**Clinical Handbooks in Neuropsychology** 

# Mark Sherer Angelle M. Sander *Editors*

# Handbook on the Neuropsychology of Traumatic Brain Injury



# Clinical Handbooks in Neuropsychology

Series Editor William B. Barr New York University New York, NY, USA

For further volumes: http://www.springer.com/series/8438

Mark Sherer • Angelle M. Sander Editors

# Handbook on the Neuropsychology of Traumatic Brain Injury



*Editors* Mark Sherer TIRR Memorial Hermann Baylor College of Medicine University of Texas Medical School at Houston Houston, TX, USA

Angelle M. Sander Baylor College of Medicine/Harris Health System Brain Injury Research Center TIRR Memorial Hermann Houston, TX, USA

ISBN 978-1-4939-0783-0 ISBN 978-1-4939-0784-7 (eBook) DOI 10.1007/978-1-4939-0784-7 Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2014938095

#### © Springer Science+Business Media, LLC 2014

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

## Preface

Traumatic brain injury (TBI) is a frequent occurrence affecting approximately 1.7 million persons per year in the United States. Some authors believe that true rates of injury are much higher as persons sustaining mild injuries may not seek medical care and thus may not be captured by surveillance systems. The recent wars in Afghanistan and Iraq have increased interest in TBI diagnosis and treatment due to media reports focusing on TBI in veterans and active duty service members.

TBI can cause a wide range of impairments of motor strength, coordination, and balance as well as sensory deficits in all domains. However, extensive research shows that long-term outcomes for persons with TBI are largely determined by cognitive impairment and neurobehavioral disturbances. Cognitive impairments are seen primarily in memory, speed of cognitive processing, and integrative functions (executive skills). Neurobehavioral sequelae of TBI can range from agitation, irritability, impulsivity, depression, and anxiety to more subtle impairments in self-awareness and social communication. These latter difficulties may be perceived as volitional by family and close others and even by healthcare providers so that persons with injury may be blamed and even shunned for deficits caused by the injuries they have sustained. For these reasons, particularly in the post-acute period, neuropsychologists and other behavioral health and cognitive care providers are well positioned to assess and intervene on key deficits caused by TBI and to improve outcomes for persons with TBI.

The Handbook on the Neuropsychology of Traumatic Brain Injury is the second volume in the Clinical Handbooks in Neuropsychology series edited by William B. Barr following the Handbook on the Neuropsychology of Aging and Dementia edited by Lisa D. Ravdin and Heather L. Katzen. As for the earlier volume, chapter authors were selected based on their knowledge of the current scientific literature on TBI as well as their extensive clinical experience in providing services to persons with TBI. In keeping with the overall purpose of the series, the chapters in this volume are aimed toward providing practical, clinically useful information for neuropsychologists working with persons with TBI. Most chapters contain clinical case examples to illustrate the points made by the authors and, hopefully, to facilitate reader assimilation and implementation of materials presented. The book may also serve as a useful tool for neuropsychology graduate students and fellows in training.

The initial section of the book provides a foundation of knowledge on epidemiology and expected outcomes for persons with TBI. The second section focuses on assessment. The adaptation of familiar neuropsychological assessment for persons with TBI is described, and there is also extensive discussion of brief assessments that can be used with persons early in recovery. Commonly used outcome measures of global functioning, supervision needs, and community participation are reviewed. Finally, there is a detailed description of neuroimaging techniques with illustration of common findings in persons with TBI.

Most of the volume focuses on interventions that are often provided by and/or directed by neuropsychologists. Potential targets of intervention range from disorders of consciousness to cognitive impairments to emotional distress. Interventions targeted to family/close others of persons with TBI are discussed as well as comprehensive post-acute problems that integrate a wide range of interventions in a multidisciplinary setting.

The fourth section addresses special issues seen in child and older adults with TBI. The final section tackles the complex issue of mild TBI. Diagnosis and prediction of clinical outcomes for persons with mild TBI is one of the most controversial areas in current neuropsychological practice. While group studies of consecutive series of civilian cases reveal excellent overall outcomes, a number of investigators have identified subgroups that appear to have greater than expected residual effects of injuries. The causes for long-term residua remain elusive. Ever more sophisticated imaging, electrophysiological, and proteomic studies reveal markers that are associated with the occurrence of mild TBI and, in some cases, with residual symptoms. Still other studies find no association of injury characteristics with residual symptoms focusing instead on pre-injury adjustment. Concern for our returning troops and veterans has only added to the confusion. These three chapters from leading experts in the field will add some clarity for readers and inform reasonable practices for assessment and treatment of persons with mild TBI.

