipline focused” approach. Team members must be comfortable with potentially competing agendas, which can occur whenever multiple disciplines are involved in a patient’s care and learn to integrate those agendas to form common goals. The potential for conflict always exists because of the differences in perspective and experiences. In situations of conflict, the use of professional deferral at strategic times can increase the likelihood of an optimal outcome and a win–win situation for all. Such deferral can convey an attitude of respect of other disciplines and a willingness to negotiate, thus improving the overall collaborative process (Sitley-Brown & Folen, 2005).

Working in an environment in which collaboration with other disciplines is expected does not necessarily suit all personality styles. Some professionals, because of personality characteristics, simply may not be adept at the necessary skills involved in collaboration. Such skills include the use of professional deferral, subjugation of interests, negotiation, assertiveness, and at times, professional humility. Well-seasoned clinicians may feel challenged, threatened, or insulted when, for example, asked for clarification of their clinical impression or a rationale for their recommendations, as often occurs. They may have similar emotions when presented with a request to adjust their entrenched work habits to be more accommodating to the needs of the team, patient, or family (Wood, 2003). Conversely, interdisciplinary collaboration is not for the faint of heart either. Clinicians with low self-esteem may be susceptible to misperceptions that other team members do not value their contributions, thus limiting their participation and input (Nair & Wade, 2003). Others may feel they cannot speak eloquently or convincingly regarding their clinical impressions and be reticent to participate. According to Wood (2003), when functioning in a collaborative, team-oriented approach, the clinician’s feelings of being less than fully competent and the resulting anxiety may be among the causes of some clinicians struggling in this context. Many health care professionals are uncomfortable or ill at ease when working in a team context due to limited preparation for working collaboratively with multiple disciplines (Wood, 2003). Yet others may be intimidated by team members who are more assertive, out spoken, or experienced. Undoubtedly, there will be some clinicians who attempt to overcome their feelings of inferiority with efforts to dominate the team in decision making or jockey for a position of authority with the patient or subset of the treatment team. As such, there is little role for personal (covert) agendas in a

collaborative process, and such agendas are likely to be counter productive. As stated by [Agich \(1982\)](#), “the ambition of an allied health professionals for prestige, while perhaps appropriate in itself, may be irrelevant, or worse, antagonistic to optimal health care” (p. 17). Working effectively in the context of other disciplines requires a balanced degree of self-esteem, respect for others, self-confidence, and personal humility ([Nair & Wade, 2003](#); [Wood, 2003](#)).

A very significant and growing barrier to cross-specialty collaborative practices is the constantly changing and increasingly constrictive nature of third-party reimbursements, which affects the entire health care system. Clinicians are being expected to do a lot more (i.e., increase productivity) with increasingly fewer resources (i.e., smaller staff, less funding, less space, less financial resources) and with more accountability (i.e., better patient and fiscal outcomes). Demand by health care administration for patient contact hours due to financial incentives coupled with clinicians competing for time with patients seriously limits opportunities for collaboration due to the time demands of needing to be elsewhere. As a result, some clinicians (particularly relatively expensive neuropsychologists) are reluctant to carve time into their schedules to participate in activities (treatment team meetings, family conferences, cotreatment) for which no reimbursement exists. It is not that neuropsychologists don’t appreciate the value of nonreimbursable activities; however, they are becoming increasingly sober to the realities of the rapidly changing fiscal demands of health care. In a recent posting to the American Psychological Association (APA), Division 22 (Rehabilitation Psychology) List Serve, [Callahan \(2010\)](#) predicted that third-party payers will become less willing to pay for expensive doctoral level providers when nearly identical (and equally effective) services can be provided by staff with master’s level training. It may prove to be more cost-effective for master’s level providers to be frontline collaborators, whereas doctoral level psychologists design treatment protocols; provide supervision/consultation to the master’s level clinician responsible for carrying out the treatment plan; and only see the most difficult, severe, and challenging patients. According to [Callahan \(2010\)](#), savvy psychologists will focus on what they can uniquely provide, such as complicated diagnostic assessment, protocol development, and management of outcome data.

Other issues that have been identified ([Nair & Wade, 2003](#); [Nijhuis et al., 2005](#); [Wertheimer et al., 2008](#)) as impeding the collaborative process include poor communication, lack of individual accountability, time constraints, poorly organized team meetings, lack of availability of team members, unavailability of

reports or records, productivity demands, limited physical proximity of all involved team members, and reduced lengths of patient stays in a hospital, facility, or program due to third-party payer demands (Diller, 1990). In addition, in many neurorehabilitation programs, although the psychologist is viewed as a core member of the interdisciplinary treatment team, he or she may function as a consultant with regard to reimbursement, physical location to the team and patient, and demands/ expectations of other professional work roles, such as serving other treatment teams or programs, and general productivity (Wertheimer et al., 2008). All these issues work in conjunction to further complicate the collaborative process in neurorehabilitation. Fair or not, it is generally the responsibility of the clinician to overcome these barriers, whether by altering his or her work habits or advocating for changes within the rehabilitation facility to promote effective cross-specialty collaboration.

## **BEING AN EFFECTIVE MEMBER OF THE REHABILITATION TEAM**

Identifying roles for the psychologist on a rehabilitation unit is a first step, but actually being effective as a team member and leader requires particular skills and behaviors. Some of these may necessitate changing the way psychologists practice independently in order to consider the broader needs of the team. Blair and Gorman (2003) identify 10 “survival tips” for neuropsychologists working in rehabilitation that address these skills. These tips can be roughly categorized as pertaining to (a) *intergration*: join the system, know your place, learn and demonstrate the art of professional deferral, and deal productively with administration; (b) *knowledge*: learn the language of physical rehabilitation and understand the basic coding and reimbursement issues; and (c) *professional identity*: become the glue in the team—translate, integrate, communicate, remain alert to ethical challenges, do not overtest, and do not forget that you are a neuropsychologist. Effective psychologists will evaluate how their own skills and behavior manifest these values.

### **Integration**

Psychologists in rehabilitation settings are faced with several unique challenges that may not be as applicable to other team members. As Blair and Gorman (2003) note, psychologists do not fit typical and historical roles on an inpatient rehabilitation unit, those being physician, therapist, or nurse. To be effective, neuropsychologists must have a clear sense of individual identity and purpose. Precisely because they do not fit into traditional roles, it is essential for them to



work to develop relationships with team members and communicate their work product in the most useful way to the team. Carving out a role that feels true to psychologists' training and philosophy may require a realignment of thinking about how to deliver services. A delicate balance must be found between the needs of the milieu in which psychologists are working and congruence with their own professional identities.

As [Blair and Gorman \(2003\)](#) note, neuropsychologists need to “join the system” of the team, learning to think in terms of collaboration, consensus, and mutual respect. The rehabilitation team system encompasses, among others, the interactions individuals have with one another, their treatment strategies and beliefs, and their own professional training and expertise. In the best of circumstances, this rich tapestry of individual contributions works to provide the best outcomes for the patients served. However, this requires everyone involved to develop interpersonal skills that may not come naturally. On the part of neuropsychologists, acquiring the skill of “professional deferral,” as explained by Blair and Gorman, may be particularly challenging. A common example is learning to accept the input of speech or occupational therapists on some cognitive issues and even deferring to their recommendations. This may be especially difficult for someone who has invested significant time in honing neuropsychological skills. It essentially boils down to the familiar refrain: Pick your battles. Learn to distinguish between those things that are essential and those that are not. Neuropsychologists offer unique benefits to the team, but attempting to direct every aspect of assessment and treatment will result in marginalization and discounting of their input. Professional deferral not only fosters goodwill, but also facilitates the effectiveness of the team approach, with ultimate benefits for the patient.

Although possibly overlooked as important to integration with the team, learning to deal with system administration is a talent worth neuropsychologists' time to develop. They must recognize that they are only one part of a larger structure and that all decisions must consider the big picture. This includes decisions that affect the rehabilitation team, such as where financial resources are invested (e.g., equipment, capital improvements) and how the rehabilitation program or structure aligns with corporate goals. Depending on the familiarity of administrators with the practice of psychology, savvy neuropsychologists may find it in their best interest to discuss and educate appropriate administrative personnel about concerns specific to psychology. Additionally, by virtue of their doctoral training, they are prepared to advocate for the needs of the rehabilitation

team generally. In order to present reasonable explanations and make appropriate, goal-congruent requests, neuropsychologists must learn the language and the concerns of administrators, including financial and staffing implications, corporate goals and strategies, and system issues.

## Knowledge

Rehabilitation psychologists and neuropsychologists have strong training in their respective specialties and are conversant with professional terminology. After all their training, however, they may yet have to obtain new knowledge if they choose to work in neurorehabilitation. For example, the basic language of rehabilitation, used in most inpatient facilities, is represented by the Functional Independence Measure (FIM). This widely accepted assessment tool is an 18-item rating scale that quantifies a patient's level of independence on tasks such as self-care, locomotion, communication, and social awareness and is applicable to all diagnoses within a medical rehabilitation population. It is in standard usage at all Commission on Accreditation of Rehabilitation Facilities (CARF)-certified rehabilitation facilities, as well as many others. Competence in understanding and scoring patients using the FIM will enhance psychologists' effectiveness as team members. Although neuropsychologists do not score many of the FIM items, they should still understand what the scores mean, what expected scores are, and how the scores of their facility compare to national data. Further, an appreciation of the demands created by the FIM allows neuropsychologists to work within parameters that may at times be challenging. Learning the meaning of terminology used by physical, occupational, and speech therapists will help neuropsychologists join the team and understand the details of the patient's status captured by their language.

Another body of knowledge that may be unfamiliar to neuropsychologists is that pertaining to the prospective payment system. Requirements of the Centers for Medicare and Medicaid Services (CMS) and other third-party payers dictate much of the work that goes on in inpatient and residential rehabilitation facilities, from the amount of therapy each day to the length of stay for any given patient. It can be challenging for neuropsychologists to learn and adapt to these demands (especially for those who may have worked independently in private practice); not being included in the "3 hour" rule for acute inpatient rehabilitation services can result in having to be creative in finding time to work with patients. Knowledge of and respect for these demands will help neuropsychologists avoid conflict with other team members over patient

availability and irritation with frustrating schedules. A thorough knowledge of how their own services are coded and billed, including appropriate use of health and behavior codes versus mental health codes, should be standard practice for psychologists working in rehabilitation settings.

### **Professional Identity**

Competent communication skills are, or should be, the forte of psychologists, and these abilities are an important contribution they can make in their roles as team members. Indeed, most of what psychologists do relies heavily on effective, accurate communication with patients, referral sources, and other clinicians. Unfortunately, communications with other professionals (or nonprofessionals) using “inside” language are not helpful and may serve to alienate psychologists from other team members. This is true for both verbal (e.g., team meetings, consultations) and written (e.g., chart notes, formal reports) communications. Neuropsychologists, in particular, are perhaps famous for their use of professional jargon. They have a language of their own, even telling jokes that rely heavily on private jargon. To be useful team members, whose input is valued and sought, jargon must be replaced with clear behavioral descriptions that team members, patients, and families can understand. There is no need to prove competence or education by the use of specialized terminology when simpler language will adequately communicate the message. Neuropsychologists who cannot or will not communicate in such a way will be disappointed to find that their input is not respected.

In addition to plain, understandable language, written reports must be concise. Reports of 40 typed pages (not unheard of) for outpatient evaluations or even multiple pages for inpatient assessments tend to be ignored at worst or skimmed at best. Although they may “like to hear themselves write,” neuropsychologists must overcome the graduate school mindset that more is better and get to the point by being concise and definitive. Although background information is useful in reporting on the patient’s cognitive, emotional, and social status, team members are eager for guidance and direction for immediate issues and will appreciate recommendations that are specifically applicable to the current rehabilitation process. Concise reports with predictable, reliable information presented in a consistent format will be appreciated by colleagues and will increase the value of neuropsychologists to other team members.

Even the most accessible, well-communicated information will be of little use if it is not provided in a timely manner. With increasingly short rehabilitation

stays, initial evaluations and reports completed days after admission may be of little use in guiding team members in their interactions with and treatment of patients. Whether doing an intake assessment, cognitive evaluation, or mood/coping assessment, timeliness is paramount. Teams count on neuropsychologists to provide information and recommendations they can quickly incorporate into their activities with the patient. For example, providing quick guidance on managing behavior problems is vital, as such problems can otherwise become entrenched. Similarly, timeliness is appreciated with ongoing psychological interventions, as the input of neuropsychologists regarding the patient's emotional status can be helpful to other staff members working with the patient.

Despite their best efforts, neuropsychologists may encounter some communication challenges not entirely within their sphere of control. Team members frequently coordinate and plan with one another in informal meetings and consultations. These informal consultations typically address "real-time" issues germane to patient progress, behavior, or medical status and are often of equal or greater importance than formal team meetings. Neuropsychologists, especially those with duties in addition to their services on the rehabilitation team, often are housed in locations not easily accessible to the team. Lack of proximity reduces the contribution they can make and limits their awareness of acute patient issues (Wertheimer et al., 2008). Because proximity issues are frequently determined by forces outside the control of neuropsychologists, efforts to increase accessibility to the treatment team become the responsibility of neuropsychologists. Paging systems are perhaps the most common method of contact when neuropsychologists are located off-unit but within the same medical system as the rehabilitation unit. Even these have their limitations, as it may not always be possible to respond to pages within an ideal time frame. Cell phones have also been used, but with the same limitations. Where neuropsychologists are located off campus and/or outside of the facility, the challenges of accessibility increase. Those who work in a consulting role, with scheduled days and/or times to provide services to the rehabilitation team, have other responsibilities that cannot readily be postponed to respond to acute needs of the team or patients. It may simply be a fact of life that neuropsychologists cannot always provide optimal real-time service. Thus, it is helpful to establish protocols addressing the most common problems likely to be encountered that can be implemented by other members of the rehabilitation team until the neuropsychologist can respond personally. For example, neuropsychologists can

create flow charts of behavioral interventions for those behavior problems most often exhibited by their populations. These should be clear and simple and can be communicated in a number of ways, depending in large measure on the physical facilities and existing communication methods. For example, a written generic plan for patients who are agitated might include checking to see whether the patient is hungry, cold, needs to use the bathroom, or is overstimulated; these plans should be located in areas easily accessible to both therapy and nursing staff. Examples might include placing them in nursing Kardex records, on patient wheelchairs, in patient rooms, and in common areas (e.g., therapy rooms, dining rooms). A side benefit of placing them in visible areas is the opportunity for family members to learn how to intervene with challenging behaviors before the patient is discharged. Similar generic plans can be developed for mood/coping issues. Such plans may delineate criteria for rating the urgency of a needed response from psychologists and can be used to help staff communicate accurate impressions.

Despite the potential challenges neuropsychologists face in communication with the larger team, their communication skills are an invaluable asset. [Blair and Gorman \(2003\)](#) encourage remembering the “psychologist” in *neuropsychologist*. In other words, clinical skills that are the foundation of all psychological work should not be forgotten. Working in a rehabilitation setting is the “best of both worlds,” as both the *neuro* and the *psychologist* aspects of training can be integrated. For example, knowledge of neuroanatomy, neuropathology, and the particulars of typical diagnoses seen on a rehabilitation unit can be integrated with knowledge of psychological reactions to illness/disability, family dynamics, and effective coping strategies. [Blair and Gorman \(2003\)](#) suggest that “although other clinicians in this setting may assess and address cognitive functions, you are likely the primary (if not the only) professional involved with the patient/family who can assist with coping and adjustment.”

Thus, effectiveness as a neuropsychologist on a rehabilitation team requires use of unique existing skills and the development of what may be new and unfamiliar abilities. Most important, neuropsychologists in these settings need to recognize the value of working within a team and be willing to adapt to a team system. This may include relaxing boundaries on preconceived notions of their own “territory,” increasing their body of knowledge to include processes and policies specific to rehabilitation programs, being sensitive to issues of communication with other team members, and taking the lead in those areas

benefitting from their skill set. The need for neuropsychologists on a rehabilitation team should be undisputed; their value to the team is dependent on their own efforts.

## **REDEFINING THE REHABILITATION TEAM**

The various therapists, physicians, nurses, dietary experts, social workers, and neuropsychologists form the heart of the rehabilitation team and, with few exceptions, are the standard in neurorehabilitation facilities. Patients and families have not traditionally been considered a part of the team. However, research has demonstrated that including patients in their health care reduces costs, improves outcomes, and increases compliance with treatment plans (Abreu, Zhang, Seale, Primeau, & Jones, 2002). Further, Sherer *et al.* (2007) investigated the factors associated with therapeutic alliance between therapists and patients/families in a postacute brain injury program. They found that the strength of therapeutic alliance was predictive of discharge functioning. Additionally, poor alliance was associated with poor patient participation in therapy. Evans, Sherer, Nakase-Richardson, Mani, and Irby (2008) subsequently found that similar patients receiving treatment from therapists who underwent training in developing therapeutic alliance with patients and families had better functional status at discharge than a control group.

Although traditional rehabilitation teams may give lip service to patients and families being a part of the team, this is not always the case in actual practice. Instead, their input may be minimized or ignored. There are numerous reasons for this, and obstacles to inclusion of patients and families are plentiful, including the structure of team meetings, cognitive impairments of patients, hidden agendas of team members, and preexisting family dynamics. For example, team meetings are often a group discussion of all patients, making the practical aspects of having patients come and go for their own discussion a deterrent and, in effect, disempowering the patient. In attempting to make such meetings more accessible to patients and families, time, in and of itself, becomes an additional obstacle (Baxter & Brumfitt, 2008). Team meetings are sometimes held at times that would be inconvenient or impossible for family members to attend. Patients may be in therapy or eating a meal during meeting times. Even finding time when all team members can attend can be difficult; lengthening team meetings by including patients and families requires a change in priorities and flexibility in assignment of clinical responsibilities. In the end, the patient's involvement may be limited to a brief, stand-up visit with team members

informing him of the outcome of the team meeting.

Adding to the challenges of meeting times and structure when attempting to increase inclusion of the patient and/or family, is that some team members may have their own agendas for goals and outcomes, which are not always congruent with those of the patient and his or her family. Such agendas are not always readily evident, and it takes careful listening and interpretation to identify how hidden staff agendas may be interfering with communication with and inclusion of patients and family members. [Sitley-Brown and Folen \(2005\)](#) point out that only when teams make decisions by consensus, they can attain the consistency required for success. They further add that consensus decisions can only be reached when incorporating the values, perspectives, and expertise of all involved and must include the ability to solve problems, negotiate, and compromise. Patients and family members are dealing with significant stressors and may not always voice their concerns or even know how to. They may feel they have no right to disagree with therapists or other professionals. Neuropsychologists, by virtue of their training and their unique role on the team, may be best equipped to give families and patients permission to offer their own goals and concerns, while listening for the presence of competing agendas within the team that may impede the effectiveness of team functioning. They can then help guide the team, including patients and families, to consensus.

Including families and patients in an active role as members of the rehabilitation team also brings to the forefront any preexisting family dynamics. As anyone who has worked in such environments can attest, these dynamics are not always healthy or adaptive. The added stresses of the patient's condition exacerbate any tensions or conflict within the family, which are then played out for all to see, including therapists and other team members. Patients sometimes find themselves in the middle of a family battle. As difficult as these situations can be for all involved, these are the times that inclusion of the family and patient in the team is even more important. The team and neuropsychologists particularly can be the buffer and the voice of reason in family conflict. However, team members, even psychologists, need to remember that they cannot solve all family problems and should focus their efforts on developing agreement and cooperation in the rehabilitation process.

Finally, the very nature of neurorehabilitation creates some unique obstacles when attempting to involve patients actively as a part of the team. Individuals with cognitive impairment, who are commonly found among the rehabilitation population, can be more challenging to involve in their own care.

Nevertheless, the Commission on Accreditation of Rehabilitation Facilities, representing the gold standard in rehabilitation facilities/programs, requires communication with patients, in the language they can understand (See [Commission on Accreditation of Rehabilitation Facilities, 2010](#), section K). Problems of receptive and expressive language, limited insight, poor memory, and impulse control can each negatively impact efforts to view the patient as a member of the team. Although rehabilitation teams are accustomed to and presumably comfortable with persons with cognitive impairment, the difficulty of dealing with impairment may make it is easy for team members to ignore and/or minimize patients' input and feelings about team processes, decisions, and recommendations. Again, neuropsychologists are well positioned to provide leadership in integrating persons with cognitive impairment into the team. [Hobson \(1999\)](#) suggests several guidelines to help professionals work in a client-centered model with persons with cognitive impairment: (a) enhance the assessment process to determine whether the patient is capable of making rational decisions about his or her care (go beyond the typical cognitive evaluation and assess practical comprehension and decision making that apply to the patient's current status); (b) simplify the involvement of the patient to his or her capacity (express information in a user-friendly way that the patient can understand); (c) be an advocate for the patient if his cognitive status prevents him from fully participating (try to consider and express the patient's point of view insofar as you are able to determine it); and (d) when available, engage a surrogate who is familiar with the patient's wishes and can assist in advocating for him or her (this may be done in cooperation with other professionals on the team, most particularly social workers).

When confronting these challenges in process and personality, neuropsychologists are ideally positioned to take the lead in helping teams redefine themselves by more actively including patients and family members. Assuming they have made themselves an accepted member of the team who respects other team members and have been recognized as possessing skills in interpersonal relations, neuropsychologists can guide team process and develop a plan for greater inclusion of patients and their families. Team training in consensus and collaboration, as well as the skills, rationale, and process of increasing inclusion of patients and families in treatment planning and goal setting can be developed and coordinated by neuropsychologists working in tandem with other professionals and administration. Although neuropsychologists may advocate for a more inclusive team and present



suggestions for practical implementation, it will take the cooperation of all team members to make a commitment to broadening the inclusion of patients and families in team decisions. Such changes take time, and neuropsychologists should be cautious, in their eagerness, about falling into the trap of attempting to impose their will on other team members.