The editors wish to thank TIRR Memorial Hermann and, especially, their co-investigators and staff from the Brain Injury Research Center for tremendous support and commitment to our decades' long research programs on persons with TBI. Several Brain Injury Research Center investigators served as authors for chapters in this volume. We also wish to thank the Physical Medicine and Rehabilitation Medicine Departments of Baylor College of Medicine and the University of Texas Medical School at Houston that have served as our academic homes and provided the physicians who have cared for many of our study participants and served as co-investigators on many of our studies. Our research programs over the years have been primarily funded by the National Institute on Disability and Rehabilitation Research (NIDRR) of the United States Department of Education. Our work included in this book, as well as the work of many of the other authors, was funded largely by NIDRR.

We both wish to thank our own families (MS—Connie, Jonathan, Mallory; AMS—Paul, Julian, Miranda) for putting up with late hours and trips to conferences and grant meetings. Most importantly, we wish to thank thousands of persons with TBI and their families/close others who have donated their time and efforts to our research programs for more than 20 years. We hope that their efforts have been rewarded by the knowledge that research in which they participated has led to new approaches to assessment and intervention that have benefited many persons with TBI.

Houston, TX, USA

Mark Sherer Angelle M. Sander

# Contents

Tarti Infouuction to Traumatic Drain Injury	Part I	Introduction	to Traumatic	<b>Brain Injury</b>
---	--------	--------------	--------------	---------------------

<b>Epidemiology and Societal Impact of Traumatic Brain Injury</b> Tresa Roebuck-Spencer and Alison Cernich	3
<b>Cognitive and Behavioral Outcomes</b> <b>from Traumatic Brain Injury</b> Julie Griffen and Robin Hanks	25
Part II Assessment	
<b>Bedside Evaluations</b> Mark Sherer, Joseph T. Giacino, Matthew J. Doiron, Allison LaRussa, and Sabrina R. Taylor	49
<b>Comprehensive Assessment</b> Thomas F. Bergquist, Maya Yutsis, and Jackie L. Micklewright	77
Outcome Assessment Nicholas J. Pastorek and Tracy L. Veramonti	95
Neuroimaging in Traumatic Brain Injury Elisabeth A. Wilde, Jill V. Hunter, and Erin D. Bigler	111
Part III Intervention	
A Systematic and Evidence-Based Approach to Clinical Management of Patients with Disorders of Consciousness Joseph T. Giacino, Christopher G. Carter, Carrie Charney, Denise Ambrosi, Matthew J. Doiron, Seth Herman, and Timothy Young	139
Behavioral Assessment of Acute Neurobehavioral Syndromes to Inform Treatment Risa Nakase-Richardson and Clea C. Evans	157

Rehabilitation of Memory Problems Associated with Traumatic Brain Injury Angelle M. Sander and Laura M. van Veldhoven	173
Rehabilitation of Attention and Executive Function Impairments Keith D. Cicerone and Kacey Little Maestas	191
Social Communication Interventions Margaret A. Struchen	213
Impaired Self-Awareness Mark Sherer and Jennifer Fleming	233
<b>Emotional Distress Following Traumatic Brain Injury</b> Allison N. Clark	257
<b>Treating and Collaborating With Family Caregivers</b> <b>in the Rehabilitation of Persons with Traumatic Brain Injury</b> Angelle M. Sander	271
<b>Comprehensive Brain Injury Rehabilitation</b> <b>in Post-hospital Treatment Settings</b> James F. Malec	283
Part IV Special Issues	
<b>Pediatric Traumatic Brain Injury:</b> <b>Outcome, Assessment, and Intervention</b> Mary R. Prasad and Linda Ewing-Cobbs	311
Assessment and Treatment of Older Adults with Traumatic Brain Injuries Felicia C. Goldstein and Harvey S. Levin	331
Part V Mild TBI	
<b>Mild Traumatic Brain Injury</b> William B. Barr	347
Malingering in Mild Traumatic Brain Injury Maria Easter Cottingham and Kyle Brauer Boone	371
Special Issues with Mild TBI in Veterans and Active Duty Service Members Heather G. Belanger, Alison J. Donnell, and Rodney D. Vanderploeg	389
Index	413

## Contributors

**Denise Ambrosi, M.S., CCC-SLP** Spaulding Rehabilitation Hospital, Charlestown, MA, USA

William B. Barr, Ph.D., ABPP Department of Neurology, NYU Langone Medical Center, New York, NY, USA

NYU School of Medicine, New York, NY, USA

**Heather G. Belanger, Ph.D., ABPP-Cn** Department of Mental Health and Behavioral Sciences, James A. Haley Veterans Hospital, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA

Department of Psychology, University of South Florida Medical School, Tampa, FL, USA

Thomas F. Bergquist, Ph.D., ABPP-Cn Mayo Clinic College of Medicine, Rochester, MN, USA

Erin D. Bigler, Ph.D., ABPP-Cn Department of Psychology and Neuroscience, Brigham Young University, Provo, UT, USA

The Brain Institute and the Department of Psychiatry, University of Utah, Salt Lake City, UT, USA

**Kyle Brauer Boone, Ph.D.** California School of Forensic Studies, Alliant International University, Los Angeles, CA, USA

**Christopher G. Carter, Psy.D.** Spaulding Rehabilitation Hospital, MA, USA

Harvard Medical School, Boston, MA, USA

MGH Institute for Healthcare Professionals, Boston, MA, USA

**Alison Cernich, Ph.D., ABPP** Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, Washington, DC, USA University of Maryland School of Medicine, Baltimore, MD, USA

**Carrie Charney, M.S., CCC-SLP** Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Keith D. Cicerone, Ph.D., ABPP-Cn Neuropsychology and Cognitive Rehabilitation, JFK-Johnson Rehabilitation Institute, Edison, NJ, USA

Rutgers-Robert Wood Johnson Medical School, New Brunswick, NJ, USA

Allison N. Clark, Ph.D. Baylor College of Medicine, Houston, TX, USA Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA

Maria Easter Cottingham, Ph.D. Private Practice, Los Angeles, CA, USA

**Matthew J. Doiron, B.A.** Neuropsychology Laboratory, Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Alison J. Donnell, Ph.D. Department of Mental Health and Behavioral Sciences, James A. Haley Veterans Hospital, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA

The Henry M. Jackson Foundation for the Advancement of Military Medicine, Rockville, MD, USA

Clea C. Evans, Ph.D. Methodist Rehabilitation Center, Jackson, MS, USA

**Linda Ewing-Cobbs, Ph.D.** Children's Learning Institute, University of Texas Health Science Center, Houston, TX, USA

**Jennifer Fleming, Ph.D.** University of Queensland School of Health and Rehabilitation Sciences, and Princess Alexandra Hospital, Brisbane, QLD, Australia

**Joseph T. Giacino, Ph.D.** Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Department of Physical Medicine and Rehabilitation, Massachusetts General Hospital, Boston, MA, USA

Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA, USA

MGH Institute for Healthcare Professionals, Boston, MA, USA

Felicia C. Goldstein, Ph.D., ABPP-Cn Neuropsychology Program, Department of Neurology, Emory University School of Medicine and Wesley Woods Center on Aging, Atlanta, GA, USA

**Julie Griffen, Ph.D.** Wayne State University School of Medicine, Rehabilitation Institute of Michigan, Detroit, MI, USA

**Robin Hanks, Ph.D., ABPP** Wayne State University School of Medicine, Rehabilitation Institute of Michigan, Detroit, MI, USA

Seth Herman, M.D. Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Harvard Medical School, Boston, MA, USA

**Jill V. Hunter, M.D.** Departments of Radiology and Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA

Department of Pediatric Radiology, Texas Children's Hospital, Houston, TX, USA

Allison LaRussa Neuropsychology Laboratory, Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Harvey S. Levin, Ph.D. Cognitive Neuroscience Laboratory, Departments of Physical Medicine and Rehabilitation, Neurosurgery and Psychiatry, Baylor College of Medicine and the Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA

Kacey Little Maestas, Ph.D. Baylor College of Medicine, Houston, TX, USA Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA

James F. Malec, Ph.D. Physical Medicine and Rehabilitation, Indiana University School of Medicine and Rehabilitation Hospital of Indiana, Indianapolis, IN, USA

Emeritus Professor of Psychology, Mayo Clinic, Rochester, MN, USA

Jackie L. Micklewright, Ph.D. Hennepin County Medical Center, Minneapolis, MN, USA

**Risa Nakase-Richardson, Ph.D.** Department of Mental Health and Behavioral Sciences, James A. Haley Veterans Hospital, Tampa, FL, USA University of South Florida Medical School, Tampa, FL, USA

Nicholas J. Pastorek, Ph.D., ABPP Michael E. DeBakey VA Medical Center, Houston, TX, USA

Mary R. Prasad, Ph.D. Children's Learning Institute, University of Texas Health Science Center, Houston, TX, USA