## SUMMARY

Since the establishment of interdisciplinary care in neurorehabilitation following WWII, the importance of collaborative care in this venue has gained increasingly widespread support for its use and implementation. Collaboration among health care disciplines in rehabilitation is considered to be a standard of care for comprehensive patient services. Special skills are required of the professional working within a collaborative context, which differ from those required by the clinician practicing independently. Psychologists, specifically neuropsychologists, by virtue of training and experience bring a set of skills to the rehabilitation team that are particularly valuable to the collaborative process—that is, if the neuropsychologist can adapt to a team-oriented approach to patient care. This may include relaxing territorial boundaries and adjusting an entrenched work style to accommodate the overall needs of the team. Assuming that neuropsychologists are particularly adept in interpersonal and communication skills, they would be able to assist the team in problem solving, negotiation, as well as working to include the patient and family as integral members of the team. As systems of reimbursement in health care become more constricted, true interdisciplinary care may become increasingly challenging. Regardless, cross-specialty collaboration is a worthwhile endeavor in the pursuit of high-quality patient care.

## REFERENCES

- Abreu, B. C., Zhang, L., Seale, G., Primeau, L., & Jones, J. S. (2002). Interdisciplinary meetings: Investigating the collaboration between persons with brain injury and treatment teams. *Brain Injury, 16*(8), 691–704.
- Agich, G. J. (1982). A historical view of health care teams. In G. J. Agich (Ed.), *Responsibility in health care* (pp. 3–21). Dordrecht, Holland: Reidel Publishing Company.
- Baxter, S. K., & Brumfitt, S. M. (2008). Benefits and losses: A qualitative study exploring healthcare staff perceptions of teamworking. *Quality & Safety in Health Care, 17*(2), 127–130.
- Blair, K. L., & Gorman, P. W. (2003). Survival tips for the neuropsychologist in an inpatient rehabilitation setting. *Rehabilitation Psychology, 48*, 310–313.
- Butt, L., & Caplan, B. (2010). The rehabilitation team. In R. G. Frank, M. Rosenthal, & B. Caplan, (Eds.), *Handbook of rehabilitation psychology* (2nd ed., pp. 451–457). Washington, DC: American Psychological Association.

- Callahan, C. D. (2010, October 28). *Re: What is happening to psychologists in inpatient rehabs?* Message posted to [Rehabpsych@lists.acs.ohio-state.edu](mailto:Rehabpsych@lists.acs.ohio-state.edu).
- Diller, L. (1990). Fostering the interdisciplinary team, fostering research in a society in transition. *Archives of Physical Medicine and Rehabilitation*, 71(5), 275–278.
- Dunn, M., Sommer, N., & Gambina, H. (1992). A practical guide to team functioning in spinal cord injury rehabilitation. In C. P. Zejdlik (Ed.), *Management of spinal cord injury* (2nd ed., pp. 229–239). Boston, MA: Jones & Barlett.
- Evans, C. C., Sherer, M., Nakase-Richardson, R., Mani, T., & Irby, J. W. (2008). Evaluation of an interdisciplinary team intervention to improve therapeutic alliance in postacute brain injury rehabilitation. *Journal of Head Trauma Rehabilitation*, 23(5), 329–338.
- Halstead, L. S. (1976). Team care in chronic illness: A critical review of the literature of the past 25 years. *Archives of Physical Medicine and Rehabilitation*, 57(11), 507–511.
- Hobson, S. J. G. (1999). Using a client-centered approach with persons with cognitive impairment. In Sumison (Ed.), *Client-centered practice in occupational therapy* (pp. 51–60). London, UK: Churchill Livingstone.
- Kemper, B., & von Wild, K. (2001). Requirements of team effectiveness in neurosurgical rehabilitation. *Acta Neurochir Supplement*, 79, 37–39.
- Commission on Accreditation of Rehabilitation Facilities. (2010). *Medical rehabilitation standards manual*. Tucson, AZ: Author, pp. 76–81.
- Melvin, J. L. (1989). Status report on interdisciplinary medical rehabilitation. *Archives of Physical Medicine and Rehabilitation*, 70(4), 273–276.
- Mullins, L. L., Keller, J. R., & Chaney, L. M. (1994). A systems and social cognitive approach to team functioning in physical rehabilitation settings. *Rehabilitation Psychology*, 39, 161–178.
- Nair, K. P., & Wade, D. T. (2003). Satisfaction of members of interdisciplinary rehabilitation teams with goal planning meetings. *Archives of Physical Medicine and Rehabilitation*, 84(11), 1710–1713.
- Neumann, V., Gutenbrunner, C., Fialka-Moser, V., Christodoulou, N., Varela, E., Giustini, A., & Delarque, A. (2010). Interdisciplinary team working in physical and rehabilitation medicine. *Journal of Rehabilitation Medicine*, 42(1), 4–8.
- Nijhuis, B. J., Reinders-Messelink, H. A., de Blécourt, A. C., Olijve, W. G., Groothoff, J. W., Nakken, H., & Postema, K. (2007). A review of salient elements defining team collaboration in paediatric rehabilitation. *Clinical Rehabilitation*, 21(3), 195–211.
- Pecukonis, E., Doyle, O., & Bliss, D. L. (2008). Reducing barriers to interprofessional training: Promoting interprofessional cultural competence. *Journal of Interprofessional Care*, 22(4), 417–428.
- Sherer, M., Evans, C. C., Leverenz, J., Stouter, J., Irby, J. W., Lee, J. E., & Yablon, S. A. (2007). Therapeutic alliance in postacute brain injury rehabilitation: Predictors of strength of alliance and impact of alliance on outcome. *Brain Injury*, 21(7), 663–672.
- Sinclair, L. B., Lingard, L. A., & Mohabeer, R. N. (2009). What's so great about rehabilitation teams? An ethnographic study of interprofessional collaboration in a rehabilitation unit. *Archives of Physical Medicine and Rehabilitation*, 90(7), 1196–1201.
- Sitley-Brown, K., & Folen, R. A. (2005). Psychologists as leaders of multidisciplinary chronic pain management teams: A model for health care delivery. *Professional Psychology: Research and Practice*, 36, 587–594.
- Strasser, D. C., Uomoto, J. M., & Smits, S. J. (2008). The interdisciplinary team and polytrauma rehabilitation: Prescription for partnership. *Archives of Physical Medicine and Rehabilitation*, 89(1), 179–181.
- Wertheimer, J. C., Roebuck-Spencer, T. M., Constantinidou, F., Turkstra, L., Pavol, M., & Paul, D. (2008). Collaboration between neuropsychologists and speech-language pathologists in rehabilitation settings. *Journal of Head Trauma Rehabilitation*, 23(5), 273–285.
- Wood, L. (2003). The rehabilitation team. In R. J. Greenwood, M. P. Barnes, T. M. McMillan, & C. D. Ward (Eds.), *Handbook of neurological rehabilitation* (2nd ed., pp. 41–50). New York, NY:

Psychology Press.

## Rehabilitative Psychopharmacology

*Anya Mazur-Mosiewicz and Chad A. Noggle*

Throughout the centuries, understanding of acquired brain injuries and their consequences has been shaped by cultural beliefs, religion, philosophy and, later, science. Although the early understanding of the brain-behavior relationship dominated throughout the centuries, it was the scientific approach that allowed complex theoretical frameworks to be built out of the relationship between brain injury and human behavior. It also led the modern theories regarding both etiology and treatment for cognitive-behavioral changes that often follow an injury to the brain. With the evolution of the scientific approach and controlled studies, medical sciences strongly popularized biological models of brain trauma. As such, it is now generally accepted that morphological changes to the central nervous system may account for mental and cognitive changes in an individual. Building on this idea, pharmacological treatment options for cognitive deficits occurring after a brain injury rely on the assumption that cognitive deficits are related mainly to a disruption in the brain neurotransmitter systems. Pharmacological treatment, therefore, aims at modulation of the neurotransmitter system function and subsequent symptom relief. Yet, it is important to recognize that psychopharmacological treatment is only one of several treatment approaches recommended for posttraumatic cognitive impairment. Additionally, for each patient, rehabilitative treatment should be integrative and emphasize the multidisciplinary approach and collaboration of specialists from different disciplines.

In the following chapter, we will discuss the principles of rehabilitation through pharmacological interventions. The majority of this discussion will

focus on pharmacological rehabilitation options following traumatic brain injury (TBI).

## **IMPACT OF BRAIN TRAUMA ON COGNITIVE PROCESSES**

Patients with TBI may experience a number of cognitive symptoms. Among the most commonly reported symptoms are disturbances related to arousal, sleep, processing speed, memory, attention, language comprehension and/or expression, and executive functions (Arciniegas, Held, & Wagner, 2002; McAllister, 1992; Waxweiler, Thurman, Sniezek, Sosin, & O'Neil, 1995). These symptoms may occur as the result of direct insult to the elements of cortical, subcortical, and/or brainstem networks that are necessary for cognitive performance. Additionally, head trauma may result in a disruption of the axonal connections and neurochemical projections of these networks, which is likely to cause further changes in cognitive processes (Arciniegas & Silver, 2006). The most persistent and severe cognitive problems tend to result from focal brain injuries. As opposed to more diffuse damage, focal brain injury is often related to specific, and relatively well-researched, cognitive problems.

The extent of cognitive problems following TBI tends to intensify with the severity of disruptions to the axonal connections. In general, axonal stretching and strain, which may occur during TBI, result in abnormally elevated production of the brain's neurochemicals. Excessive levels of some neurotransmitters, including glutamate and acetylcholine, carry some neurotoxic effects. In TBI, these effects are the most pronounced in the areas that are related to the activity of neurotransmitters: forebrain, hippocampus, striatum, and frontal areas (Arciniegas & Silver, 2006). Neurotoxicity of the neurotransmitters in these regions is likely to result in the disruption of cognitive functions that are dependent on these particular sites and which include arousal regulation, attention and concentration, memory functions, and executive processes. Although increased levels of neurotransmitters tend to abate, this reduction is often succeeded by long-term cognitive deficits.

## **PRINCIPLES BEHIND PHARMACOLOGICAL TREATMENT**

Before deciding on the most optimal pharmacological treatment to address TBI-associated cognitive problems, several principles of pharmacological treatment have to be considered. First of all, a pharmacological approach has to be preceded by a detailed review of the individual's medical, psychological, and social history as well as an in-depth diagnostic examination. The clinical

examination can be challenging due to an overlapping of cognitive symptoms and interference of other, post-TBI problems such as headaches, severe pain, sleep disturbance, excessive fatigue, mood disturbance, anxiety, and physical complaints. As such, it is suggested that evaluation and treatment of noncognitive symptoms should be, if possible, a prerequisite to cognitive pharmacological treatment ([Arciniegas & Silver, 2006](#)). In addition, systematic assessment should be conducted using objective neuropsychological assessment tools, with simple bedside examinations used only in those instances in which a patient is not fit for a standard battery of tests.

Pharmacological treatment to address posttraumatic cognitive impairment is more complicated than in many other diseases due to the heterogeneity of the TBI patient population and relatively low predictability of the TBI pathological outcomes ([Maas, Marmarou, Murray, & Steyerberg, 2004](#)). Pharmacological treatment can affect cognitive, physiological, and behavioral features of patients. Generally, selection of pharmacological agents, and their final effect on patients, depends on the interaction among several factors, including the severity and type of anatomical and morphological damage, alteration to the brain neurochemistry, and secondary outcomes of TBI. Other factors that should be considered as impacting the possible rehabilitative outcome are time since the initial damage, medication, and/or other psychoactive substance taken at the time of the injury, pre-existing conditions (e.g., some medications can lower the threshold for seizures and should not be recommended in patients diagnosed with seizures or epilepsy), and concurrent medication ([Tenovuo, 2006](#)). Although these variables should guide both the choice and administration of pharmacological agents, the final outcome is not always predictable in TBI. It is generally acknowledged that pharmacological treatment can be useful in cognitive impairment after TBI, but negative or nonexistent outcomes are not uncommon. This variability of outcome is often attributed to the altered sensitivity of the injured brain. An injured brain may become more sensitive to the psychoactive agents; or contrastingly, it may become resistant to pharmacological treatment ([Tenovuo, 2006](#)). Considering this fact, administration of psychoactive medication for patients with TBI should adhere to specific principles that reflect the need for individualized treatment monitoring and careful adjustment of the drugs. Silver, Arciniegas, and Yudofsky (2005) suggest adhering to several rules in administering medication for TBI patients. First, with the initiation of the treatment, the medication doses should be low and increased at a slower rate than in patients without a TBI. Second, acknowledging that the TBI patients may

be more sensitive to side effects of psychopharmacological treatment than other patients, some cognitive and neurological symptoms may require standard treatment dosages. Third, there is a need for continuous monitoring and reassessment, with dose reduction and/or discontinuation as ongoing options for consideration as the target symptoms remit. In other words, with continued recovery, the dosage of medication should be adjusted appropriately or medication should be discontinued. Fourth, as TBI patients may require multiple medications, monitoring the drug–drug interaction is crucial during the treatment. Fifth, partial response to treatment may require augmenting treatment with a second agent that represents a different mechanism of action. In general, augmenting one agent with another substance is preferred over switching to an agent with the same pharmacological profile as the original medication. Finally, if neuropsychiatric symptoms that are a focus of the treatment increase after medication administration, the dosage of the medication should be lowered. If the symptoms further intensify or persist, the medication should be discontinued. In addition, the effectiveness and possible side effects of the pharmacological treatment have to be closely monitored and the dosage adjusted depending on the patient’s response and presenting needs.

## **PHARMACOLOGICAL AGENTS AND THEIR FUNCTION**

There is an extensive body of research showing the effectiveness of specific groups of pharmacological agents in recovery from posttraumatic cognitive impairments. In general, deregulation of arousal and slow processing speed seem to be best addressed by catecholaminergic augmentation. Improvement of executive and attention functions are supported by both catecholaminergic and cholinergic augmentation. Finally, memory deficits are usually approached by supporting cholinergic systems (Arciniegas & Silver, 2006). In addition to pharmacological approach, patients with posttraumatic cognitive impairment usually benefit from other nonpharmacologic treatments. In fact, nonpharmacologic treatment options may not be effective on their own, in which case a combined pharmacological–cognitive rehabilitation approach is recommended. Of course, these are only general recommendations, and their specifics largely depend on the overall picture of the posttraumatic condition of the patient. Yet, these suggestions are also supported by a number of studies, a short review of which will follow next.

As eluded to, there are two main routes of medication treatment with the TBI population, which include augmentation of cerebral catecholaminergic and

cholinergic functions. Medications that address these neurotransmitter systems can have multiple functional outcomes. As such, the following discussion is intended to focus on the major groups of medications, and how these groups may impact the recovery and rehabilitation of cognitive impairment following TBI.

## Catecholamine Systems

Among the major neurotransmitters that modulate functioning of the brain are two catecholamines: dopamine and norepinephrine. As is the case with many other neurotransmitters, the catecholamines interact with multiple receptors and impact various networks of the brain (Arciniegas & Silver, 2006). For optimal brain function, there has to be appropriate balance in the neurotransmitters as an excess of the catecholamines can result in negative cognitive outcome. The increased levels of catecholamines and subsequent heightened metabolism are the effect of TBI-induced stimulation of the brain's sympathoadrenomedullary axis and serotonergic system. The dramatic increase in catecholamines often follows the initial injury and increases the risk of further complications. The acute change is mostly relevant to regional concentrations of brain catecholamines, which may persist for prolonged periods postinjury and are indicative of poor recovery prognosis (Donnemiller et al., 2000; Hamill, Woolf, McDonald, Lee, & Kelly, 1987; Woolf, Hamill, Lee, Cox, & McDonald, 1987). Considering their involvement in altered brain neurochemistry following TBI, it is not surprising that catecholamines are the focus of pharmacological therapy. Numerous studies show that pharmacologic rehabilitation that centers on catecholamine augmentation agents yields positive results, with a major impact on cognition, arousal, attention, and a number of other mental processes.

### Bromocriptine

Bromocriptine (Parlodel) is the central nervous system (CNS) stimulant and cognitive enhancer that appears to act directly on postsynaptic dopamine type 2 (D2) receptors. In TBI, bromocriptine is used to help reduce vegetative state and apathy, as well as improve cognitive initiation, attention, and processing speed impairments (Ben Smail, Samuel, Rouy-Thenaisy, Régnault, & Azouvi, 2006; Passler & Riggs, 2001; Powell, al-Adawi, Morgan, & Greenwood, 1996). It has been suggested as useful in recovery from communication deficits, increasing performance on executive functioning tasks, and augmenting learning and memory (Glenn & Wroblewski, 2005; Warden et al., 2006; Zafonte, Lexell, & Cullen, 2001). Additionally, bromocriptine has anticonvulsant properties;



therefore, it is suggested to be safe for individuals at risk of posttraumatic seizures ([Arciniegas & Silver, 2006](#)). When administered in low doses, it is believed to act as a presynaptic D2 agonist. In other words, in small doses, bromocriptine reduces dopaminergic release and decreases its function in dopaminergically mediated networks of the brain. With larger doses, bromocriptine seems to increase the function of cerebral dopaminergic systems ([Silver et al., 2005](#)). This augmentation of dopaminergic systems is the main focus of the cognitive therapy in TBI, and it is shown to be effective in severe to mild cognitive impairment.

In addition, bromocriptine has been shown to yield positive effects on patients in posttraumatic vegetative state as suggested by Passler and Riggs (2001), who used five participants, all of whom emerged from their vegetative status and regained somewhat functional levels of their abilities. Yet, it is important to mention that the recovery of physical and cognitive functioning in Passler and Riggs's (2001) study was supported not only by bromocriptine but also by other extensive rehabilitation treatments. Subsequent literature suggests that bromocriptine supports general recovery of several cognitive functions, including orientation, sustained attention, processing speed, and functional independence ([Ben Smaïl et al., 2006](#)). Despite reports of positive results on some aspects of cognitive performance, there are also reports of its adverse effects. [Whyte et al. \(2008\)](#) reported on the results of a 6-week-long double-blind, placebo-controlled, crossover study. According to the findings, bromocriptine had little effect on cognition, including attention. Among side effects of bromocriptine, the most commonly listed are nausea, vomiting, headache, and postural hypotension. Less frequently reported side effects include nasal congestion, digital vasospasm, and the CNS effects such as psychosis, hallucinations, nightmares, or insomnia. Its hypoglycemic effect may complicate diabetes treatment, and monitoring cardiac patients for arrhythmias is necessary during the treatment duration.

## Amantadine

Amantadine (Symmetrel, Symadine) is a nonstimulant dopamine enhancer. It appears to increase the release of dopamine and, at the same time, decrease dopamine reuptake, enhance dopamine receptors sensitivity, and stimulate dopamine receptors. Not knowing its exact mechanism of action, it has been proposed that the clinical efficacy of amantadine in enhancing cognitive performance after TBI is related to the increase of dopamine neurotransmission

(Meythaler, Brunner, Johnson, & Novack, 2002; Writer & Schillerstrom, 2009). In the clinical application with TBI patients, amantadine appears to remediate posttraumatic cognitive impairments and frontally mediated behavioral impairments (Chandler, Barnhill, & Gualtieri, 1988; Gualtieri, Chandler, Coons, & Brown, 1989; Kraus & Maki, 1997). Commonly used in patients with chronic TBI, amantadine has been often scrutinized by clinical studies (Beers, Skold, Dixon, & Adelson, 2005). As a result, amantadine has been reported to improve a number of symptoms, which include arousal, orientation, initiation, fatigue, attention, rigidity, agitation, purposeful movement, attention, concentration, goal-directed behavior, sequencing skills, and processing time (Beers et al., 2005; Kraus et al., 2005; Leone & Polsonetti, 2005; Napolitano, Elovic, & Qureshi, 2005; Writer & Schillerstrom, 2009; Zafonte et al., 2001).

Amantadine seems to have high clinical utility in both adult and pediatric populations. In a study that included subacute to remote injury, mild to severe injury, and inpatient and outpatient settings, Kraus et al. (2005) showed high effectiveness of nonstimulant dopamine enhancement in improving executive functions. Efficacy of amantadine in cognitive disorders was also demonstrated in a double-blind, placebo-controlled, crossover study design by Meythaler et al. (2002). Their results suggested that amantadine facilitated a consistent trend toward a more rapid functional improvement, if the patients started taking amantadine during the subacute postinjury period. They observed improvements in *Mini-Mental Status Examination* scores, *Disability Rating Scale* scores, *Glasgow Outcome Scale* scores, and in the cognitive domain score of the *Functional Independence Measure* (FIM).

### L-Dopa and Carbidopa

L-dopa (levodopa) is considered a nonstimulant dopamine enhancer; it serves as a precursor dopamine, which in the net result enhances dopamine neurotransmission in the CNS. It is usually coadministered with carbidopa to inhibit peripheral metabolism of L-dopa. Administered to individuals with posttraumatic vegetative state, or a state of minimal consciousness, L-dopa has been reported to improve and facilitate the rehabilitative process in acute TBI (Waldron-Perrine, Hanks, & Perrine, 2008). Literature also reports positive effects of L-dopa/carbidopa on alertness, concentration, fatigue, hypomania, oral communication, and memory as well as a range of motor deficits (Lal, Merbtiz, & Grip, 1988; Liepert, 2008). Yet, the reports on the effectiveness of L-dopa/carbidopa are often based on specific case studies and may be not

generalizable over all TBI patients ([Matsuda, Komatsu, Yanaka, & Matsumura, 2005](#)). L-dopa/carbidopa treatment has been associated with a number of dopaminergic side effects such as dyskinesias, anxiety, hallucinations, paranoia, or psychosis.

## Dopamine and Norepinephrine Enhancers

### Methylphenidate

Methylphenidate is a widely studied stimulant medication. There seems to be general consensus that the agent supports functional recovery following a head trauma ([Arciniegas & Silver, 2006](#); [Gualtieri & Evans, 1988](#); [Kaelin, Cifu, & Matthies, 1996](#); [Karaküçük, Pasaoglu, Pasaoglu, & Oktem, 1997](#)). Its mechanism of action focuses on the presynaptic neuron by promoting the release of dopamine and norepinephrine from presynaptic vesicles. At the same time, it blocks the neurotransmitters' reuptake into the presynaptic cell. Amplifying the dopaminergic systems in the CNS, methylphenidate is proposed to improve cognitive functions that are dependent upon dopamine, such as executive processes ([Jorge & Robinson, 2003](#)). The most positive outcome areas in methylphenidate therapy are aspects of attention, memory, processing speed, arousal, and sensory-perceptual-motor skills as well as global cognition ([Gualtieri & Evans, 1988](#); [Lee et al., 2005](#); [Whyte et al., 1997](#); [Whyte, Hart, Vaccaro, et al., 2004](#)). In post-TBI treatment, methylphenidate has been employed with heterogeneous populations, which include both pediatric and adult groups, as well as acute, subacute, and long-term rehabilitation conditions ([Writer & Schillerstrom, 2009](#)).

Methylphenidate is one of the best-researched psychostimulants in respect to posttraumatic cognitive impairment, with both double-blind, placebo controlled case studies supporting its positive results. A closer look at the most recent of these projects, which engage functional neuroimaging techniques, permits an understanding of the cognitive mechanism of action for methylphenidate. In double-blind controlled situations, it seems that methylphenidate's effects may be related to changes in the brain activation. In particular, [Newsome et al. \(2009\)](#) provided some evidence that methylphenidate may increase processing efficiency associated with cognitive control during working memory tasks in patients with TBI. In their project, Newsome and colleagues administered 15 mg of methylphenidate ( $n = 4$ ) or placebo ( $n = 5$ ) to TBI patients twice a day for 1 month. With pre-and posttreatment assessment of

brain activation, methylphenidate decreased the brain activation in the anterior cingulate, thalamus, cuneus, and cerebellum, which are all associated with working memory. Yet, this is one of the first reports of this type and should be considered with appropriate caution.

Positive results have been shown in other clinical groupings as well. For example, [Meyers, Weitzner, Valentine, and Levin \(1998\)](#) demonstrated improvements in cognitive function in a nonrandomized study of 30 primary brain tumor patients with methylphenidate beginning at 5 mg twice per day. The cognitive improvements were noted in psychomotor speed, memory, visual-motor function, executive function and motor speed, and dexterity. Improvements occurred despite MRI evidence of progressive neurologic injury in more than half of the patients in this small trial.

### Dextroamphetamine

Similar to the effects of methylphenidate, the stimulant medication dextroamphetamine increases the presynaptic release of dopamine and norepinephrine and blocks their subsequent reuptake. Used to address cognitive problems following TBI, dextroamphetamine is argued to be most effective in addressing problems related to arousal, processing speed, and attention ([Arciniegas & Silver, 2006](#)). Although the mechanism of action is similar as in methylphenidate, there is much less published regarding the experimental and clinical use of dextroamphetamine in TBI. The existing evidence suggests dextroamphetamine may improve poststroke aphasia as well as assist in overall cognitive recovery ([Bleiberg, Garmoe, & Cederquist, 1993](#); [Liepert, 2008](#)). Despite some positive evidence, large randomized controlled trials are needed to better assess the effectiveness of dextroamphetamine in TBI-related cognitive impairment.

## **Other Stimulant Medications Affecting Multiple Neurotransmitter Systems**

### Modafinil

Eugeroic agents such as modafinil (M) have demonstrated improvement in cognitive function in a variety of clinical settings. Modafinil is a multiple-system agent. Despite known interactions with receptors for norepinephrine, serotonin, dopamine, gamma-aminobutyric acid (GABA), adenosine, histamine, melatonin, and benzodiazepines, the exact mechanism of action for modafinil remains

unclear. Along with histaminergic action, modafinil appears to increase the release of monoamines, norepinephrine and dopamine in particular. In posttraumatic clinical application, the agent has been reported effective in treating arousal and attention deficits (Teitelman, 2001). In addition, it may be effective in decreasing the levels of fatigue and cognitive impairment during post-TBI treatment (Arciniegas & Silver, 2006; Napolitano et al., 2005). Despite some positive reports, the efficacy of modafinil in treating fatigue and excessive daytime sleepiness in TBI patients remains disputed. Jha et al. (2008) used a double-blind, placebo-controlled crossover trial with 53 participants diagnosed with TBI. The participants were randomly assigned to receive up to 400 mg of modafinil, or equal number of inactive placebo tablets, and the results were analyzed after 4 and 10 weeks since the initial treatment administration. The authors reported no statistically significant differences at either week 4 or week 10, for either fatigue or excessive daytime sleepiness. These results seem to contradict earlier reports. As such, due to a limited number of studies on modafinil and their contradicting results on its effectiveness, the agent should not be recommended for wide clinical use with post-TBI cognitive impairments (Arciniegas & Silver, 2006; Waldron-Perrine, Hanks, & Perrine, 2008).

Within other clinical groupings, modafinil has demonstrated improvements in cognition. For example, Modafinil demonstrated the capacity for significantly improving fatigue, speed of memory, quality of episodic memory, and continuity of attention in a sample of women posttreatment for breast cancer (Kohli et al., 2009).

## Lamotrigine

Lamotrigine is an anticonvulsant recently used in bipolar disorder and post-TBI aggression (Calabrese et al., 1999; Maltese, 1999; Pachet, Friesen, Winkelaar, & Gray, 2003). In TBI, lamotrigine appears to decrease the risk of hippocampal neuronal injury and, therefore, reduces the risk of cognitive impairment. Specifically, lamotrigine inhibits the presynaptic release of glutamate and reduces the neurotoxic levels of zinc after TBI, leading to better cognitive outcomes (Hellmich et al., 2007). The most often observed positive effect of lamotrigine in posttraumatic treatment is related to improvements in arousal during the postacute recovery period (Pachet et al., 2003; Showalter & Kimmel, 2000). Yet, the reports are typically based on case studies, which offer limited validity and generalizability. As such, further clinical trials are needed to identify the efficacy of the drug in post-TBI cognitive rehabilitation.

## Cholinesterase Inhibitors

TBI often results in disruption of the cholinergic system of the brain resulting in memory and executive impairment following TBI. There is an abundance of research suggesting that right after TBI, the brain experiences a period of hypercholinergic activity that is shortly followed by a chronic state of hypocholinergic activity. As such, it is understood that chronic changes in cholinergic functioning in the brain are a common product of TBI (Dewar & Graham, 1996; Murdoch, Perry, Court, Graham, & Dewar, 1998; Shao et al., 1999). As TBI often impacts acetylcholine centers in hippocampal areas, the main cognitive complaints related to acetylcholinergic dysfunction are problems in short-term memory formation and sustained attention (Arcieniegas et al., 1999). Other areas affected by abnormal cholinergic activity include bilateral temporal, cingulate, and parietal cortical areas (Murdoch et al., 1998; Tenovuo, 2005). Hippocampal and frontal cortical cholinergic areas appear to be involved in attention, learning, as well as storage and retrieval of new information (Lawrence & Sahakian, 1995).

Central acetylcholinesterase inhibitors constitute a class of drugs known for increasing the levels of synaptic acetylcholine in the brain. These agents have been commonly used for treatment in some neurodegenerative diseases and are considered relatively safe and effective (Lawrence & Sahakian, 1998). Acetylcholinesterase inhibitors enhance global cognitive performance in Alzheimer's patients (Rogers, Farlow, Doody, Mohs, & Friedhoff, 1998). Following this trend, acetylcholinesterase inhibitors have been considered in other disorders that are associated with cholinergic loss, such as TBI. Cholinesterase-inhibitor agents commonly used in TBI include physostigmine (Eserine), tacrine (Cognex), donepezil (Aricept), rivastigmine (Exelon), and galantamine (Razadyne, Razadyne ER, Reminyl, and Nivalin).

### Physostigmine

Physostigmine has demonstrated effectiveness in both the acute and postacute periods following TBI (Griffin, van Reekum, & Masanic, 2003). Its impact on memory functions has been recognized by both case studies and double-blind placebo projects. In general, physostigmine is shown to improve memory loss after TBI, as compared with placebo or scopolamine, a cholinergic antagonist. Although not all patients seem to improve while taking the drug, there is almost 50% significant recovery in area of memory, particularly for long-term storage

(Cardenas et al., 1994). Other areas that are positively affected by the agent, and are verified by double experimental methods, are sustained attention and standing balance (Cardenas et al., 1994; Levin et al., 1986). Despite positive effects, physostigmine may not be the most optimal agent to use in TBI. Namely, physostigmine has been criticized for its general affects on all cholinergic areas and not just the targeted brain region, has a short half-life period, and its oral absorption rates are variable (Arciniegas & Silver, 2006).

## Rivastigmine

Rivastigmine appears to improve general cognitive performance, particularly memory. Shown to be more effective than placebo, it seems to be well tolerated and safe in patients with severe and moderate posttraumatic cognitive deficits (Silver et al., 2006). Tenovuo, Alin, and Helenius (2009) conducted a randomized double-blind crossover trial with a large randomized sample. When compared with placebo, patients who were given rivastigmine for several weeks performed better on simple math tasks and tests of vigilance. Yet, of the 69 participants, 17 withdrew from the study due to the adverse effect of the treatment. Additionally, the effect of rivastigmine for chronic symptoms of TBI was judged “weak” by the authors.

## Donepezil and Galantamine

Of all the cholinesterase inhibitors, donepezil is the most often used to treat posttraumatic cognitive impairments following TBI. *Donepezil* appears to enhance memory function, attention, and overall cognitive performance when tried with TBI patients (Taverni, Seliger, & Lichtman, 1998; Whitlock, 1999). Donepezil reversibly inhibits acetylcholinesterase, leading to increased synaptic levels of the neurotransmitter acetylcholine. Whelan, Walker, and Schultz (2000) showed that the agent improved overall cognitive performance, as measured by full-scale IQ score. The results were based on a group of 22 TBI patients who underwent 13 months of donepezil treatment. For shorter treatment, the drug seems to mostly affect memory and overall neuropsychiatric performance, as showed by the results of a 4-week study by Masanic, Bayley, VanReekum, and Simard (2001). Zhang, Plotkin, Wang, Sandel, and Lee (2004) provided further evidence that donepezil is beneficial in treatment of short-term memory and sustained attention impairments. Interestingly, their study showed positive effects of the drug regardless of the time that lapsed since the initial injury to medication administration during the postacute recovery period (the participants

ranged from 2 to 24 months postinjury). Standardized measures of attention/concentration, verbal and figural memory as well as confused mood and health-related quality of life were improved over baseline after 24 weeks of treatment with few intolerable toxicities in a sample of 24 brain tumor patients following radiation therapy (Shaw et al., 2006). The importance of initiating the treatment early in the intervention is argued, however, by studies showing limited improvement if the treatment is initiated during the later stages of the postacute recovery period (Walker et al., 2004).

Donepezil, used in chronic post-TBI cognitive impairment, seems to improve the subjective perception of patients' everyday functioning, along with improved performance on objective behavioral measures. Specifically, significant positive changes are found in processing speed, learning, and divided attention (Khateb, Ammann, Annoni, & Diserens, 2005). The rehabilitative improvement seems to be contingent on the dosage; Morey, Cilo, Berry, and Cusick (2003) showed that patients who received 10 mg daily made significant improvements, but patients treated with 5 mg of donepezil per day did not.

Although there are several acetylcholinesterase inhibitors that seem to be discussed in the TBI literature, there is no clear evidence as to which is the most effective in treating posttraumatic cognitive impairment. Comparison studies are rare. Existing literature suggest that in randomized assignment to treatment that consists of donepezil, galantamine, or rivastigmine, about 61% of patients show improvement with little or no difference in drug tolerability. No significant differences among the three drugs were found based on patients' responses (Tenovuo, 2005). To date, there has not been a large-scale placebo study that would compare the effectiveness of these agents on objective neuropsychological measures. In general, the effectiveness of acetylcholinesterase inhibitors in treating post-TBI cognitive dysfunction may depend on whether the dysfunction is a result of cholinergic deregulation.

## SUMMARY

Rehabilitative interventions can take many forms. Although most attention is commonly given to cognitive and behavioral techniques, in viewing neurological insults as physiological processes, it only makes sense that physiological treatments can serve a role in neuropsychological rehabilitation. In particular, agents that augment catecholaminergic and cholinergic functions have demonstrated the greatest efficacy through clinical trials. Still, much remains to be learned about the extent of pharmacological rehabilitation's utility. Moving



forward, research may concentrate on better determining ways of identifying the agent that will offer the greatest effectiveness based on the nature and extent of the neurological insult and possibly host traits. What is clear is that medicinal interventions may have a role in neuropsychological rehabilitation and, therefore, professionals in and around this area need to educate themselves about these agents and their utility.

## REFERENCES

- Arciniegas, D., Adler, L., Topkoff, J., Cawthra, E., Filley, C. M., & Reite, M. (1999). Attention and memory dysfunction after traumatic brain injury: Cholinergic mechanisms, sensory gating and a hypothesis for further investigation. *Brain Injury*, *13*(23), 52–63.
- Arciniegas, D. B., & Silver, J. M. (2006). Pharmacotherapy of posttraumatic cognitive impairments. *Behavioural Neurology*, *17*(1), 25–42.
- Arciniegas, D. B., Held, K., & Wagner, P. (2002). Cognitive impairment following traumatic brain injury. *Current Treatment Options in Neurology*, *4*(1), 43–57.
- Beers, S. R., Skold, A., Dixon, C. E., & Adelson, P. D. (2005). Neurobehavioral effects of amantadine after pediatric traumatic brain injury: A preliminary report. *Journal of Head Trauma Rehabilitation*, *20*(5), 450–463.
- Ben Smail, D., Samuel, C., Rouy-Thenaisy, K., Régnauld, J., & Azouvi, P. (2006). Bromocriptine in traumatic brain injury. *Brain Injury*, *20*(1), 111–115.
- Bleiberg, J., Garmoe, W., & Cederquist, J. (1993). Effect of Dexedrine on performance consistency following brain injury: A double-blind placebo crossover case study. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *6*, 245–248.
- Calabrese, J., Bowden, C., McElroy, S., Cookson, J., Andersen, J., Keck, P. E., Jr., . . . Ascher, J. A. (1999). Spectrum of activity of lamotrigine in treatment-refractory bipolar disorder. *American Journal of Psychiatry*, *156*, 1019–1023.
- Cardenas, D. D., McLean, A., Jr., Farrell-Roberts, L., Baker, L., Brooke, M., & Haselkorn, J. (1994). Oral physostigmine and impaired memory in adults with brain injury. *Brain Injury*, *8*(7), 579–587.
- Chandler, M. C., Barnhill, J. L., & Gualtieri, C. T. (1988). Amantadine for the agitated head-injury patient. *Brain Injury*, *2*(4), 309–311.
- Dewar, D., & Graham, D. I. (1996). Depletion of choline acetyltransferase activity but preservation of M1 and M2 muscarinic receptor binding sites in temporal cortex following head injury: A preliminary human postmortem study. *Journal of Neurotrauma*, *13*(4), 181–187.
- Donnemiller, E., Brenneis, C., Wissel, J., Scherfler, C., Poewe, W., Riccabona, G., & Wenning, G. K. (2000). Impaired dopaminergic neurotransmission in patients with traumatic brain injury: A SPECT study using 123I-beta-CIT and 123I-IBZM. *European Journal of Nuclear Medicine*, *27*(9), 1410–1414.
- Glenn, M. B., & Wroblewski, B. (2005). Twenty years of pharmacology. *Journal of Head Trauma Rehabilitation*, *20*(1), 51–61.
- Griffin, S. L., van Reekum, R., & Masanic, C. (2003). A review of cholinergic agents in the treatment of neurobehavioral deficits following traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, *15*(1), 17–26.
- Gualtieri, C. T., & Evans, R. W. (1988). Stimulant treatment for the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*, *2*(4), 273–290.
- Gualtieri, T., Chandler, M., Coons, T. B., & Brown, L. T. (1989). Amantadine: A new clinical profile for traumatic brain injury. *Clinical Neuropharmacology*, *12*(4), 258–270.
- Hamill, R. W., Woolf, P. D., McDonald, J. V., Lee, L. A., & Kelly, M. (1987). Catecholamines predict outcome in traumatic brain injury. *Annals of Neurology*, *21*(5), 438–443.

- Hellmich, H. L., Eidson, K. A., Capra, B. A., Garcia, J. M., Boone, D. R., Hawkins, B. E., . . . Prough, D. S. (2007). Injured Fluoro-Jade-positive hippocampal neurons contain high levels of zinc after traumatic brain injury. *Brain Research, 1127*(1), 119–126.
- Jha, A., Weintraub, A., Allshouse, A., Morey, C., Cusick, C., Kittelson, J., Harrison-Felix, C., . . . Gerber, D. (2008). A randomized trial of modafinil for the treatment of fatigue and excessive daytime sleepiness in individuals with chronic traumatic brain injury. *Journal of Head Trauma Rehabilitation, 23*(1), 52–63.
- Jorge, R., & Robinson, R. G. (2003). Mood disorders following traumatic brain injury. *International Review of Psychiatry, 15*(4), 317–327.
- Kaelin, D. L., Cifu, D. X., & Matthies, B. (1996). Methylphenidate effect on attention deficit in the acutely brain-injured adult. *Archives of Physical Medicine and Rehabilitation, 77*(1), 6–9.
- Karaküçük, E. I., Pasaoglu, H., Pasaoglu, A., & Oktem, S. (1997). Endogenous neuropeptides in patients with acute traumatic head injury. II: Changes in the levels of cerebrospinal fluid substance P, serotonin and lipid peroxidation products in patients with head trauma. *Neuropeptides, 31*(3), 259–263.
- Khateb, A., Ammann, J., Annoni, J. M., & Diserens, K. (2005). Cognition-enhancing effects of donepezil in traumatic brain injury. *European Neurology, 54*(1), 39–45.
- Kohli, S., Fisher, S. G., Tra, Y., Adams, M. J., Mapstone, M. E., Wesnes, K. A., . . . Morrow, G. R. (2009). The effect of modafinil on cognitive function in breast cancer survivors. *Cancer, 115*(12), 2605–2616.
- Kraus, M. F., & Maki, P. (1997). Case report: The combined use of amantadine and l-dopa/carbidopa in the treatment of chronic brain injury. *Brain Injury, 11*(6), 455–460.
- Kraus, M. F., Smith, G. S., Butters, M., Donnell, A. J., Dixon, E., Yilong, C., & Marion, D. (2005). Effects of the dopaminergic agent and NMDA receptor antagonist amantadine on cognitive function, cerebral glucose metabolism and D2 receptor availability in chronic traumatic brain injury: A study using positron emission tomography (PET). *Brain Injury, 19*(7), 471–479.
- Lal, S., Merbtiz, C. P., & Grip, J. C. (1988). Modification of function in head-injured patients with Sinemet. *Brain Injury, 2*(3), 225–233.
- Lawrence, A. D., & Sahakian, B. J. (1995). Alzheimer disease, attention, and the cholinergic system. *Alzheimer Disease and Associated Disorders, 9*(Suppl. 2), 43–49.
- Lawrence, A. D., & Sahakian, B. J. (1998). The cognitive psychopharmacology of Alzheimer's disease: Focus on cholinergic systems. *Neurochemical Research, 23*(5), 787–794.
- Lee, H., Kim, S. W., Kim, J. M., Shin, I. S., Yang, S. J., & Yoon, J. S. (2005). Comparing effects of methylphenidate, sertraline and placebo on neuropsychiatric sequelae in patients with traumatic brain injury. *Human Psychopharmacology, 20*(2), 97–104.
- Leone, H., & Polsonetti, B. W. (2005). Amantadine for traumatic brain injury: Does it improve cognition and reduce agitation? *Journal of Clinical Pharmacy and Therapeutics, 30*(2), 101–104.
- Levin, H. S., Peters, B. H., Kalisky, Z., High, W. M., von Laufen, A., Eisenberg, H. M., . . . Gary, H. E. (1986). Effects of oral physostigmine and lecithin on memory and attention in closed head-injured patients. *Central Nervous System Trauma: Journal of the American Paralysis Association, 3*(4), 333–342.
- Liepert, J. (2008). Pharmacotherapy in restorative neurology. *Current Opinion in Neurology, 21*(6), 639–643.
- Maas, A. I., Marmarou, A., Murray, G. D., & Steyerberg, E. W. (2004). Clinical trials in traumatic brain injury: Current problems and future solutions. *Acta neurochirurgica., 89*, 113–118.
- Maltese, T. M. (1999). Adjunctive lamotrigine treatment for major depression. *American Journal of Psychiatry, 156*(11), 1833.
- Masanic, C. A., Bayley, M. T., VanReekum, R., & Simard, M. (2001). Open-label study of donepezil in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 82*(7), 896–901.
- Matsuda, W., Komatsu, Y., Yanaka, K., & Matsumura, A. (2005). Levodopa treatment for patients in persistent vegetative or minimally conscious states. *Neuropsychological Rehabilitation, 15*(3–4), 414–427.

- McAllister, T. W. (1992). Neuropsychiatric sequelae of head injuries. *Psychiatric Clinics of North America*, 15(2), 395–413.
- Meyers, C. A., Weitzner, M. A., Valentine, A. D., & Levin, V. A. (1998). Methylphenidate therapy improves cognition, mood, and function of brain tumor patients. *Journal of Clinical Oncology*, 16(7), 2522–2527.
- Meythaler, J. M., Brunner, R. C., Johnson, A., & Novack, T. A. (2002). Amantadine to improve neurorecovery in traumatic brain injury-associated diffuse axonal injury: A pilot double-blind randomized trial. *Journal of Head Trauma Rehabilitation*, 17(4), 300–313.
- Morey, C. E., Cilo, M., Berry, J., & Cusick, C. (2003). The effect of Aricept in persons with persistent memory disorder following traumatic brain injury: A pilot study. *Brain Injury*, 17(9), 809–815.
- Murdoch, I., Perry, E. K., Court, J. A., Graham, D. I., & Dewar, D. (1998). Cortical cholinergic dysfunction after human head injury. *Journal of Neurotrauma*, 15(5), 295–305.
- Napolitano, E., Elovic, E. P., & Qureshi, A. I. (2005). Pharmacological stimulant treatment of neurocognitive and functional deficits after traumatic and non-traumatic brain injury. *Medical Science Monitor*, 11(6), RA212–RA220.
- Newsome, M. R., Scheibel, R. S., Seignourel, P. J., Steinberg, J. L., Troyanskaya, M., Li, X., & Levin, H. S. (2009). Effects of methylphenidate on working memory in traumatic brain injury: A preliminary fMRI investigation. *Brain Imaging and Behavior*, 3(3), 298–305.
- Pachet, A., Friesen, S., Winkelaar, D., & Gray, S. (2003). Beneficial behavioural effects of lamotrigine in traumatic brain injury. *Brain Injury*, 17(8), 715–722.
- Passler, M. A., & Riggs, R. V. (2001). Positive outcomes in traumatic brain injury-vegetative state: Patients treated with bromocriptine. *Archives of Physical Medicine and Rehabilitation*, 82(3), 311–315.
- Powell, J. H., al-Adawi, S., Morgan, J., & Greenwood, R. J. (1996). Motivational deficits after brain injury: Effects of bromocriptine in 11 patients. *Journal of Neurology, Neurosurgery, and Psychiatry*, 60(4), 416–421.
- Rogers, S. L., Farlow, M. R., Doody, R. S., Mohs, R., & Friedhoff, L. T. (1998). A 24-week, double-blind, placebo-controlled trial of donepezil in patients with Alzheimer's disease. Donepezil Study Group. *Neurology*, 50(1), 136–145.
- Shao, L., Ciallella, J. R., Yan, H. Q., Ma, X., Wolfson, B. M., Marion, D. W., . . . Dixon, C. E. (1999). Differential effects of traumatic brain injury on vesicular acetylcholine transporter and M2 muscarinic receptor mRNA and protein in rat. *Journal of Neurotrauma*, 16(7), 555–566.
- Shaw, E. G., Rosdhal, R., D'Agostino, R. B., Lovato, J., Naughton, M. J., Robbins, M. E., & Rapp, S. R. (2006). Phase II study of donepezil in irradiated brain tumor patients: Effect on cognitive function, mood, and quality of life. *Journal of Clinical Oncology*, 24(9), 1415–1420.
- Showalter, P. E., & Kimmel, D. N. (2000). Stimulating consciousness and cognition following severe brain injury: A new potential clinical use for lamotrigine. *Brain Injury*, 14(11), 997–1001.
- Silver, J. M., Koumaras, B., Chen, M., Mirski, D., Potkin, S. G., Reyes, P., . . . Gunay, I. (2006). Effects of rivastigmine on cognitive function in patients with traumatic brain injury. *Neurology*, 67(5), 748–755.
- Silver, M., Arciniegas, D. B., & Yudofsky, S. C. (2005). Psychopharmacology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 609–639). Arlington, VA: American Psychiatric.
- Taverni, J. P., Seliger, G., & Lichtman, S. W. (1998). Donepezil medicated memory improvement in traumatic brain injury during post acute rehabilitation. *Brain Injury*, 12(1), 77–80.
- Teitelman, E. (2001). Off-label uses of modafinil. *American Journal of Psychiatry*, 158(8), 1341.
- Tenovuo, O. (2005). Central acetylcholinesterase inhibitors in the treatment of chronic traumatic brain injury-clinical experience in 111 patients. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 29(1), 61–67.
- Tenovuo, O. (2006). Pharmacological enhancement of cognitive and behavioral deficits after traumatic brain injury. *Current Opinion in Neurology*, 19(6), 528–533.
- Tenovuo, O., Alin, J., & Helenius, H. (2009). A randomized controlled trial of rivastigmine for chronic

- sequels of traumatic brain injury-what it showed and taught? *Brain Injury*, 23(6), 548–558.
- Waldron-Perrine, B., Hanks, R. A., & Perrine, S. A. (2008). Pharmacotherapy for postacute traumatic brain injury: A literature review for guidance in psychological practice. *Rehabilitation Psychology*, 53(4), 426–444.
- Walker, W., Seel, R., Gibellato, M., Lew, H., Cornis-Pop, M., Jena, T., & Silver, T. (2004). The effects of Donepezil on traumatic brain injury acute rehabilitation outcomes. *Brain Injury*, 18(8), 739–750.
- Warden, D. L., Gordon, B., McAllister, T. W., Silver, J. M., Barth, J. T., Bruns, J., . . . Zitnay, G. (2006). Guidelines for the pharmacologic treatment of neurobehavioral sequelae of traumatic brain injury. *Journal of Neurotrauma*, 23(10), 1468–1501.
- Waxweiler, R. J., Thurman, D., Sniezek, J., Sosin, D., & O’Neil, J. (1995). Monitoring the impact of traumatic brain injury: A review and update. *Journal of Neurotrauma*, 12(4), 509–516.
- Whelan, F. J., Walker, M. S., & Schultz, S. K. (2000). Donepezil in the treatment of cognitive dysfunction associated with traumatic brain injury. *Annals of Clinical Psychiatry*, 12(3), 131–135.
- Whitlock, J. A. (1999). Brain injury, cognitive impairment, and donepezil. *Journal of Head Trauma Rehabilitation*, 14(4), 424–427.
- Whyte, J., Hart, T., Schuster, K., Fleming, M., Polansky, M., & Coslett, H. B. (1997). Effects of methylphenidate on attentional function after traumatic brain injury. A randomized, placebo-controlled trial. *American Journal of Physical Medicine & Rehabilitation/Association of Academic Physiatrists*, 76(6), 440–450.
- Whyte, J., Hart, T., Vaccaro, M., Grieb-Neff, P., Risser, A., Polansky, M., & Coslett, H. B. (2004). Effects of methylphenidate on attention deficits after traumatic brain injury: A multidimensional, randomized, controlled trial. *American Journal of Physical Medicine and Rehabilitation*, 83(6), 401–420.
- Whyte, J., Vaccaro, M., Grieb-Neff, P., Hart, T., Polansky, M., & Coslett, H. B. (2008). The effects of bromocriptine on attention deficits after traumatic brain injury: A placebo-controlled pilot study. *American Journal of Physical Medicine & Rehabilitation/Association of Academic Physiatrists*, 87(2), 85–99.
- Woolf, P. D., Hamill, R. W., Lee, L. A., Cox, C., & McDonald, J. V. (1987). The predictive value of catecholamines in assessing outcome in traumatic brain injury. *Journal of Neurosurgery*, 66(6), 875–882.
- Writer, B. W., & Schillerstrom, J. E. (2009). Psychopharmacological treatment for cognitive impairment in survivors of traumatic brain injury: A critical review. *Journal of Neuropsychiatry and Clinical Neurosciences*, 21(4), 362–370.
- Zafonte, R. D., Lexell, J., & Cullen, N. (2001). Possible applications for dopaminergic agents following traumatic brain injury: Part 2. *Journal of Head Trauma Rehabilitation*, 16(1), 112–116.
- Zhang, L., Plotkin, R. C., Wang, G., Sandel, M. E., & Lee, S. (2004). Cholinergic augmentation with donepezil enhances recovery in short-term memory and sustained attention after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 85(7), 1050–1055.

# Telerehabilitation and Teleneuropsychology: Emerging Practices

*Michael McCue and C. Munro Cullum*

## OVERVIEW OF TELEREHABILITATION

Telerehabilitation (TR) has been defined as the delivery of rehabilitation services via information and telecommunication technologies (i.e., assessment, monitoring, intervention, supervision, education, consultation, and counseling; [American Telemedicine Association, 2009](#)). The field of TR incorporates technologies that have the potential to make more efficient use of health care resources, as well as impact the quality of life of consumers with disabilities ([Krupinski et al., 2002](#); [McCue, Fairman, & Pramuka, 2010](#); [Rosen, 1999](#)). TR emerged as a response to the need to provide access to rehabilitation services for individuals who lived in remote areas without needed resources and services. In particular, the specialized services and expertise required by individuals with neurological disorders and developmental disorders are often only available in populated urban areas. TR offers a mechanism of access to necessary assessment and treatment resources.

The term *telerehabilitation* is relatively new, but remote medical interventions date back to the 1880s, when some physicians experimented with telecommunication technologies after the invention of the telephone in 1876 ([Scalvini, Vitacca, Paletta, Giordano, & Balbi, 2004](#)). The U.S. Government first supported telemedicine through agencies such as the U.S. Department of

Veterans Affairs (VA) (U.S. Department of Veterans Affairs). The first recorded use of telemedicine in the VA was in 1957, for a telemental health project in Nebraska (Cooper et al., 2001).

A review of TR is provided by Winters (2002). Winters classifies TR as falling under *telehealth*, a broader category of services that uses telecommunication to provide health information and care across distance. Telehealth is broken into three subcategories: telemedicine, telehealthcare, and ehealth/education. These technologies have been utilized across a multitude of disciplines, including, but not limited to, telepsychiatry and teleneurology. TR falls under telehealthcare along with telehomecare, telenursing, and telecoaching. These terms, and others (e.g., teletherapy, telecounseling), are often utilized interchangeably throughout the literature. Schmeler, Schein, McCue, and Betts (2009) proposed that TR warrants a separate and parallel identity alongside telehealthcare and telemedicine.

From the informatics technology perspective, Parmanto and Saptono (2009) discuss several differences between telemedicine and TR. They provide a classification of telemedicine and TR along the continuums of duration of service provision and technical intensity of the clinical service. Based upon a review of 238 papers on various “tele” applications, they mapped the applications into four quadrants. What they found was that, in general, telemedicine applications such as telesurgery, telepathology, teledermatology, and teleradiology were characterized by short-duration clinical episodes but had a high degree of intensity with respect to the amount of information exchanged (e.g., file sizes) or the speed and resolution of video required. Alternately, TR applications such as telecounseling, telespeech therapy, telecoaching, and telemental health services were of longer duration with reasonably lesser requirements for technical intensity. Recommendations were made for various technologies based upon where the clinical activity fell on the intensity and duration dimensions. This analysis identified several differences between telemedicine and TR. These differences are further emphasized when one considers the characteristics and contextual factors involved in rehabilitation (versus traditional medical practice), particularly when working with a neurological rehabilitation population.

Because of the complexity of impairments and functional implications associated with cognitive disability, rehabilitation efforts are often required well beyond traditional acute and postacute interventions. In order to ensure that individuals with cognitive disabilities successfully meet the challenges of

independent living, community participation and socialization, education and training, and ultimately employment, community-based rehabilitation resources and options are essential. Given factors such as the emphasis on acute and immediate postacute rehabilitation, funding constraints on outpatient and community-based services, resource accessibility, resource shortages, and the unique challenges of providing functionally relevant and effective services that impact goals of community functioning and employment, the need has extended beyond the capacity of current rehabilitation systems and providers to supply these essential services.

The practice of rehabilitation invokes characteristics and contextual factors that are unique and different from medicine. In rehabilitation, encounters between clients and professionals are usually characterized as repetitive, established relationships that take place over time. This is in contrast with what often happens in medicine, wherein treatment of an illness is generally of short duration, and as the condition improves, there is less need for physician time and medical contact. Based upon evidence in rehabilitation with persons with significant disabilities, such as traumatic brain injury (TBI), needs for services often escalate over time as individuals recover, so the needs that these individuals have actually become greater and more involved as they move forward. Unfortunately, reimbursement systems are much less likely to provide postacute and community-based services that many persons need. Moreover, the difficulties people experience are often most evident where and when they interact with demands in their community, at work, when they are shopping, when they are interacting with peers, or when they are trying to have a social relationship, rather than in the clinical settings, where most individuals receive services.

Another characteristic component of rehabilitation requires and enables clients to practice extensively what they learn in the clinic, and then apply it to their lives. Rehabilitation is typically a multidisciplinary endeavor. As a result, we are required to work together to ensure that what happens in one rehabilitation setting carries over, rather than reinventing the wheel with each professional contact. It is important to incorporate a mechanism for what is learned or gained to carry over from one situation or setting to the next, and to promote a continuity of care that meets patients' needs as they progress.

The importance of attention to contextual factors in rehabilitation is emphasized in the International Classification of Functioning (ICF) of [World Health Organization \(2001\)](#) that stresses that an individual's functioning must be

understood within the context of his or her environment (Kuipers, Foster, Smith, & Fleming, 2009). Recognizing that the social and physical environment can be facilitative (or inhibitory), rehabilitation that can occur within the patient's own home and community has greater relevance to the patient. Ylvisaker (2003) states that for individuals with brain injury, cognitive rehabilitation that occurs in the natural setting, and within the context of everyday interaction and demand domains is more relevant to the individual. Willer and Corrigan (1994) cite that the issue of generalization can be a major obstacle to achieving a successful rehabilitation outcome. What is learned or accomplished in one setting (e.g., a clinic) does not necessarily generalize to other settings. Willer and Corrigan further assert that the problem of failure of generalizability can be successfully addressed by conducting rehabilitation in the environment in which the skills must be applied.

In summary, TR might be conceptualized as a delivery system for rehabilitation that utilizes technology to serve clients, clinicians, and systems by minimizing the barriers of distance, time, and cost. The existing literature indicates that the rationale for the use of TR has been to reduce costs, increase geographical accessibility, and/or as a mechanism to extend limited resources. The potential to enhance outcomes beyond what may result from face-to-face interventions by enabling naturalistic, in vivo assessments and interventions represents additional potential TR applications. There is considerable support for the value of interventions delivered in the natural environment, ranging from addressing efficacy concerns by addressing problems of generalization to increasing patient participation, including environmental context into rehabilitation, and increasing patient satisfaction. Further clinical and research exploration should explore TR as a tool for the delivery of rehabilitation services in vivo.

## **EMPIRICAL EVIDENCE FOR TR**

The evidence for the efficacy of TR is limited by the great diversity of clinical applications, disabilities served, technologies utilized, and professional disciplines involved in the delivery of TR services. The existing research reflects a general lack of randomized clinical trials, small sample sizes, and a lack of common metrics and outcome measures. Currently, there is evidence of considerable variability in outcome measurement tools in rehabilitation service. Literature reviews indicate significant challenges to identification of outcome measures that could be applied uniformly to evaluating the cost-effectiveness,



usability and satisfaction, and clinical efficacy of TR. In spite of the need for a more practical–clinical outcome-grading system, no consensus has been reached on common, practical, standard outcome measurements for the worldwide exchange of information and data in TR. This has made it difficult for groups implementing TR programs and using customized outcome schemes to compare their data with that obtained across various rehabilitation disciplines, client populations, clinical applications, or countries, where varieties of measures are employed.

Much of the research literature on TR has focused outcome measures on decreasing costs, saving travel time, and improving access to specialty services and expert practitioners (Bashshur, 2002). The rationale proposed to support the exploration and implementation of TR has been primarily based upon the use of various technologies to address geographic and economic barriers, and potentially enhance cost-effectiveness. An alternative perspective is that TR technologies can be implemented to provide rehabilitation services in the individual's environment (home, community, workplace, etc.) and that such interventions may further enhance rehabilitation outcomes.

Recently, there have been three comprehensive literature reviews on TR. Kairy, Lehoux, Vincent, and Visintin published a systematic review of TR in 2009 in *Disability and Rehabilitation*. A second review was conducted by the Canadian Institute of Health Economics in 2010. Most recently, a publication in *Neurorehabilitation* by Rogante, Grigioni, Cordella, and Giacomozzi (2010) surveyed TR evidence from 1998 to 2008. Together, these reviews provide preliminary, yet compelling evidence supporting the use of TR services in a variety of contexts and conditions.

Kairy *et al.* (2009) conducted a systematic review and meta-analysis of 28 studies published in peer-reviewed journals that utilized a TR component in a population with physical deficits, using observational or experimental methodologies. Studies were excluded if they dealt with mental illness without physical disability. Data that were extracted included information regarding study design, target population, description of the rehabilitation intervention, and the technology utilized. Outcomes were reported with respect to clinical measures of efficacy, service delivery outcomes (process data), health care utilization data, and costs. Studies were grouped and outcomes were summarized according to the type of rehabilitation intervention and the setting in which the intervention was delivered (e.g., home, clinical setting, etc.).

Studies involved patients with a variety of conditions, including neurologic

disorders (TBI, stroke, multiple sclerosis), speech and language impairments, orthopedic injuries, and cardiac rehabilitation. With respect to clinical outcomes, “studies report positive clinical outcomes, with improvement in physical, functional and psychological measures following a telerehabilitation intervention” (p. 340). Process variables were less frequently addressed in the studies reviewed, but findings indicated good program attendance and compliance with rehabilitation recommendations by patient recipients of TR interventions.

Patient and clinician satisfaction with TR in these studies was high, with greater satisfaction reported by patients. Only five studies addressed cost analysis, with no consistency across these studies with respect to how they calculated costs. Although these few investigations suggested lower health care facility costs, the limited number of studies, and methodologic limitations “prevents us from any definite drawing conclusions about the cost-effectiveness for telerehabilitation” (p. 443).

[Kairy et al. \(2009\)](#) noted that almost half of the 28 studies were rated as well-conducted randomized control trials or quasi-experimental designs with control groups, providing stronger evidence upon which to base conclusions regarding efficacy. Based upon their meta-analysis, the authors concluded, “that telerehabilitation can lead to similar clinical outcomes compared to traditional rehabilitation programs, with possible positive impacts on some areas of healthcare utilization” (p. 445). They found insufficient evidence to support the cost effectiveness of TR, however.

The Institute of Health Economics of Canada and the Finnish Office for Health Technology Assessment published a review article on the effectiveness of TR applications ([Hailey, Roine, Ohinmaa, & Dennett, 2011](#)). This review identified 61 studies that met the criteria of scientifically valid research on the outcomes of TR applications, controlled studies comparing TR with a non-TR alternative, and uncontrolled studies reporting TR outcomes with 20 or more subjects.

The reviewed research was classified by *study performance* (considering patient selection, description of interventions, methodologic descriptions, patient disposition, and outcomes reported) and by *classification of the study design* (from large randomized controlled trials to noncontrolled series). These scores were then combined to obtain an overall quality score that would address the confidence that decision makers should place in the findings that were reported. From 1870 publications that were identified in their literature search, 66 papers

describing 61 unique studies meeting the selection criteria were included in the review (Hailey et al., 2011).

This systematic review organized the findings by clinical category (e.g., cardiology, neurology, speech disorders, rheumatology). For the purpose of this chapter, only the findings regarding the neurology-related applications of TR are discussed. Of 18 total studies in this section, 8 involved TR with TBI. Five of the eight studies provided evidence that TR interventions were successful, while in the remaining three studies, effectiveness was unclear. “High quality studies with persons who had suffered traumatic brain injury (TBI) showed benefits in ameliorating depressive symptoms, improving behavioral outcomes and increasing the probability of returning to employment” (Hailey et al., 2011, p. 9).

Overall conclusions of Hailey et al. (2011) were similar to those of Kairy et al.’s (2009) review in that further research with stronger studies was recommended. Authors concluded that TR “shows promise in many fields but compelling evidence of benefit, and of impact on routine rehabilitation, seems likely to await the availability of adequate research funding and a high level of commitment by rehabilitation professionals to engage in longer-term studies” (Hailey et al., 2011, p. 21).

In an attempt to provide an overview of the literature on TR, Rogante et al. (2010) more broadly surveyed TR evidence published in peer-reviewed journals from 1998 to 2008. Literature falling into various categories was included: reviews, technical reports, patient management and clinical applications, and opinion or editorial articles. The authors gathered information on the pathologies and impairments treated by the TR technology, the number of patients included, and the technology involved. A total of 146 articles were reviewed that represented all articles from 1998 to 2008, resulting in the authors using the search term *telerehabilitation* or *telerehabilitation*.

Rogante et al. (2010) reported that acceptance of TR by patients and clinicians was generally high, although most reviews of the topic lack clear objective measures for evaluation of perception and acceptance. Insufficient evidence was found to confirm the cost-effectiveness of TR. Regarding technical approaches; audio/video technologies were the most frequently described, but that “in real clinical applications, the trend is to use combined technologies rather than a single, specialized one” (Rogante et al., 2010, p. 294). When analyzing patient management data, the authors reported that an audio/video link was important for connecting patients and health care providers, and that “bi-directional videoconference are at the same time the most studied and most used

technologies in telerehabilitation” (p. 294). Rogante *et al.* (2010) concluded that the literature lacks the comprehensive and scientifically rigorous studies required to provide evidence for TR.

## TELENEUROPSYCHOLOGY AND COGNITIVE ASSESSMENT

The telephone is the earliest telecommunication technology used for teleneuropsychological assessment. Telephone, and its modern version of interactive voice-response technology, is attractive because it is the simplest and most ubiquitous telecommunication technology available. It has been used in a number of teleneuropsychological assessment applications, including assessment of cognitive and psychomotor recovery after surgery (Mundt, Geralts, & Moore, 2006) and cognitive function of the elderly (Debling, Amelang, Hasselbach, & Stürmer, 2005). Several standardized mental status screening examinations have been developed specifically for telephone administration (e.g., the *Telephone Interview for Cognitive Status [TICS]*; Brandt *et al.*, 1993). Most have demonstrated adequate reliability and provide for a highly cost-effective means of obtaining some information regarding patients’ gross cognitive status without requiring an office visit or elaborate equipment. However, telephone communication does not allow the psychologist to make eye contact, observe facial expression and physical movements, and significantly limits the types of tasks that can be administered. Furthermore, telephone-based testing has other limitations such as: (a) the possibility of patients obtaining assistance from others during testing, (b) writing down responses or cues during testing, (c) using a Smart phone or computer to assist responses, (d) hearing limitations, (e) environmental distractors, and (f) the potential effects of nonstandardized testing conditions. Such factors necessarily relegate teletesting suitable only for limited teleneuropsychological applications (Ball, Tyrrell, & Long, 1999).

Real-time teleneuropsychological assessment is primarily achieved with the use of videoconferencing (VC). To conduct these evaluations, a set of videoconferencing equipment is located at the psychologist’s site and another set is located at the remote client site. The two stations are usually connected at minimum by a standard telephone line, a dedicated ISDN (integrated services digital network) line (128 kbit/s), by three parallel ISDN lines with a total bandwidth of 384 kbit/s (Schopp, Johnstone, & Merrell, 2000), or by Internet protocol (IP; Parmanto *et al.*, 2010). Having cameras at both ends with mobile and zoom capacity is very useful, when there is a need to focus on the patient or what he or she is doing with his or her hands (e.g., drawing). The video images

transmitted to the patients are either live picture of the psychologist or visual test materials presented via document camera. The use of a split-screen presentation of the patient and what he or she is viewing is also helpful, when visual stimuli are being presented. Alternately, a document camera may be added to the videoconferencing equipment to enhance the resolution and stability of the visual and printed materials, though this may not be necessary, depending upon the visual stimuli that are being presented. Because visual and audio feedback from the patient at the remote site is critical to the flow of the examination and to observation and recording of test performance, sufficient resolution and speed are required (30 frames/s) for the picture not to flicker or blur moving images.

### **CHALLENGE OF ESTABLISHING EVIDENCE FOR TELEASSESSMENT**

To date, only a handful of scientific studies have been published documenting the feasibility, reliability, and validity of telecognitive assessment. Investigations have varied in their assessment tools and study populations as well, with most focusing on brief cognitive screening tools, such as the Mini Mental State Examination (*MMSE*) (e.g., [Ball, Scott, McLaren, & Watson, 1993](#); [Loh, Donaldson, Flicker, Maher, & Goldswain, 2007](#); [Loh et al., 2004](#)), *MMSE + Clock Drawing* ([Montani et al., 1997](#)), Cambridge Cognition Examination (CAMCOG) ([Ball & Puffett, 1998](#)), and *Short Portable Mental Status Questionnaire* ([Menon et al., 2001](#)). Several other investigations have examined a variety of standard and experimental neuropsychological tests in healthy volunteers ([Hildebrand, Chow, Williams, Nelson, & Wass, 2004](#); [Jacobsen, Sprenger, Andersson, & Krogstad, 2003](#)), patients with alcohol abuse ([Kirkwood, Peck, & Bennie, 2000](#)), and patients with mild dementia ([Cullum, Weiner, Gehrmann, & Hynan, 2006](#); [Vestal, Smith-Olinde, Hicks, Hutton, & Hart, 2006](#)).

In practice, many clinicians applying this technology to neuropsychological assessment are relying upon the “why wouldn’t it work in this setting?” approach, when, in fact, psychometric data supporting the validity of this testing medium remain limited. Even the more “extensive” batteries of tests that have been used in telecognitive assessment applications noted earlier tend to be fairly brief and rely heavily upon verbally oriented measures that do not require much in the way of manipulable test materials. For example, one of the larger studies to date ([Cullum et al., 2006](#)) included 33 subjects (14 mild cognitive impairment [MCI] and 19 AD) and consisted of a brief battery of tests commonly used in the

evaluation of patients with known or suspected dementia (*MMSE*, *Hopkins Verbal Learning Test-Revised*, *Digit Span*, *Letter fluency*, *Category fluency*, *Boston Naming Test* [15-item version], and *Clock Drawing*). Alternate forms and items were used in counterbalanced fashion between in-person and traditional face-to-face testing. Results revealed good agreement between results across test conditions, with intraclass correlations ranging from 0.58 to 0.88 across measures. Since that pilot study, we have tested a larger number of subjects in similar fashion and found evidence of good test–retest reliability in healthy controls and subjects with dementia, including those with *MMSE* scores as low as 15. Subjects showed good tolerability of the VC-based interaction, and most found it to be an acceptable means of undergoing neurocognitive assessment.

Of course, the VC environment poses limits in terms of the nature and types of cognitive tests that can be administered. Most traditional neuropsychological tests involve paper–pencil and question–answer types of tasks. Thus, although some tests would lend themselves seemingly well to VC-based interactions, portions of the neuropsychological examination would require modification, and certain tasks would need to be omitted because of the lack of direct patient contact. For example, question–answer types of tasks and those heavily dependent upon verbal instructions and responses may require little or no modification. Remote administration of measures requiring psychomotor responses, such as the Block Design subtest of the *WAIS-IV*, however, might require a support person to be in the room with the person being tested, or at least necessitate the availability of test stimuli at the other end, with modified instructions and procedures to complete the test. Other issues to be addressed include the applicability of existing test norms to VC-based test administration to ensure that telecognitive test results could be interpreted in the same way as standard testing. Thus far, only a handful of standard neuropsychological tests have been examined within the VC context, and caution must be used to interpret scores from measures that have not yet been studied in this environment.

## **POTENTIAL LIMITING FACTORS IN TELEHEALTH TECHNOLOGY**

The speed at which audiovisual data can be transmitted, or *bandwidth* (commonly reflected by the number of kilobits per second, or *kbs*), is an important factor for clinical and research applications of VC technology within an assessment context. Bandwidth must be sufficient to transmit more than 30 frames per second, for example, to eliminate jerkiness of movement. Early

studies with low bandwidth (<384 kbs) sometimes reported poor image quality due to tiling and pixilation with movement (Jones, Johnston, Reboussin, & McCall, 2001), but some telemedicine programs have been effective with lower bandwidth. For example, the University of Kansas operated a telepsychiatry service to a county jail at 128 kbs that was well accepted by the inmates (Zaylor, Whitten, & Kingsley, 2000). At 128 kbs, there can be a slight delay (0.5–1.0 s) in transmission to which users must adapt, but at 512 kbs, for example, the interaction virtually replicates live interactions. Sound transmission has sometimes been a problem in the past, but this is less frequently reported in published studies (Hilty, Luo, Morache, Marcelo, & Nesbitt, 2002). *Distance* can have an impact on the coordination of images and sound, with greater distance resulting in greater delay of sound, but usually not to the point where it greatly compromises communication. Even where there is a fraction of a second delay in sound, that is readily adapted to on both ends. Modern data transmission speeds and increased equipment reliability have significantly reduced distance as a barrier to VC-based interactions. For example, in our testing of more than 200 subjects via VC, transmission errors did occur more frequently when using a remote link, but we only lost connection once and experienced momentary glitches in transmission in fewer than 5% of test sessions, with none of these episodes judged to adversely impact examiner–examinee interactions or test results.

*Cost* has been posited as perhaps one of the most significant limiting factors, although reduced costs of equipment and data transmission, in addition to more widespread availability, have made a major impact in terms of cost reduction. In the past, startup costs were prohibitive, but VC systems are now in common use and are thus readily available in many settings across the globe. Although the issue of cost is difficult to ascertain in an absolute sense due to the myriad of factors involved (e.g., see Kennedy, 2005), published reviews of the cost-effectiveness of telepsychiatric services have included some discrepant findings. However, most authors have concluded that telepsychiatry is cost-effective (Hilty et al., 2002; Monnier, Knapp, & Frueh, 2003; Schopp, Johnstone, & Merveille, 2000), particularly when improved access to specialty care is considered as a counterbalancing factor (Kennedy, 2005). Tang, Chiu, Woo, Hjelm, and Hui (2001) estimated cost per telemedicine session at \$91.81, which was slightly less than their \$105.78 cost for an onsite visit. Ruskin et al. (2004) estimated marginal costs to a VA facility at \$86 for VC treatment versus \$63 for in-person treatment, not including physician travel time. They calculated

that at 22 miles distance, costs were equivalent, but at distances more than 22 miles, VC was less expensive. [Hassall, Wootton, and Guilfoyle \(2003\)](#) reported that VC-based assessments conducted in rural, frail elderly cost \$84.93, compared to \$90.25 for face-to-face assessments. Based upon data from the randomized clinical trial by [O'Reilly et al. \(2007\)](#) that included 495 subjects, it was calculated that the 241 VC-based interventions were at least 10% less costly on average than the 254 in-person sessions that were conducted. Similarly, in reviewing over 1800 telemental health visits at the National Naval Medical Center at Bethesda from a remote military medical clinic, [Grady \(2002\)](#) concluded that telemedicine can be done at comparable or reduced costs. Recently, [Shore, Brooks, Savin, Manson, and Libby \(2007\)](#) compared the direct costs of conducting structured psychiatric interviews via VC compared with in-person interviews in a rural sample of American Indians over a 2-year period. Results revealed that VC-based interviews were more costly than in-person interviews in 2003, although significant cost savings were realized through the use of VC-based interviews in 2005, as a result of decreased equipment and transmission costs. They furthermore concluded that the use of VC technology may decrease costs of clinical research in rural, remote, and underserved populations. In our setting, the UT Southwestern VC link from the university VC studio to the Choctaw Nation Healthcare Center in Southeastern Oklahoma currently costs approximately \$120/h. Additional costs include the time of support and technical personnel at the remote site and local cost of clinician's or examiner's time. Because in-person evaluations at the remote site would require travel time and an overnight stay for a full-day's worth of patients, VC-based assessment appears more cost-effective in this setting. Thus, at this point in time, cost does not appear to be a major limiting factor, given the availability of the technology required to establish VC connections.

In terms of VC-based psychiatric evaluations and interventions, it is important to note that patients as well as clinicians report levels of satisfaction with VC-based services at least as high as with traditional face-to-face services ([Hilty et al., 2002](#); [Hilty, Nesbitt, Kuenneth, Cruz, & Hales, 2007](#); [Kobak, 2004](#); [O'Reilly et al., 2007](#); [Shore & Manson, 2004](#); [Shores et al., 2004](#)). In reviewing the initial 5 years of the University of Arizona Teleconsultations Programme, [Cruz, Krupinski, Lopez, and Weinstein \(2005\)](#) reported a total of 1086 telemedicine consults on 206 patients. High satisfaction was reported with the services, although 17% of patients and 18% of providers found equipment problems distracting at times. [Shores et al. \(2004\)](#) reported that although many



telepsychiatry subjects indicated that they would have preferred to see a physician in person, 94% felt they could communicate with the physician via VC as well as in person, and 76% of Kobak's (2004) subjects said they preferred telepsychiatry services to traveling long distances to a doctor's office. In an investigation using two remote military bases for telepsychiatry, Grady and Melcer (2005) found that compliance was actually *better* for telepsychiatry versus in-person appointments. We were unable to find any data in the literature regarding acceptability and satisfaction with VC-based neurocognitive testing, although our experience from an ongoing investigation of telecognitive assessment found high-acceptability ratings. Specifically, 98% of 40 subjects (including healthy older controls and individuals with mild cognitive impairment or early dementia) tested in the traditional face-to-face manner and via VC-administered testing expressed high satisfaction with both test conditions, and 60% expressed no preference in test modality (Parikh et al., 2012).

One of the major advantages of telemedicine is its potential to reach patients in rural areas and provide access to specialists and specialized services that may otherwise be unavailable or difficult to obtain (Darkins, 2006; Hilty, Yellowlees, & Nesbitt, 2006). In reviewing 200 consecutive first-time telepsychiatry consultations, Hilty et al. (2007) concluded that, "Telepsychiatry programs may enhance access, satisfaction, and quality of rural care." Similar conclusions have been reached by others reviewing the literature, including Norman's (2006) overview of the use of telepsychiatry in the United Kingdom. Many other countries have developed telemedicine programs in psychiatry, dermatology, and radiology, and the future appears bright for increased utilization of VC technology in the clinical neurosciences, and in the diagnosis and management of dementia in particular (Vilalta-Franch, Garre-Olmo, López-Pousa, Coll-De Tuero, & Monserrat-Vila, 2007). At this point, there is no reason to believe that similar programs, including teleneuropsychological or telecognitive assessment would be any less successful, with the aforementioned testing caveats in mind. The additional opportunities for bringing cutting-edge research into such settings are also promising and may lead to an enhanced understanding of rural and underserved patient needs and improved health care.

## TR TECHNOLOGIES

Information and communication technologies for providing TR can be classified as either synchronous (in real-time—when face-to-face contact between parties is required), or asynchronous. Asynchronous applications are referred to as

“store and forward” technologies because data are captured at one point in time, stored (temporarily or permanently), and transmitted later for review or interpretation. The exchange may be directly between a patient and clinician, or between a technician who engages the patient in the testing and interview, and manages the technology. Technology may also involve web-based, robotic, or virtual-reality-based formats, and used autonomously by patients remotely, with the clinician monitoring patient responses and modifying the tasks accordingly.

Videoconferencing is the most popular synchronous rehabilitation technology, although other technologies such as chat, SMS text messaging, and audio telephony have been applied in real-time rehabilitation applications. Often, varied technologies are used in combination to address clinical needs. It is not unusual to use store-and-forward, interactive, audio, and video still images in a variety of combinations and applications.

Videoconferencing *systems* are a set of interactive telecommunication technologies that allow two or more locations to interact via two-way video and audio transmissions simultaneously. These systems typically consist of a video monitor, video camera, speakers, microphone, and a CODEC. The CODEC uses hardware and/or software to simultaneously compress and decompress digital video and audio information and sends it to another CODEC, where the same process is also being done. These systems are proprietary and can only be used to communicate with similar systems, rendering them expensive and often inaccessible to individuals and small community-based agencies.

More recently, use of the Internet to transfer clinical information and data is becoming more prevalent. IP-based videoconferencing systems such as Skype ([Baset & Schulzrinne, 2004](#)) and Apple Face Time ([Grothus & Sadun, 2011](#)) allow individuals to conference over the Internet using only a video camera attached to a laptop or home computer. Although this is still an inexpensive and widely available technology, the level of security required to meet confidentiality and privacy requirements in accordance with HIPAA regulations is difficult to document or may be unattainable by the user.

Advances in information technology applied to the clinical need not only for secure data transmission and storage, including that required for video conferencing, but have also produced prototype systems for TR. For example, the Versatile and Integrated System for Telerehabilitation, or VISYTER, is a software platform that has been applied to various TR applications, including remote psychological and neuropsychological assessment, remote memory training, and remote self-management of clinical applications ([Parmanto et al.,](#)

2010). An evaluation of this system, which not only includes high-quality video conferencing, but also allows for integration of electronic health records, stimuli presentation, scoring, remote camera control, and standard clinical forms and protocols, has produced very favorable satisfaction from patients and clinicians (Parmanto et al., 2010).

Other technologies, including remote desktop control by the therapist (or desktop “push”), are examples of how rehabilitation services, such as job coaching and career development counseling, can be applied remotely (Pramuka et al., 2006).

Real-time access may also be provided through *wireless technologies* that transfer information over a distance without the use of electrical conductors or “wires.” The distances involved may be short (a few meters as in television remote control) or long (thousands of miles for radio communications). This technology offers the advantage of increased freedom for use within various environments and allows unrestricted movement. Smart phones offer the capacity to provide various rehabilitation interventions and monitoring applications in the home and community environments, including video conferencing.

Other technologies, including monitoring technologies such as environmental sensors and body monitoring, accelerometers, and virtual reality have been demonstrated in the TR literature.

## PRACTICE ISSUES

### Ethical Considerations and Competence

The provision of TR and teleneuropsychological services raises a variety of issues that clinicians and researchers must consider. In addition to the various guidelines offered by the American Telemedicine Association on the use of teletechnology in health care delivery, various psychology-specific considerations must be addressed. Preliminary suggestions for ethical teleneuropsychology practice are discussed by Grosch, Gottlieb, and Cullum (2011) and include some of the following points.

First, The APA Ethics Code, under “Use of Assessments” (Standard 9.02) states:

9.02(a). Psychologists administer, adapt, score, interpret, or use assessment techniques, interviews, tests, or instruments in a manner and for purposes that are appropriate in light of the research on or evidence of the usefulness and proper application of the techniques.

9.02(b). Psychologists use assessment instruments whose validity and reliability have been established for use with members of the population tested. When such validity or reliability has not been established, psychologists describe the strengths and limitations of test results and interpretation. ([American Psychological Association, 2002](#), p. 1071)

As such, psychologists are reminded to use care and judgment when looking into providing teleneuropsychological services, as some ethical as well as legal issues arise within the context of this new medium of service provision. Of course, establishing specialty competence in neuropsychology is paramount as a first step, but then becoming aware of the unique aspects of telehealth-based service provision is also necessary.

The American Psychological Association Ethics Code under “Use of Assessments” (Standard 9.02) states that psychologists use assessment techniques “whose validity and reliability have been established for use with members of the population tested. When such validity or reliability has not been established, psychologists describe the strengths and limitations of test results and interpretation” ([American Psychological Association, 2002](#), p. 1071). Thus, it is important that the field of neuropsychology develop research that establishes the equivalency of teleneuropsychological-based evaluations to traditional face-to-face interactions, to the extent that this is appropriate.

### **Licensure and Billing**

It is well known that a state or provincial license is required to practice psychology in the United States and Canada, although within the context of a telehealth-based patient contact, the question arises as to just “where” the practice is occurring. As of 2010, only a few states had laws regarding telehealth services that specifically mentioned psychologists ([American Psychological Association Practice Organization, 2010](#)). Even though variability across licensing jurisdictions on this topic remains, most state psychology boards take the approach that the psychology provider must be licensed in his or her “home” state, but they must also be licensed in the state where the patient is located. In terms of reimbursement for telehealth-based services, Medicare has approved billing of telehealth-based services, including the Neurobehavioral Status Examination, although this is a new development, and most states have yet to develop consistent procedures for handling such claims. Thus, psychologists who want to provide telehealth-based services are advised to consult with their local and regional insurance carriers before setting up a teleneuropsychological practice.

## **Special Considerations Regarding Informed Consent, Privacy, and Confidentiality**

Informed consent is a requirement for neuropsychological practice, and in the case of telehealth-based services, requires some expansion beyond the standard office-based considerations. For example, patients should be informed that information shared via Internet connections may not be completely secure (e.g., there is ongoing debate as to whether commonly used programs such as Skype meet Health Insurance Portability and Accountability Act guidelines) and that some of their information may be inadvertently obtained by other parties. Along these lines, patients need to be made aware that other breaches to privacy and confidentiality may occur within a telehealth context, if third parties are present in the room at either end. It is also important to avoid the involvement of third parties with the patient who might “coach” or assist the patient during teleneuropsychological assessment procedures. Special note should be made if sessions will utilize store-and-forward procedures, which should include mention of how data will be handled and safeguarded (as well as how long and where data will be kept). Full disclosure of such risks is important in this context.

Because there may be a need for some sort of staff involvement at the remote end of a teleneuropsychology encounter, it is important to identify such individuals and make necessary arrangements ahead of time. The American Telemedicine Association (2009) recommends that adequate staff are available to meet patient needs before, during, and after a telehealth encounter, and this may include technical staff for data-transmission problems and/or clinical staff that can assist with any problem behaviors or clinical situations that may merit direct intervention.

### **SUMMARY**

TR, telepsychology, and teleassessment have emerged as promising potential delivery systems for assessment and intervention services. Technological advancements yield increased quality of psychologist–client interactions, greater flexibility in meeting more diverse remote clinical applications, and enhanced security that meets required standards. Longstanding barriers to accessing specialized assessments and increased cost-effectiveness have been targeted in TR and telepsychology outcomes research. More recently, naturalistic assessment and “in vivo” rehabilitation have been posed as potentially more

valuable than traditional clinic or office-based services. Although the evidence for the comparative effectiveness and cost-benefit of TR and telepsychology remains insufficient, data thus far warrant continued clinical and empirical exploration.

## REFERENCES

- American Psychological Association. (2002). Ethical principles of psychologists and code of conduct. *American Psychologist*, 57, 1060–1073.
- American Telemedicine Association. (2009, October). *Practice guidelines for videoconferencing-based telemental health*. Retrieved February 24, 2010, from <http://www.americantelemed.org/files/public/standards/PracticeGuidelinesforVideoconferencing-Based%20TelementalHealth.pdf>
- American Psychological Association Practice Organization. (2010, Summer). *Telehealth: Legal basics for psychologists: Good practice*. Retrieved November, 2010 from <http://www.apapracticecentral.org/>
- Ball, C., & Puffett, A. (1998). The assessment of cognitive function in the elderly using videoconferencing. *Journal of Telemedicine and Telecare*, 4(Suppl. 1), 36–38.
- Ball, C. J., Scott, N., McLaren, P. M., & Watson, J. P. (1993). Preliminary evaluation of a Low-Cost VideoConferencing (LCVC) system for remote cognitive testing of adult psychiatric patients. *British Journal of Clinical Psychology/The British Psychological Society*, 32(Pt. 3), 303–307.
- Ball, C., Tyrrell, J., & Long, C. (1999). Scoring written material from the mini-mental state examination: A comparison of face-to-face, fax and video-linked scoring. *Journal of Telemedicine and Telecare*, 5(4), 253–256.
- Baset, S. A., & Schulzrinne, H. (2004). *An analysis of the Skype peer-to-peer Internet telephony protocol*. Retrieved March 5, 2012, from Cornell University Library <http://arxiv.org/abs/cs/0412017v1>.
- Bashshur, R. L. (2002). Telemedicine/telehealth: An international perspective. *Telemedicine and health care. Telemedicine Journal and Ehealth*, 8(1), 5–12.
- Brandt, J., Welsh, K. A., Breitner, J. C., Folstein, M. F., Helms, M., & Christian, J. C. (1993). Hereditary influences on cognitive functioning in older men. A study of 4000 twin pairs. *Archives of Neurology*, 50(6), 599–603.
- Cooper, R., Fitzgerald, S., Boninger, M., Brienza, D. M., Shapcott, N., Cooper, R., & Flood, K. (2001). Telerehabilitation: Expanding access to rehabilitation expertise. *Proceedings of the IEEE*, 89(8), 1174–1191.
- Cruz, M., Krupinski, E. A., Lopez, A. M., & Weinstein, R. S. (2005). A review of the first five years of the University of Arizona telepsychiatry programme. *Journal of Telemedicine and Telecare*, 11(5), 234–239.
- Cullum, C. M., Weiner, M. F., Gehrman, H. R., & Hynan, L. S. (2006). Feasibility of telecognitive assessment in dementia. *Assessment*, 13(4), 385–390.
- Darkins, A. (2006). Changing the location of care: Management of patients with chronic conditions in Veterans Health Administration using care coordination/home telehealth. *Journal of Rehabilitation Research and Development*, 43(4), vii–xii.
- Debling, D., Amelang, M., Hasselbach, P., & Stürmer, T. (2005). Assessment of cognitive status in the elderly using telephone interviews. *Zeitschrift für Gerontologie und Geriatrie*, 38(5), 360–367.
- Grady, B. J. (2002). A comparative cost analysis of an integrated military telemental healthcare service. *Telemedicine Journal and Ehealth*, 8(3), 293–300.
- Grady, B. J., & Melcer, T. (2005). A retrospective evaluation of telemental healthcare services for remote military populations. *Telemedicine Journal and Ehealth*, 11(5), 551–558.
- Grosch, M. C., Gottlieb, M. C., & Cullum, C. M. (2011). Initial practice recommendations for

- teleneuropsychology. *Clinical Neuropsychologist*, 25(7), 1119–1133.
- Grothus, M., & Sadun, E. (2011). *Taking your iPod touch to the Max*. Berkeley, CA: Apress.
- Hailey, D., Roine, R., Ohinmaa, A., & Dennett, L. (2011). Evidence on the effectiveness of telerehabilitation applications: A systematic review. *Journal of Telemedicine and Telecare*, 17(6), 281–287.
- Hassall, S., Wootton, R., & Guilfoyle, C. (2003). The cost of allied health assessments delivered by videoconference to a residential facility for elderly people. *Journal of Telemedicine and Telecare*, 9(4), 234–237.
- Hildebrand, R., Chow, H., Williams, C., Nelson, M., & Wass, P. (2004). Feasibility of neuropsychological testing of older adults via videoconference: Implications for assessing the capacity for independent living. *Journal of Telemedicine and Telecare*, 10(3), 130–134.
- Hilty, D. M., Luo, J. S., Morache, C., Marcelo, D. A., & Nesbitt, T. S. (2002). Telepsychiatry: An overview for psychiatrists. *CNS Drugs*, 16(8), 527–548.
- Hilty, D. M., Nesbitt, T. S., Kuenneth, C. A., Cruz, G. M., & Hales, R. E. (2007). Rural versus suburban primary care needs, utilization, and satisfaction with telepsychiatric consultation. *Journal of Rural Health*, 23(2), 163–165.
- Hilty, D. M., Yellowlees, P. M., & Nesbitt, T. S. (2006). Evolution of telepsychiatry to rural sites: Changes over time in types of referral and in primary care providers' knowledge, skills and satisfaction. *General Hospital Psychiatry*, 28(5), 367–373.
- Jacobsen, S. E., Sprenger, T., Andersson, S., & Krogstad, J. M. (2003). Neuropsychological assessment and telemedicine: A preliminary study examining the reliability of neuropsychology services performed via telecommunication. *Journal of the International Neuropsychological Society*, 9(3), 472–478.
- Jones, B. N., Johnston, D., Reboussin, B., & McCall, W. V. (2001). Reliability of telepsychiatry assessments: Subjective versus observational ratings. *Journal of Geriatric Psychiatry and Neurology*, 14(2), 66–71.
- Kairy, D., Lehoux, P., Vincent, C., & Visintin, M. (2009). A systematic review of clinical outcomes, clinical processes, healthcare utilization, and costs associated with telerehabilitation. *Disability and Rehabilitation*, 7, 1–21.
- Kennedy, C. A. (2005). The challenges of economic evaluations of remote technical health interventions. *Clinical and Investigative Medicine*, 28(2), 71–74.
- Kirkwood, K. T., Peck, D. F., & Bennie, L. (2000). The consistency of neuropsychological assessments performed via telecommunication and face to face. *Journal of Telemedicine and Telecare*, 6(3), 147–151.
- Kobak, K. A. (2004). A comparison of face-to-face and videoconference administration of the Hamilton Depression Rating Scale. *Journal of Telemedicine and Telecare*, 10(4), 231–235.
- Krupinski, E., Nypaver, M., Poropatich, R., Ellis, D., Safwat, R., & Sapci, H. (2002). Telemedicine/telehealth: An international perspective: Clinical applications in telemedicine/telehealth. *Telemedicine Journal and Ehealth*, 8(1), 13–34.
- Kuipers, P., Foster, M., Smith, S., & Fleming, J. (2009). Using ICF-environment factors to enhance the continuum of outpatient ABI rehabilitation: An exploratory study. *Disability and Rehabilitation*, 31(2), 144–151.
- Loh, P. K., Donaldson, M., Flicker, L., Maher, S., & Goldswain, P. (2007). Development of a telemedicine protocol for the diagnosis of Alzheimer's disease. *Journal of Telemedicine and Telecare*, 13(2), 90–94.
- Loh, P. K., Ramesh, P., Maher, S., Saligari, J., Flicker, L., & Goldswain, P. (2004). Can patients with dementia be assessed at a distance? The use of telehealth and standardised assessments. *Internal Medicine Journal*, 34(5), 239–242.
- McCue, M., Fairman, A., & Pramuka, M. (2010). Enhancing quality of life through telerehabilitation. *Physical Medicine and Rehabilitation Clinics of North America*, 21(1), 195–205.
- Menon, A. S., Kondapavalru, P., Krishna, P., Chrismer, J. B., Raskin, A., Hebel, J. R., & Ruskin, P. E. (2001). Evaluation of a portable low cost videophone system in the assessment of depressive symptoms

- and cognitive function in elderly medically ill veterans. *Journal of Nervous and Mental Disease*, 189(6), 399–401.
- Monnier, J., Knapp, R. G., & Frueh, B. C. (2003). Recent advances in telepsychiatry: An updated review. *Psychiatric Services*, 54(12), 1604–1609.
- Montani, C., Billaud, N., Tyrrell, J., Fluchaire, I., Malterre, C., Lauvernay, N., . . . Franco, A. (1997). Psychological impact of a remote psychometric consultation with hospitalized elderly people. *Journal of Telemedicine and Telecare*, 3(3), 140–145.
- Mundt, J. C., Gerals, D. S., & Moore, H. K. (2006). Dial “T” for testing: Technological flexibility in neuropsychological assessment. *Telemedicine Journal and Ehealth*, 12(3), 317–323.
- Norman, S. (2006). The use of telemedicine in psychiatry. *Journal of Psychiatric and Mental Health Nursing*, 13(6), 771–777.
- O’Reilly, R., Bishop, J., Maddox, K., Hutchinson, L., Fisman, M., & Takhar, J. (2007). Is telepsychiatry equivalent to face-to-face psychiatry? Results from a randomized controlled equivalence trial. *Psychiatric Services*, 58(6), 836–843.
- Parikh, M., Grosch, M., Graham, L., Weiner, M., Hynan, L., Shore, J., & Cullum, C. M. (2012). Consumer acceptability of teleneuropsychology. Oral address presented at the 40th Annual Meeting of the International Neuropsychological Society, Montreal.
- Parmanto, B., & Saptono, A. (2009). Telerehabilitation: State of the art from an informatics perspective. *International Journal of Telerehabilitation*, 1(1), 73–84.
- Parmanto, B., Saptono, A., Pramana, G., Pulantara, W., Schein, R. M., Schmeler, M. R., . . . Brienza, D. M. (2010). VISYTER: Versatile and integrated system for telerehabilitation. *Telemedicine and Ehealth*, 16(9), 1–6.
- Pramuka, M., Chase, S. L., Danilko, N., McCue, M., van Roosmalen, L., & Demuth, B. (2006, June). Telerehabilitation and vocational rehabilitation: Supported self-employment using web-based applications. *Proceedings of the RESNA Annual Conference*, Atlanta, GA.
- Rogante, M., Grigioni, M., Cordella, D., & Giacomozzi, C. (2010). Ten years of telerehabilitation: A literature overview of technologies and clinical applications. *NeuroRehabilitation*, 27(4), 287–304.
- Rosen, M. J. (1999). Telerehabilitation. *Neurorehabilitation*, 3, 3–18.
- Ruskin, P. E., Silver-Aylaiian, M., Kling, M. A., Reed, S. A., Bradham, D. D., Hebel, J. R., . . . Hauser, P. (2004). Treatment outcomes in depression: Comparison of remote treatment through telepsychiatry to in-person treatment. *American Journal of Psychiatry*, 161(8), 1471–1476.
- Sadun, E. (2010). Video calling with facetime. In *Taking your iPod touch to the Max*. Berkeley, CA: Apress L. P.
- Scalvini, S., Vitacca, M., Paletta, L., Giordano, A., & Balbi, B. (2004). Telemedicine: A new frontier for effective healthcare services. *Monaldi Archives for Chest Disease = Archivio Monaldi per le malattie del torace/Fondazione clinica del lavoro, IRCCS [and] Istituto di clinica fisiologica e malattie apparato respiratorio, Università di Napoli, Secondo ateneo*, 61(4), 226–233.
- Schmeler, M., Schein, R., McCue, M., & Betts, K. (2009). Telerehabilitation clinical and vocational applications for assistive technology: Research opportunities and challenges. *International Journal of Telerehabilitation*, 1(1), 59–72.
- Schopp, L., Johnstone, B., & Merrell, D. (2000). Telehealth and neuropsychological assessment: New opportunities for psychologists. *Professional Psychology: Research & Practice*, 31(2), 179–183.
- Schopp, L. H., Johnstone, B. R., & Merveille, O. C. (2000). Multidimensional telecare strategies for rural residents with brain injury. *Journal of Telemedicine and Telecare*, 6(Suppl. 1), S146–S149.
- Shore, J. H., Brooks, E., Savin, D. M., Manson, S. M., & Libby, A. M. (2007). An economic evaluation of telehealth data collection with rural populations. *Psychiatric Services*, 58(6), 830–835.
- Shore, J. H., & Manson, S. M. (2004). Telepsychiatric care of American Indian veterans with post-traumatic stress disorder: Bridging gaps in geography, organizations, and culture. *Telemedicine Journal and eHealth*, 10, S64–S69.
- Shores, M. M., Ryan-Dykes, P., Williams, R. M., Mamerto, B., Sadak, T., Pascualy, M., . . . Peskind, E. R.



- (2004). Identifying undiagnosed dementia in residential care veterans: Comparing telemedicine to in-person clinical examination. *International Journal of Geriatric Psychiatry*, 19(2), 101–108.
- Tang, W. K., Chiu, H., Woo, J., Hjelm, M., & Hui, E. (2001). Telepsychiatry in psychogeriatric service: A pilot study. *International Journal of Geriatric Psychiatry*, 16(1), 88–93.
- Vestal, L., Smith-Olinde, L., Hicks, G., Hutton, T., & Hart, J. (2006). Efficacy of language assessment in Alzheimer's disease: Comparing in-person examination and telemedicine. *Clinical Interventions in Aging*, 1(4), 467–471.
- Vilalta-Franch, J., Garre-Olmo, J., López-Pousa, S., Coll-De Tuero, G., & Monserrat-Vila, S. (2007). Telemedicine and dementia: A need for the 21st century. *Revista de Neurologia*, 44(9), 556–561.
- Willer, B., & Corrigan, J. D. (1994). Whatever it takes: A model for community-based services. *Brain Injury*, 8(7), 647–659.
- Winters, J. M. (2002). Telerehabilitation research: Emerging opportunities. *Annual Review Biomedical Engineering*, 4, 287–320.
- World Health Organization. (2001). *International classification of functioning, disability and health (ICF)*. Geneva: Author.
- Ylvisaker, M. (2003). Context-sensitive cognitive rehabilitation after brain injury: Theory and practice. *Brain Impairment*, 4(1), 1–16.
- Zaylor, C., Whitten, P., & Kingsley, C. (2000). Telemedicine services to a county jail. *Journal of Telemedicine and Telecare*, 6(Suppl. 1), S93–S95.



## Index

## academics

academic functioning, assessment of, [212](#)

of child with brain injury, [30–31](#)

skills, [224–225](#)

*N*-acetyl aspartate (NAA), [239](#)

acetylcholinesterase inhibitors, for aphasia, [75](#)

AChE inhibitors (AChEIs), for memory deficits, [100](#)

acquired brain injury (ABI), [23](#), [25](#)

acute hospitalization to inpatient rehabilitation, [292](#)

inpatient to home transition, [293](#), [294](#), [295](#), [296](#)

and memory deficits, [86–89](#)

interventions for, [93–94](#)

pediatrics with, [94–95](#)

return to productivity, [297](#)

transition from school to work, [299](#)

acquired medical disorders, [41–42](#)

activities of daily living (ADL), [32](#), [45](#), [111](#), [112](#), [115](#), [117](#), [120](#), [168](#), [171](#), [222](#)

activity-dependent plasticity, [30](#)

acute disseminated encephalomyelitis (ADEM), [119](#)

acute recovery, [14](#)

adaptive functioning, assessment of, [224](#)

Adderall, for attention and executive function, [43](#)

S-adenosyl methionine (SAM), for traumatic brain injury, [208](#)

adults

brain injury in, [206–208](#)

vs. children, brain developmental differences, [26](#)

disorders of attention and executive function in, [39–40](#)

memory batteries and measures, [90–91](#)

age

influence on brain injury, [29–31](#), [241](#)

and neurological recovery, [15–16](#)

and stroke, [163](#)

agraphia, [169](#)

alexia, [169](#)

without agraphia. *See* pure word blindness

Alzheimer's disease (AD), [40](#)

and memory deficits, [87–88](#)

amantadine, for traumatic brain injury, [318–319](#)

American Congress of Rehabilitation Medicine, [91](#), [95](#)

Brain Injury Interdisciplinary Special Interest Group, [93](#)

D-amphetamine, for aphasia, [74](#)

- amphetamines
  - for attention and executive function, [43](#)
- Amsterdam Memory Training Program, [95](#)
- analog reality, [113](#)
- Animal Fluency, [70](#)
- anomia, [169](#)
- anomic aphasia, [61–62](#)
- anosodiaphoria, [173](#)
- anosognosia, [116](#)
  - after stroke, [172](#)
- anosognosia for hemiplegia (AHP), [172](#)
- Anosognosia Questionnaire, [173](#)
- anoxia, neuroimaging and rehabilitation outcome in, [248–249](#)
- anoxic brain injury (ABI), [243](#), [248–249](#)
- anterograde amnesia, [86](#)
- anticholinergics, for memory deficits, [100](#)
- anticonvulsants, impact on attention and executive function, [42](#)
- antiepileptic drugs (AEDs), for seizure disorder, [129](#)
- antihypertensives, for attention and executive function, [44](#)
- antiseizure medications, [128](#)
- Anton’s syndrome, [172](#)
- anxiety, [134](#)
  - educational interventions, [135](#)
  - family support, [136](#)
  - occupational interventions, [135–136](#)
- aphasia
  - anomic, [61–62](#)
  - assessment of, [66–70](#)
  - brain tumors and, [64](#)
  - cerebrovascular accidents and, [63–64](#)
  - clinical presentations of, [67](#)
  - defined, [57](#)
  - disconnection/dissociative
    - conduction, [61](#)
    - transcortical, [60–61](#)
  - infections and, [65](#)
  - malformations and, [63–64](#)

- neurodegenerative processes and, 65–66
- nondominant hemisphere, 63
- primary
  - Broca's, 58–59
  - global, 59–60
  - Wernicke's, 59
- primary progressive, 65–66
- pure word blindness, 63
- pure word deafness, 62
- pure word mutism, 63
- recovery from, 18
- rehabilitation of, 57–75
  - classical schools of aphasia therapy, 71–72
  - computer-based rehabilitation, 73
  - constraint-induced language therapy, 72
  - melodic intonation therapy, 72–73
  - pharmacotherapy, 74–75
  - transcranial direct current stimulation, 73–74
  - transcranial magnetic stimulation, 73–74
- and stroke, 169–170
- temporary impairments and, 66
- traumatic brain injury and, 64

Aphasia Screening Test, 68

aphemia. *See* pure word mutism

arterial ischemic stroke (AIS), 24

arts, for attention and executive function, 48

Asperger's disorder

- and attention and executive function, 39

## assessment

- of academic functioning, 212
  - of aphasia, 66–70
  - of attention and executive function deficits, 42–43, 215–218
  - of cognitive functioning, 212, 213–215
  - of disorders of neglect, 174–175
  - of emotional/behavioral difficulties, 222, 224
  - of intellectual functioning, 213–214
  - of language, 218–219
  - of malingering, 225
  - of memory deficits, 89–91, 220
    - current parameters, 89
    - pediatric and adult memory batteries and measures, 90–91
  - of motivation, 225
  - of perceptual organizational skills, 221–222
  - of semantics, 218
  - of sensorimotor skills, 222
  - of syntax, 218
  - of visuo perceptual skills, 221
  - of visuospatial deficits, 113–114
  - of visuospatial skills, 221
  - See also* neuropsychological assessment
- assistive technology, for memory deficits, 101
- atherosclerotic plaques, 165
- atomoxetine (Strattera), for attention and executive function, 43–44
- atomoxetine, for attention and executive function, 43
- attention, visuospatial, 112
- attentional disorders, 111
- attention and executive function deficits, 37–50
  - acquired medical disorders, 41–42
  - assessment of, 42–43, 215–218
  - conceptual and biological background, 37–38
  - life span disorders, 38–40
  - psychiatric disorders, 40–41
  - rehabilitation interventions, 43–50
- attention deficit hyperactivity disorder (ADHD), 38–40, 138
- attention process training (APT), 3–4, 95



attention training, for attention and executive function, [44](#), [45](#)  
Audioblox, for attention and executive function, [44](#)  
auditory verbal agnosia. *See* pure word deafness autism, [262](#)  
    executive function in, [39](#)  
autoimmune diseases, and attention and executive function, [42](#)  
AutoMinder, for memory deficits, [101](#)

Baddeley, four-component model of working memory, [83](#)  
*Bayley Scales of Infant Development, Third Edition (Bayley-3)*, [215](#)  
Beck Anxiety Inventory (BAI), [189](#)  
Beck Depression Inventory (BDI), [189](#)  
behavioral compensation, for visuospatial deficits, [115](#)  
behavioral differences, in brain, [29](#)  
behavioral interventions, [268](#)  
    *See also* cognitive-behavioral therapy (CBT)  
behavioral scanning, for visuospatial deficits, [116](#)  
Behavior Rating Inventory of Executive Function (BRIEF) checklist, [43](#)  
Behavioural Inattention Test (BIT), [113](#), [117](#)  
Bender-Gestalt II, [113](#)  
Benton Judgment of Line Orientation Test (JLOT), [113](#)  
Benton Visual Form Discrimination Test, [113](#)  
*Benton Visual Retention Test—Fifth Edition (BVRT)*, [91](#)  
bipolar disorder, [137](#)  
    and attention and executive function, [40](#)  
    educational interventions, [138](#)  
    family support, [138–139](#)  
    occupational interventions, [138](#)  
blood-oxygenation-level-dependent (BOLD) contrast, [239](#)  
Booklet Category Test (BCT), [43](#)  
Boston Diagnostic Aphasia Examination (BDAE), [170](#)  
Boston Diagnostic Aphasia Examination-3rd Ed. (BDAE-3), [68](#), [69](#)  
Boston Naming Test (BNT), [68–70](#), [74](#), [170](#), [188](#)

brain

developmental differences in, [26–29](#)

imaging, [244](#)

*Brain Frames*, for attention and executive function, [46](#)

## brain injury

acquired. *See* acquired brain injury (ABI)

in adults, [206–208](#)

approaches to rehabilitation, [31–32](#)

in childhood, [208–210](#)

influence of age and maturation on brain injury, [29–31](#)

nondegenerative forms of, [96–99](#)

pediatric, [95](#)

psychotherapy after. *See* psychotherapy after brain injury

school reintegration after. *See* school reintegration after brain injury severity, assessment of, [213](#)

traumatic. *See* traumatic brain injury (TBI)

Brain Injury Association of America (BIAA), [23](#)

Brain Injury-Interdisciplinary Special Interest Group (BI-ISIG), [71](#)

brain plasticity, structural, [14–15](#)

*Brain Train* system, for attention and executive function, [46](#)

brain tumors, [24–25](#), [209](#), [235–236](#)

and language dysfunction, [64](#)

Broca's aphasia (nonfluent aphasia), [58–59](#)

clinical presentations of, [67](#)

bromocriptine

for aphasia, [74](#)

for traumatic brain injury, [318](#)

for visuospatial deficits, [119](#)

Brown Location Test (BLT), [188](#)

bupropion (Wellbutrin)

for attention and executive function, [44](#)

for depression, [150](#)

California Verbal Learning Test (CVLT)

children's version (CVLT-C), [90–91](#)

Second Edition (CVLT-II), [90–91](#), [187](#)

caloric stimulation, for visuospatial deficits, [116](#)

Cambridge Test of Prospective Memory (CAMPRMPT), [89](#)

Captain's Log, for attention and executive function, [46](#), [47](#)

carbidopa, for traumatic brain injury, [319](#)

carbomazepine, for traumatic brain injury, [259](#)

carbon monoxide (CO) poisoning, [240](#)

Catapres, for attention and executive function, [44](#)

catecholamine systems, in TBI, [317–319](#)

Center for Epidemiologic Studies Depression Scale (CES-D), [175](#)

Centers for Disease Control and Prevention (CDC), [24](#), [101](#)

Central Brain Tumor Registry of the United States (CBTRUS), [24](#)

central nervous system, plasticity in, [14](#)

cerebral hypoxia. *See* hypoxia cerebral vascular accidents (CVAs), [24](#), [162](#), [164](#), [207](#)

neuroimaging and, [242–243](#)

rehabilitation outcome in, [247–248](#)

*Cerebri anatome*, [1](#)

cerebrovascular accidents, and language dysfunction, [63–64](#)

cerebrovascular dementia (CVD), [41](#)

*See also* vascular dementia

chemotherapy, and attention and executive function, [41](#)

childhood

brain injury in, [208–210](#)

cancers, [209–210](#)

injuries, [25–26](#)

early insult vs. later, [25–26](#)

focal vs. diffuse, [25](#)

children  
vs. adults, brain developmental differences, 26  
impact of brain injury on, 205–206  
with visuospatial deficits, rehabilitation of, 119

Children's Coma Scale, 213

Children's Memory Scale (CMS), 90

Children's Orientation and Amnesia Test (COAT), 213

cholinesterase-inhibitors  
for TBI, 321–322

classical conditioning, 86

classical schools of aphasia therapy, 71–72

classroom interventions, 267–268

clinical characteristics, of language disorders, 58

Clinical Evaluation of Language Fundamentals-4th Ed., 68

clinical interview, 211–212

clobazam, for traumatic brain injury, 260

Clock Drawing Test, 113

closed head injuries, 25

Cogmed, for attention and executive function, 45

cognitive assessment, 331  
*See also* teleassessment

cognitive behavioral therapy (CBT), 5–6  
for anxiety, 135, 136  
for attention and executive function, 44  
for depression, 100, 136  
after TBI, 150  
for PTSD, 149  
for stroke, 133

cognitive compensation, for visuospatial deficits, 116–117

cognitive development, 27

cognitive functioning, assessment of, 212, 213–215

cognitive neuropsychology school of aphasia, 72

cognitive overload, and traumatic brain injury, 18

cognitive rehabilitation (CR), 1–3, 185, 203, 225  
computer-assisted, 6  
for language dysfunction, 94  
use of technology in, 6–7

for visuospatial neglect, [113](#)  
Cognitive Remediation Program (CRP), [226](#)  
cognitive reserve, and neurological recovery, [17](#)  
cognitive restoration, for visuospatial deficits, [118](#)  
cognitive stability, [28](#)  
cognitive therapies, for children with visuospatial deficits, [119](#)

- cognitive training
  - for attention and executive function, 44
  - computer-assisted, 47
- collateral sprouting, 14
- Communication Abilities in Daily Living-2nd Ed., 68
- compensation, 14
  - functional, 13–14
  - structural, 14–15
  - for visuospatial deficits, 114–119
- compensatory neurorehabilitation, 32
- compliance, with rehabilitation program, 32–33
- comprehensive/holistic day treatment, 179
- comprehensive inpatient rehabilitation (CIR), 177
- Comprehensive Receptive & Expressive Vocabulary Test-2nd Ed., 68
- Comprehensive Test of Nonverbal Intelligence (CTONI), 214
- computed tomography (CT), 233–234
  - in critical illness, 244
- computer-assisted/technological techniques, for attention and executive function, 44–47
- computer-assisted cognitive training, for attention and executive function, 47
- computer-based rehabilitation, for aphasia, 73
- computerized alertness training, for visuospatial neglect, 116, 120
- computerized exercises, for memory deficits, 96
- computerized rehabilitation, 6
  - skills training programs, 6
- computerized tomography (CT) scan, 146
- conceptual models of cognitive rehabilitation, 7
- Concerta, for attention and executive function, 43
- concrete and abstract thought processing, 27–28
- conduction aphasia, 61
  - clinical presentations of, 67
  - repetition, 61
  - reproduction, 61
- consolidation, 83, 84, 187
- constraint-induced language therapy (CILT), for aphasia, 72, 75
- content of treatment tasks, 4
- Continuous Performance Test-Second Edition (CPT-2)*, 43, 187



cortical blindness, [172](#)  
Controlled Oral Word Association Test (COWAT), [68](#)  
Cookie Theft picture narrative-Part of BDAE-3, [68](#), [69](#), [70](#)  
cranial irradiation (CRT), [210](#)  
critical illness, neuroimaging in, [244–245](#), [249–250](#)  
critical periods, and neurological recovery, [15–16](#)  
cross-specialty collaboration, [303](#)  
    barriers to effective collaborative process, [305–307](#)  
    effective, [304](#)  
    facilitation of, [304–305](#)  
    interdisciplinary model, [303](#)  
    multidisciplinary model, [303](#)  
    post–World War II, [303](#)  
    rehabilitation team  
        effective member of, [307–310](#)  
        redefinition of, [310–312](#)  
curriculum-based assessment, of visuospatial neglect, [113](#)  
Cutting’s Questionnaire, [173](#)

DAI. *See* traumatic axonal injury

Daytrana, for attention and executive function, [43](#)

declarative memory, [85](#)

Defense and Veterans Brain Injury Center, [94](#)

Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, [94](#)

degenerative disorders, and memory deficits, [86–89](#), [96–99](#)

Delis-Kaplan (D-KEF), [43](#), [68](#), [70](#)

dementia

of the Alzheimer type (DAT), 65

with Lewy bodies (DLBs), and memory deficits, 88

multi-infarct, 65

semantic, 65–66

depression, 136

educational interventions, 136–137

family support, 137

and neurological recovery, 17

occupational interventions, 137

after traumatic brain injury, treatment for, 149, 150

unipolar, and attention and executive function, 40–41

*See also* poststroke depression (PSD)

Descartes, 1

desipramine, for traumatic brain injury, 269

developmental differences, in brain, 26–29

functional, 28–29

intellectual, 27–28

neurological, 26

social, emotional, and behavioral differences, 29

development and injury, assessment of, 204

brain injury, impact of, 205–206

considerations with, 210–213

differences in referral questions and presenting problems across, 206–210

domains and measures, 213–226

overview, 205

Dexedrine, for attention and executive function, 43

dextroamphetamine, for traumatic brain injury, 320

diabetes mellitus, 163

*Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition (DSM-IV)*, 175

diaschisis, 14, 166

disconnection/dissociative aphasia syndromes

conduction, 61

transcortical motor, 60–61

*Differential Abilities Scale, Second Edition (DAS-2)*, 113, 214

diffuse head injury, 25

diffusion tensor imaging (DTI), [235–236](#), [246](#)  
tractography, [190](#)

diffusion weighted imaging (DWI), [234–235](#)

disorders of attention and executive function  
in adult and aging, [39–40](#)  
in development, [38–39](#)

domains and measures, in neuropsychological assessment, [213](#), [225](#)  
academic skills, [224–225](#)  
adaptive functioning, [224](#)  
alertness and orientation, [213](#)  
attention, [215–216](#)  
cognitive rehabilitation and psychotherapy, [225–226](#)  
emotion, behavior, and personality, [222–224](#)  
executive functioning, [216–218](#)  
injury severity, [213](#)  
intellectual and overall cognitive functioning, [213–215](#)  
language, speech, and communication, [218–219](#)  
learning and memory, [220–221](#)  
sensorimotor and praxis functions, [222](#)  
visuospatial, visuoperceptual, and perceptual organizational skills, [221–222](#)

domains of life, brain injury impact on, [31](#)

donepezil HCl (Aricept)  
for attention and executive function, [44](#)  
for memory deficits, [100](#)  
for traumatic brain injury, [321–322](#)

L-dopa  
for aphasia, [75](#)  
for traumatic brain injury, [319](#)

dopamine agonists, for visuospatial deficits, [118–119](#)

dopamine enhancer, in TBI, [319–320](#)

Dr. Brain series, for attention and executive function, [46](#)

drama, for attention and executive function, [48](#)

dual-trace theory, [83](#)

dysarthria, [57](#)

dysphasia. *See* aphasia

dysphonia, [57](#)

early brain insult (EBI), 30

*Earobics*, for attention and executive function, 46

education, to patients and their families, 204

educational interventions, 127

- for anxiety, 135
- for bipolar disorder, 138
- for depression, 136–137
- of neurological disorders, 128
- for posttraumatic stress disorder (PTSD), 139–140
- psychiatric problems, 134
- for seizure disorder, 128–129
- for stroke, 132–133
- for suicidal ideation, 141
- for traumatic brain injury, 131

See also occupational interventions

EEG biofeedback, for attention and executive function, 47

elderly, disorders of attention and executive function in, 39–40

electrical stimulation mapping (ESM), 192

electrocorticography (ECoG), 194

electroencephalography (EEG), 128, 189, 238–239

electronic memory aids, 93

emotional/behavioral difficulties, assessment of, 222, 224

emotional differences, in brain, 29

employment, brain injury impact on, 31

*EmPOWER*, for attention and executive function, 46

encoding, 187

engagement, with rehabilitation program, 32–33

## environment

-centered cognitive rehabilitation approaches, 4

home, 136, 264

school, 30, 133, 267

VC, 332

work, 31, 131

environmental compensation, for visuospatial deficits, 115

environmental engineering model, for visuospatial deficits, 115

environmental modification neurorehabilitation, 32

epilepsy, and memory deficits, 89

epilepsy surgery and temporal lobe resection, 185

attention, 187

behavioral retraining, cognitive rehabilitation through, 196–197

cognitive/neurobehavioral morbidity, 194

diffusion tensor imaging (DTI) tractography, 190

electroencephalography, 189

executive functioning, 188

2-[18F]-fluoro-2-deoxy-D-glucose-positron emission tomography, 190

fMRI of language and memory, 191

intellectual functioning, 186–187

intracarotid amobarbital procedure (IAP), 190–191

intracranial subdural electrode recordings and electrical stimulation mapping, 191

language, 188

memory abilities, 187–188

motor tasks, 188–189

neural plasticity and functional reorganization, 195

neurodiagnostic tools, 189

neuropsychological assessment, 186

personality assessment, 189

pharmacological therapy, cognitive rehabilitation through, 197

postoperative language reorganization, 195

postoperative memory reorganization, 195–196

postoperative structural reorganization, 196

postsurgical cognitive rehabilitation, 196

presurgical assessment, 186

proton magnetic resonance spectroscopy, 190

single-photon emission computed tomography (SPECT), 190  
structural MRI, 189  
surgical approaches, 194  
visual spatial, 188  
*See also* seizures

episodic memory, 85, 87, 220  
errorful learning (EF), for memory deficits, 97  
errorless learning (EL), 4, 97  
    for memory deficits, 97–98, 99  
errorless trial training, for visuospatial neglect, 116  
evoked potentials (EP), 239  
executive function deficits (EFDs), 39  
    *See also* attention and executive function deficits  
executive impairment, 94  
exercise, for attention and executive function, 48  
expanded rehearsal technique, for memory deficits, 98  
experience-dependent plasticity, 30  
explicit memory, 85, 97, 220  
Expressive One-Word Picture Vocabulary Test, 68  
Expressive Vocabulary Test-Second Edition, 68  
external aids, enhancing memory function through, 92–93  
eye patching, for visuospatial deficits, 115, 117

Facial Recognition, 188  
falls, 24  
family burden, 262–263  
family-centered service (FCS), 260–261  
family-focused treatment (FFT), for bipolar disorder, 138  
family members, in visuospatial deficits rehabilitation, 120

- family support
  - anxiety, [135](#)
  - bipolar disorder, [138–139](#)
  - depression, [137](#)
  - posttraumatic stress disorder (PTSD), [140](#)
  - seizure disorder, [130](#)
  - stroke, [133](#)
  - suicidal ideation, [141](#)
  - traumatic brain injury, [131–132](#)
- family system, [260–264](#)
  - cultural considerations, [263–264](#)
  - family burden, [262–263](#)
  - family-centered service, [260–261](#)
  - financial burden, [263](#)
  - siblings, [264](#)
- FAS Fluency, [70](#)
- feedback training, for visuospatial neglect, [116](#)
- financial burden, [263](#)
- first-letter mnemonics, [92](#)
- 504 Plan, [265](#)
- fixed battery approaches, [210–211](#)
- flexible battery approaches, [211](#)
- fluent aphasia, [59](#), [67](#), [169](#)
- fluent primary progressive aphasia. *See* semantic dementia
- fluent-progressive nonfluent aphasia. *See* semantic dementia (SD)
- 2-[18F]-fluoro-2-deoxy-D-glucose-positron emission tomography, [190](#)
- fluorodeoxyglucose (FDG), [237](#)
- fluoxetine, for traumatic brain injury, [268](#)
- focal head injury, [25](#)
- Focalin, for attention and executive function, [43](#)
- Freesurfer software, [246](#)
- Friedman (memory assistive technology), for memory deficits, [101](#)
- frontotemporal dementia (FTD), and memory deficits, [88](#)
- Full Scale IQ (FSIQ), [214](#)
- functional compensation, [15](#)
- functional differences, in brain, [28–29](#)
  - lobes, development of, [28–29](#)

physiological differences in white/gray matter, 26  
functional independence measure (FIM), 117, 168  
functional magnetic resonance imaging (fMRI), 239, 247  
    in critical illness, 244  
    of language and memory, 191  
functional neuroimaging, 237  
    electroencephalography (EEG), 238–239  
    functional magnetic resonance imaging (fMRI), 239  
    magnetic resonance spectroscopy (MRS), 239  
    magnetoencephalography (MEG), 238–239  
    positron emission tomography (PET), 237–238  
    single-photon emission computed tomography (SPECT), 237–238  
functional plasticity, 30  
functional school of aphasia therapy, 71–72  
future directions for neuropsychological rehabilitation, 7–8  
  
galantamine, for traumatic brain injury, 321–322  
Galveston Orientation and Amnesia Test (GOAT), 213  
General Ability Composite, 214–215  
Geriatric Depression Scale (GDS), 175  
Glasgow Coma Scale (GCS), 206, 213  
glia, 26  
global aphasia, 59–60  
    clinical presentations of, 67  
glutamate, for memory deficits, 100–101  
“Go/No-Go” test, 187  
goal management training (GMT), 4, 99, 250



Golgi type II cells

gradient recalled echo (GRE), [234](#)

gray matter, physiological differences in, [26](#)

group therapy, for TBI, [151](#)

“growing into deficit” phenomenon, [95](#)

Guafacine, for visuospatial deficits, [119](#)

Halstead-Reitan Neurological Test Battery (HRB), [210](#)

handedness, and neurological recovery, [17](#)

head injury  
  childhood, 25

hemianopsia, right, 63

hemiattentional disorders, 111

hemifield sensory distortions, 111

hemifield suppression, 111

hemineglect, 111, 112, 114

hemorrhagic stroke (HS), 24, 165–166, 207

hemosiderin, detecting, 234

Henschen’s axiom, 15

history, of neuropsychological rehabilitation, 1–2

holistic interventions, 225

holistic neuropsychological rehabilitation, 179

Hooper Visual Organization Test, 113

Huntington’s disease, and memory deficits, 88

hydrocephalus, and attention and executive function, 42

hypertension, 163, 179

hypoperfusion, 166

hypoxia, 24

implicit memory, 85, 86, 89, 97

individualized education plan (IEP), 30–31, 226, 266

individualized training programs, for attention and executive function, 48–50

Individualized Transition Plan (ITP), 266

Individualized Treatment Plan (ITP), 49, 50

Individuals With Disabilities Education Act (IDEA), 30, 265

infections, and language dysfunction, 65

informants, 211

instrumental activities of daily living (IADL), 177

integration, 307–308

intellectual differences, in brain, 27–28  
  cognitive stability, 28  
  concrete and abstract thought processing, 27–28

intellectual functioning, assessment of, 213–214

interferon, for traumatic brain injury, 208

interhemispheric competition theory, 112

internal strategies, enhancing memory function through, 92–93

intracarotid amobarbital procedure (IAP), [190–191](#)  
intracranial subdural electrode recordings, [191](#)  
    and electrical stimulation mapping (ESM) stimulation parameters, [192](#)  
    and standard ESM language tasks, [192–194](#)  
ISAAC, for memory deficits, [101](#)  
ischemic penumbra, [242](#)  
ischemic stroke, [24](#), [164–165](#)  
    pathophysiology of, [242](#)

Judgment of Line Orientation (JLO), [188](#)

Kaufman Brief Intelligence Test (K-BIT), [214](#)  
Kennard Principle, [15](#), [119](#), [205](#)  
kindling, [14–15](#)  
knowledge, [308–309](#)

lamotrigin, for traumatic brain injury, [320](#)

language

assessment of, [218–219](#)

*See also* speech

lead exposure, and attention and executive function, [39](#)

lead poisoning, and attention and executive function, [41](#)

learning, [220–221](#)

left-sided recovery, and neurological recovery, [17](#)

lesion/pathology, differential diagnosis and defining, [239](#)

neuroimaging and cerebral vascular

accidents, [242–243](#)

neuroimaging and traumatic brain injury, [241–242](#)

neuroimaging following anoxia, [243](#)

neuroimaging following critical illness, [244–245](#)

life span  
  disorders, [38–40](#)  
  neurological injuries across, [23–26](#)

lifestyle treatment programs, for attention and executive function, [47–48](#)

limb activation training (LAT), for visuospatial deficits, [115](#)

lingraphica. *See* computer visual communication therapy (cVIC)

lobes, development of, [28–29](#)

long-term memory (LTM), [82](#), [85](#), [194](#)

long-term potentiation, [14](#)

long-term recovery, [14](#)

lopogenic primary progressive aphasia, [66](#)

lupus, and attention and executive function, [42](#)

Luria-Nebraska Neuropsychological Battery (LNNB), [210–211](#)

M1/M4 muscarinic agonists, for memory deficits, [100](#)

magnetic resonance imaging (MRI), [189](#), [234](#)  
  in critical illness, [244](#)

magnetic resonance spectroscopy (MRS), [239](#)  
  in pediatric traumatic brain injury, [247](#)

magnetoencephalography (MEG), [238–239](#)

maladaptive life style factors, [164](#)

malformations, and language dysfunction, [63–64](#)

malingering, assessment of, [225](#)

mammalian memory systems, [85](#)

Manhattan Stroke Registry, [161](#)

Matrices (Wechsler and Raven’s versions), [43](#)

maturation, brain, [25–26](#)  
  influence on brain injury, [29–31](#)

Mazes and Tower of London, [43](#)

medical settings, [258–260](#)  
  care teams, [258–259](#)  
  cultural concerns, [259](#)  
  rehabilitation strategies within medical facility, [259–260](#)

melodic intonation therapy (MIT), for aphasia, [72–73](#)

memantine HCl (Namenda)  
  for attention and executive function, [43](#), [44](#)  
  for memory deficits, [100](#)

## memory

abnormalities of, [86–89](#)

defined, [81–82](#)

groups, [99](#)

intact system, strategies to exploit, [96–99](#)

and stroke, [170–172](#)

student's difficulties with, [129](#)

theory and constructs, [81–86](#)

    modality-specific memory, systems, and subtypes, [84–85](#)

    time-dependent and stage-of-processing models of, [82–84](#)

*See also* memory deficits

- memory abilities
  - nonverbal memory, [188](#)
  - verbal memory, [187](#)
- MemoryClip, for memory deficits, [101](#)
- memory deficits, [81–102](#)
  - assessment of, [89–91](#), [220](#)
  - facilitating residual explicit memory or enhance function through external aids, [92–93](#)
  - future directions, [101–102](#)
  - intervention parameters and measured outcomes, [100](#)
  - pharmacologic augmentation, [100–101](#)
  - rehabilitation of, [91–96](#)
    - intervention methods, [92](#)
- See also* memory
- Memory Glasses, for memory deficits, [101](#)
- memory notebooks, [6](#), [93](#)
- memory prosthetics, [93](#)
- mental imagery training, for visuospatial deficits, [118](#)
- metacognitive strategy instructions (MSI), for attention and executive function, [44](#)
- Metadate CD, for attention and executive function, [43](#)
- methylphenidate, for traumatic brain injury, [268](#), [319–320](#)
- mild traumatic brain injury (mTBI)
  - in adults, [207](#)
  - in childhood, [208](#)
  - and language dysfunction, [94](#)
- mindfulness meditation therapy, [5](#)
- Mini-Mental Status Examination (MMSE), [167](#)
- misoplegia, [173](#)
- Missouri Partnership for Enhanced Delivery of Services (MO-PEDS) program, [259](#)
- mnemonics, [92](#), [95](#)

modafinil

for attention and executive function, [43](#)

for traumatic brain injury, [320](#)

modality-specific memory, [84–85](#)

deficits, after TBI, [87](#)

models, rehabilitation

current models, [3–4](#)

molecular memory, [84](#)

monocular eye patching, for visuospatial deficits, [117](#)

Montreal Cognitive Assessment (MoCA), [167](#)

mood, and neurological recovery, [17](#)

mood disorders, and attention and executive function, [40](#)

“the more knowledgeable other” (MKO), [28](#)



motivation

assessment of, [225](#)

and neurological recovery, [17](#)

motor vehicle accidents (MVAs), [24](#)

*MultiCue*, [73](#)

Mullen Scales of Early Learning, [215](#)

multi-infarct dementia (MID), [65](#)

Multilingual Aphasia Examination (MAE), [68](#), [69](#), [70](#)

multiple neurotransmitter systems, in TBI, [320](#)

multiple sclerosis (MS)

and attention and executive function, [42](#)

neuroimaging in, [246](#)

multiple trace theory (MTT), [84](#)

music therapy, for attention and executive function, [48](#)

nascent revolution, [81](#)

National Adult Reading Test-2nd Ed., [68](#)

National Alliance on Mental Illness (NAMI), [141](#)

National Head Injury Foundation (NHIF), [23](#)

neck muscle vibration (NMV), for visuospatial deficits, [117](#)

## neglect

assessment of disorders of, [174–175](#)

neuropathological mechanisms of, [174](#)

visual and spatial, [173](#)

*See also* hemineglect; visuospatial neglect

NEPSY II, [21](#), [113](#)

neural markers of rehabilitation effectiveness, [246](#)

neural repair, for memory deficits, [101](#)

neuroanatomical networks, [37](#)

neuroanatomy, of language disorders, [58](#)

neurochemical complexities, [37–38](#)

neurocognitive assessment, [331](#)

*See also* teleassessment

neurocognitive domains, [30](#)

neurocognitive enhancement therapy (NET), for memory deficits, [96](#)

neurodegenerative processes, and language dysfunction, [65–66](#)

neuroimaging, [233](#)

as biomarker of injury, [239–245](#)

as biomarker of rehabilitation effectiveness, [245–250](#)

and cerebral vascular accidents, [242–243](#)

following anoxia, [243](#)

following critical illness, [244–245](#)

functional, [237–239](#)

incorporation with neurocognitive techniques, [7](#)

and rehabilitation outcome in anoxia, [248–249](#)

and rehabilitation outcome in cerebral vascular accident, [247–248](#)

and rehabilitation outcome in critical illness, [249–250](#)

and rehabilitation outcome in traumatic brain injury, [246–247](#)

structural, [233–236](#)

and traumatic brain injury, [241–242](#)

neurological differences in brain, between children and adults, [26](#)

neurological disorders, educational and occupational rehabilitation of, [128](#)

seizure disorder, [128](#)

stroke, [132](#)

traumatic brain injury, [130](#)

neurological recovery and plasticity, [13–19](#), [29–30](#)

factors affecting rehabilitation, [15–17](#)

- neuropsychological and neurobehavioral tendencies, [17–18](#)
- principles of, [13–14](#)
- structural compensation, [14–15](#)
- treatment and rehabilitation, [18–19](#)
- neurologic music therapy (NMT), for attention and executive function, [48](#)
- neurology intensive care unit (NICU), [166](#)
- neurons, [26](#)
- Neuro-Page, [93](#)
- neuroplasticity. *See* plasticity
- neuropsychological assessment, [203](#)
  - development and injury, [204](#)
    - considerations with, [210–213](#)
    - differences in referral questions and presenting problems across, [206–210](#)
    - domains and measures, [213–226](#)
    - impact of brain injury, [205–206](#)
    - overview, [205](#)
  - future directions, [226–227](#)
  - rehabilitation setting, [203–204](#)
- Neuropsychological Assessment Battery (NAB), [211](#)
- neuropsychological compensation, for visuospatial deficits, [116](#)

neuropsychological rehabilitation  
criteria for modern approaches, 4–5  
current status of, 2–7  
neuropsychological sequelae, 30

neuropsychology

contribution to cognitive rehabilitation, [5–6](#)

neuropsychonline cognitive rehabilitation therapy (NCRT), for attention and executive function, [45](#)

neurorehabilitation, [32](#)

practitioner traits in. *See* psychotherapy after brain injury

transition process in. *See* transition process in neurorehabilitation

neurosurgical intervention, [185](#)

neurotoxic agents exposure, and attention and executive function, [41](#)

nonassociative learning, [86](#)

nondeclarative memory, [85](#)

nondominant hemisphere aphasias, [63](#)

nonfluent aphasias, [58–59](#), [67](#), [169](#)

nonfluent primary progressive aphasia, [66](#)

non-traumatic brain injury (non-TBI), [23](#)

in children, [206](#), [208](#), [210](#)

nonverbal learning disability (NLD), and ADHD, [39](#)

nonverbal memory, [188](#)

norepinephrine enhancer, in TBI, [319–320](#)

normal pressure hydrocephalus (NPH), and attention and executive function, [42](#)

North American Adult Reading Test, [68](#)

nutrition, for attention and executive function, [48](#)

obesity, [164](#)

observations, [212–213](#)

obsessive-compulsive disorder (OCD), and attention and executive function, [40](#)

occupational interventions  
  anxiety, [135–136](#)  
  bipolar disorder, [138](#)  
  depression, [137](#)  
  of neurological disorders, [128](#)  
  posttraumatic stress disorder (PTSD), [140](#)  
  psychiatric problems, [134](#)  
  seizure disorder, [129–130](#)  
  stroke, [133](#)  
  suicidal ideation, [141](#)  
  traumatic brain injury, [131](#)  
  *See also* educational interventions

oppositional-defiant disorder (ODD), and attention and executive function, [40](#)

optokinetic stimulus (OKS), for visuospatial deficits, [116–117](#)

orientation groups, [99](#)

Orientation Log test (O-Log), [213](#)

paraphasic errors, [169](#)

Parkinson's disease  
  and memory deficits, [88](#)

PASAT (Paced Serial Addition test), [43](#)

Paul Coverdell Stroke Registry, [161](#)

Peabody Picture Vocabulary Test-4th Ed., [68](#)

pediatric brain injury, [95](#)  
  treatment duration for, [100](#)

pediatric memory batteries and measures, [90–91](#)

perceptual organizational skills, assessment of, [221–222](#)

person-centered cognitive rehabilitation approaches, [4](#)

pharmacological rehabilitation, [268–269](#), [315](#)  
  agents and function, [317–322](#)  
  for attention and executive function, [43–44](#)  
  cognitive processes, brain trauma impact on, [315–316](#)  
  cognitive rehabilitation through, [197](#)  
  for memory deficits, [100–101](#)  
  principles of pharmacological treatment, [316–317](#)  
  for visuospatial deficits, [118–119](#)

pharmacotherapy, for aphasia, [74–75](#)

phonological awareness, assessment of, [218](#)  
phonological processing, [218](#)  
physostigmine, for traumatic brain injury, [321](#)  
plasticity, [13–19](#), [29–30](#)  
    activity-dependent, [30](#)  
    in central nervous system, [14](#)  
    experience-dependent, [30](#)  
    functional, [30](#)  
    structural brain, [14–15](#), [30](#)  
    *See also* neurological recovery and plasticity  
positive predictive value (PPV), [176](#)  
positron emission tomography (PET), [237–238](#)  
posterior cerebral artery (PCA), [207](#)  
poststroke depression (PSD), [175–177](#)  
posttraumatic amnesia (PTA), [206–207](#), [213](#)  
posttraumatic stress disorder (PTSD), [139](#)  
    educational interventions, [139](#)  
    family support, [140](#)  
    occupational interventions, [140](#)  
praxis, assessment of, [222](#)  
premorbid education level, and neurological recovery, [17](#)

- primary aphasia
  - Broca's, 58–59
  - global, 59–60
  - Wernicke's, 59
- primary intracerebral hemorrhagic infarcts, 166
- primary progressive aphasia (PPA), 65–66
  - fluent, 65–66
  - logogenic, 66
  - nonfluent, 66
- priming, 86, 99
- principles of rehabilitation, 4
- prismatic adaptation therapy (PAT), for visuospatial deficits, 117
- prism spectacles, for visuospatial deficits, 115
- problems presentation, across development, 206–210
- procedural memory, 85
  - after TBI, 87
- professional identity, 309–310
- Promoting aphasia communication effectiveness (PACE), 71–72
- propranolol, for aphasia, 74
- prosody, 218
- prospective memory, 86, 89
  - impairment, 94
  - after TBI, 87
- proton magnetic resonance spectroscopy, 190
- See also* magnetic resonance spectroscopy (MRS)
- Prozac, for depression, 150
- pruning, 26
- PSSCogRehab, for attention and executive function, 45–46
- psychiatric disorders, 40–41, 134
  - anxiety, 134
    - educational interventions, 135
    - family support, 136
    - occupational interventions, 135–136
  - bipolar disorder, 137
    - educational interventions, 138
    - family support, 138–139
    - occupational interventions, 138



- depression, 136
- educational interventions, 136–137
- family support, 137
- occupational interventions, 137
- posttraumatic stress disorder (PTSD), 139
  - educational interventions, 139
  - family support, 140
  - occupational interventions, 14
- suicidal ideation, 140
  - educational interventions, 141
  - family support, 141
  - occupational interventions, 141
- psychological interventions, 268
- psychotherapist, 276
  - within attributes of, 277–281
    - cognitive capabilities, 279
    - existential qualities, 281
    - narcissistic/ego structures, 279–280
    - personality traits, 277–279
    - relational behaviors, 280–281
  - between attributes of treatment team, 281–284
    - cognitive capabilities, 282
    - existential qualities, 284
    - interpersonal and communication skills, 282–284
  - external influences, 277
  - as supervisor, 284–285
  - as team leader, 284–285
- psychotherapy, 225–226
  - after brain injury, 275
    - case study, 285–287
    - collaborative model of, 276
    - psychotherapist. *See* psychotherapist
  - integration with cognitive rehabilitation, 5, 7
  - in patients with TBI, 150
- pure word blindness, 63
- pure word deafness, 62
- pure word mutism, 63

quantitative neuroimaging techniques, [245](#)

Question, Persuade, and Refer (QPR), [141](#)

radiation treatment, and attention and executive function, [41](#)

reality-orientation groups, [99](#)

Receptive One-Word Picture Vocabulary Test, [68](#)

reentry, into community, [33](#)

referral questions, [206](#)

regenerative sprouting, [14](#)

rehabilitation effectiveness, [245](#)

- neuroimaging and rehabilitation outcome
  - in anoxia, [248–249](#)
  - cerebral vascular accident, [247–248](#)
  - critical illness, [249–250](#)
  - traumatic brain injury, [246–247](#)

Reitan Evaluation of Hemispheric Abilities and Brain Improvement Training (REHABIT), [3](#)

repeatable battery of neuropsychological status (RBANS), [167–168](#)

repetition aphasia, [61](#)

reproduction aphasia, [61](#)

repetitive transcranial magnetic stimulation (rTMS) for visuospatial deficits, [117](#)

research

- on cognitive rehabilitation, [7](#)
- regarding physiological differences in white/ gray matter, [26](#)

restoration, for visuospatial deficits, [118–119](#)

restorative neurorehabilitation, [32](#)

retrieval, [187](#)

retrograde amnesia (RA), [86](#)

retrospective memory, [86](#)

- impairment, [94](#)

Rey Auditory Verbal Learning Test (RAVLT), [90](#), [171](#), [187](#)

Rey-Osterrieth Complex Figure Test (ROCF), [91](#), [113](#), [188](#)

right half-field eye patching, for visuospatial deficits, [117](#)

right-sided recovery, and neurological recovery, [17](#)

rivastigmine, for traumatic brain injury, [321](#)

Rivermead Behavioural Memory Test, [89](#) RBMT-3, [89](#)

scanning therapy, [226](#)

schizophrenia  
  and attention and executive function, [40](#)  
  and memory deficits, rehabilitation for, [95](#)

school reintegration after brain injury, [264–269](#)  
  behavioral interventions, [268](#)  
  classroom interventions, [267–268](#)  
  environment, school, [267](#)  
  504 Plan, [265](#)  
  IDEA 2004, [265](#)  
  IEP, [266](#)  
  ITP, [266](#)  
  pharmaceutical interventions, [268–269](#)  
  psychological interventions, [268](#)

seizure disorder, [128](#)  
  and attention and executive function, [41–42](#)  
  educational interventions, [128–129](#)  
  family support, [130](#)  
  occupational interventions, [129–130](#)

selective norepinephrine reuptake inhibitors (SNRIs), for depression in children, [150](#)

Selective Reminding Test (SRT), [187](#)

selective sensory stimulation, [226](#)

selective serotonin reuptake inhibitors (SSRIs), [260](#)  
  for depression in children, [150](#)

self-identity development, [29](#)

semantic dementia, [65–66](#)

semantic memory, [85](#), [87](#), [220](#)

semantics, assessment of, [218](#)

SenseCam, for memory deficits, [101](#)

sensorimotor skills, assessment of, [222](#)

sensory-based apraxia, [114](#)

sensory stimulation, for visuospatial deficits, [116–117](#)

sentence repetition, [68](#), [70](#)

severe traumatic brain injury (sTBI)  
  in adults, [207](#)  
  in children, [208](#)

sex differences, and neurological recovery, [16–17](#)

- short-term memory (STM) model, 82
- siblings, 264
- simulators, for visuospatial deficits, 118
- single-photon emission computed tomography (SPECT), 190, 237–238
- skill transfer, 48–49
- sleep, for attention and executive function, 47–48
- sleep apnea, 48
- sleep disorders, and attention and executive function, 42
- social cognitive theory (SCT), 141
- social differences, in brain, 29
- spaced retrieval (SR), for memory deficits, 94, 98
- spaced retrieval method, 4
- space remapping training, for visuospatial deficits, 118
- spatial working memory, and visuospatial neglect, 112
- standardized measures, 212, 225
- Stanford-Binet, Fifth Edition (SB5)*, 214
- Stanford-Binet Intelligence Scales-Fifth Edition Visual Spatial Processing subtest*, 113
- stroke, 24, 132, 161
  - acute and postacute assessment, 166–169
  - acute comprehensive inpatient
    - rehabilitation, 177–178
  - and attention and executive function, 41
  - anosognosia and unawareness syndromes after, 172
  - aphasia and, 169–170
  - cognitive and emotional assessment of, 166
  - comprehensive day treatment programs, 178–179
  - economic impact of, 162
  - educational interventions, 132–133
  - epidemiology and clinical characteristics of, 161
  - family support, 133
  - future directions, 179
  - hemorrhagic, 165–166
  - home-based therapies, 178
  - incidence and prevalence of, 161
  - ischemic, 165
  - and language dysfunction rehabilitation, 95

- mechanisms and types of, 164
- memory and, 170–172
- modifiable risk factors of, 163–164
- neglect, assessment of disorders of, 174–175
- neglect, neuropathological mechanisms of, 174
- neuropsychological evaluation of cognitive impairments, 169
- occupational interventions, 133
- outpatient rehabilitation therapies, 178
- poststroke depression (PSD), evaluation of, 175–177
- rehabilitation with stroke survivors, 177
- risk factors for, 162
- stroke, 162
- subacute rehabilitation, 178
- unawareness syndromes after, 172
- visual and spatial neglect, 173
- See also* cerebrovascular accident (CVA)

Stroop Test, 187

- structural brain plasticity, 14–15, 30
- structural compensation, 14–15
- structural neuroimaging, 233
  - computed tomography (CT), 233–234
  - diffusion tensor imaging (DTI), 235–236
  - diffusion weighted imaging (DWI), 234–235
  - magnetic resonance imaging (MRI), 234
- subarachnoid hemorrhage (SAH), 166
- substance abuse, 152
- suicidal ideation, 140–141
  - educational interventions, 141
  - family support, 141
  - occupational interventions, 141
- sustained attention training (SAT), for visuospatial neglect, 116
- Symbol Digit Modality Tests (SDMT), 187
- synapses, 26
- synaptic pruning, 26
- syntax, assessment of, 218
- systemic–dynamic approach, 27
- systems with children and adolescents, 257

family system, [260–264](#)  
medical settings, [258–260](#)  
school reintegration after brain injury, [264–269](#)

tamoxifen (TAM), for traumatic brain injury, [208](#)

Tasks of Executive Function, [43](#)

technology, use in cognitive rehabilitation, [6–7](#)

teleassessment, [332](#)

tele-cognitive rehabilitation, for attention and executive function, [44](#)

telehealth, [327](#)

    technology, [332–334](#)

telemedicine and telerehabilitation, differences between, [327–328](#)

- teleneuropsychology
  - assessment, 331
  - competence, 335–336
  - confidentiality, 336
  - ethical considerations, 335–336
  - informed consent, 336
  - licensure and billing, 336
  - privacy, 336
- telerehabilitation, 7, 327–329
  - competence, 335–336
  - confidentiality, 336
  - empirical evidence for, 329–331
  - ethical considerations, 335–336
  - informed consent, 336
  - licensure and billing, 336
  - privacy, 336
  - technology, 334–335
  - and telemedicine, differences between, 327–328
- temporal lobe epilepsy (TLE), and memory deficits, 89
- temporal lobe resection. *See* epilepsy surgery and temporal lobe resection
  - temporary impairments, and language dysfunction, 66
- Tenex, for attention and executive function, 44
- Test for Auditory Comprehension of Language-3rd Ed., 68
- Test of Memory and Learning—Second Edition (TOMAL-2)*, 90
- Test of Motivation/Malingering (TOMMS), 225
- Test of Nonverbal Intelligence, Third Edition (TONI-3)*, 214
- Test of Visual Motor Integration, 113
- thought processing, concrete/abstract, 27–28
- thrombosis, 165
- time-dependent/stage-of-processing models of memory, 82–84
- Token Test, 68–70
- topiramate, for traumatic brain injury, 260
- Tourette's, 39
- TOVA (Test of Variables of Attention), 43
- traditional language-oriented school of aphasia therapy, 71
- Trail Making Tests, 187
  - Trailmaking B, 43

- transcortical aphasias, [60–61](#)
  - clinical presentations of, [67](#)
- transcutaneous electrical nerve stimulation (TENS), for visuospatial deficits, [117](#)
- transcranial direct current stimulation (tDCS), for aphasia, [73–74](#)
- transcranial magnetic stimulation (TMS), for aphasia, [73–74](#)
- transient ischemic attack (TIA), [163](#), [164](#), [165](#)
- Transition Assistance Program, [295](#)
- transition process in neurorehabilitation, [291](#)
  - acute hospitalization to inpatient rehabilitation, [292–293](#)
  - inpatient to home transition, [293–297](#)
  - return to productivity, [297–298](#)
  - school to work, [298–299](#)
- traumatic axonal injury, [241](#)
- traumatic brain injuries (TBIs), [18](#), [23](#), [24](#), [130](#), [145](#), [205](#), [206](#)
  - acute hospitalization to inpatient rehabilitation, [293](#)
  - in adults, [207](#)
  - and attention and executive function, [41](#)
  - in childhood, [208](#)
  - cognitive rehabilitation treatment, [147–148](#)
  - depression after, [149](#)
  - educational interventions, [131](#)
  - epidemiology and pathophysiology, [145–146](#)
  - family burden, [262–263](#)
  - family support, [131–132](#)
  - inpatient to home transition, [293](#), [294](#), [296](#)
  - internalization techniques, [147](#)
  - and language dysfunction, [64](#)
  - and memory deficits, [86–87](#)
  - metacognitive approaches, [147–148](#)
  - mild (mTBI), [94](#), [207](#), [208](#)
  - neuroimaging and, [241–242](#)
    - rehabilitation outcome in, [246–247](#)
- occupational interventions, [131](#)
  - pharmacological rehabilitation. *See* pharmacological rehabilitation rehabilitation
    - cognitive sequelae and, [147–148](#)



emotional and psychosocial sequelae and, [149–152](#)  
initial admission to, [146](#)  
return to productivity, [297](#)  
risk factors for, [146](#)  
severe (sTBI), [207](#), [208](#)  
suggestions for future, [152](#)  
transition from school to work, [298](#), [299](#)  
trepanation, [1](#)  
tricyclic antidepressants, for attention and executive function, [44](#)  
tumors. *See* brain tumors  
Two Part Picture test, [113](#)

unawareness syndromes after stroke, [172](#)  
unilateral spatial neglect (USN), [173](#)  
U Special Kids (USK), [259](#)

valproic acid, for traumatic brain injury, [259–260](#)  
vanishing cues (VC), for memory deficits, [85](#), [98](#)  
vanishing cues method, [4](#)  
vascular cognitive impairment, [168](#)  
vascular dementia, [168](#)  
    and memory deficits, [88–89](#)  
verbal memory, [187](#)  
vicarious functioning, [15](#)

videoconferencing  
for attention and executive function, 44  
-based interventions, 7

virtual reality (VR) programs, 6–7  
for attention and executive function, 46  
computerized alertness programs, 116  
training, for visuospatial deficits, 118

visual action therapy, for aphasia, 72

visual agnosia, 111

visual arts, for attention and executive function, 48

visual field cuts, 111

visual imagery, 95

visual mnemonics/visual imagery, 92–93

Visual Naming Test, 68

Visual Object and Space Perception Battery, 113

visual scanning training (VST), for visuospatial deficits, 115, 120

Visual Spatial Search Task (VISSTA), 114

visuospatial deficits, 111–121  
assessment of, 113–114  
compensation, 114–119  
future directions, 119–121  
remediation strategies, 114  
sequelae and neuropathology, 111–113

visuospatial memory tests, 91

visuospatial neglect, 112–113

visuospatial skills, assessment of, 221

Vocabulary subtest-Part of the Wechsler intelligence tests, 68

voxel-based morphometry (VBM), 245

Wada test, 190

Wallerian degeneration, 242

Webspiration, 49

Wechsler Abbreviated Test of Intelligence (WASI), 214

*Wechsler Adult Intelligence Scale, Fourth Edition(WAIS-IV)*, 113, 187, 214

*Wechsler Memory Scale—Fourth Edition(WMS-IV)*, 90

Wechsler scales, 43

Wernicke’s aphasia, 169

Wechsler Test of Adult Reading, [68](#), [70](#)

Wernicke's aphasia (fluent aphasia), [59](#)  
clinical presentations of, [67](#)

Western Aphasia Battery, [68](#), [75](#)

white matter

    damage, [243](#)

    physiological differences in, [26](#)

Wide Range Achievement Test-3, [68](#), [70](#)

Wide Range Assessment of Memory and Learning–Second Edition (WRAML-2), [90](#)

Wisconsin Card Sorting Test (WCST), [43](#), [188](#)

Woodcock-Johnson III-Tests of Achievement, [68](#), [70](#)

Woodcock Johnson-III Test of Cognitive Abilities, [113](#)

working memory (WM), [45](#), [82](#), [101](#), [112](#), [220](#)

    four-component model of, [83](#)

Yellow Ribbon Project, [141](#)