**Tresa Roebuck-Spencer, Ph.D., ABPP-Cn** Jefferson Neurobehavioral Group, Metairie, LA, USA

University of Oklahoma, Norman, OK, USA

**Angelle M. Sander, Ph.D.** Baylor College of Medicine/Harris Health System, Houston, TX, USA

Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA

Mark Sherer, Ph.D., ABPP, FACRM TIRR Memorial Hermann, Houston, TX, USA

University of Texas Medical School at Houston, Baylor College of Medicine, Houston, TX, USA

Margaret A. Struchen, Ph.D. Department of Psychology/Neuropsychology, TIRR Memorial Hermann, Houston, TX, USA

Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA

**Sabrina R. Taylor, Ph.D.** Neuropsychology Laboratory, Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Rodney D. Vanderploeg, Ph.D., ABPP-Cn Department of Mental Health and Behavioral Sciences, James A. Haley Veterans Hospital, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA

Department of Psychology, University of South Florida Medical School, Tampa, FL, USA

Department of Psychiatry and Neurosciences, University of South Florida, Tampa, FL, USA

Laura M. van Veldhoven, Ph.D. Baylor College of Medicine, Houston, TX, USA

**Tracy L. Veramonti, Ph.D.** Mentis Neuro Rehabilitation, Houston, TX, USA Department of Physical Medicine & Rehabilitation, Baylor College of Medicine, Houston, TX, USA

Department of Psychology, University of Houston, Houston, TX, USA

**Elisabeth A. Wilde, Ph.D.** Departments of Physical Medicine and Rehabilitation, Neurology, and Radiology, Baylor College of Medicine, Houston, TX, USA

Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA

**Timothy Young, M.D.** Spaulding Rehabilitation Hospital, Charlestown, MA, USA

Harvard Medical School, Boston, MA, USA

**Maya Yutsis, Ph.D.** Polytrauma Transitional Rehabilitation Program, VA Palo Alto Health Care System, Palo Alto, CA, USA

Part I

Introduction to Traumatic Brain Injury

## Epidemiology and Societal Impact of Traumatic Brain Injury

#### Tresa Roebuck-Spencer and Alison Cernich

#### Abstract

Traumatic brain injury (TBI) is a leading cause of death and disability in the United States. The most recent statistics from the Centers for Disease Control and Prevention document that an estimated 1.7 million people sustain a TBI annually. Nearly 80 % of these individuals will be treated in the ER and released, and a significant majority of these injuries are estimated to be of mild severity. Falls and motor vehicle accidents are the two most common causes of injury with males showing greater rates of TBI than females. Additional risk factors for TBI include age, socioeconomic status, race/ethnicity, and previous injury history. TBI results in significant economic burden to the individual and society and can have long-lasting and devastating effects on an individual's ability to return to family, social, and occupational roles. This chapter will review the epidemiology and societal impact of TBI with particular focus on the epidemiology of TBI in the general population, in the military, and in sports-related events.

#### Keywords

Incidence • Prevalence • Epidemiology • Military TBI • Sports concussion • Prevention • Risk factors

T. Roebuck-Spencer, Ph.D., ABPP-Cn (⊠) Jefferson Neurobehavioral Group, Metairie, LA, USA

University of Oklahoma, Norman, OK, USA e-mail: tresa\_roebuck@hotmail.com

A. Cernich, Ph.D., ABPP Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, Washington, DC, USA

University of Maryland School of Medicine, Baltimore, MD, USA

Traumatic Brain Injury (TBI) describes an acquired injury to the brain from an external source that results in some alteration of cognitive or behavioral functioning. These effects may be transient, long-lasting, or permanent depending on injury specifics and severity. TBI is a leading cause of death and disability affecting persons of all ages, sexes, races/ethnicities, and incomes [1]. Understanding the epidemiology of TBI is essential for the development, implementation, and

evaluation of programs and policies to reduce and prevent TBI-related deaths and disability [1]. Such population-based statistics are also invaluable to health practitioners treating TBI by providing a context within which to understand the scope of the problem and allowing for appreciation of which individuals or groups are at greatest risk for TBI. This chapter will first review the definition and injury classification systems of TBI. Next, this chapter will discuss the incidence of TBI with a focus on how incidence is affected by age, sex, injury mechanism, and other risk factors. Sports-related concussion and TBI in the military will be discussed separately as these groups are not well represented in traditional epidemiology studies. This chapter will end with discussion of the prevalence, economic burden, and societal impact of TBI.

#### **Definition of Traumatic Brain Injury**

TBI is caused by a sudden external event leading to compromised brain functioning that is not caused by a neurodegenerative or congenital/neurodevelopmental condition. Compromise or injury to the brain is typically defined as a manifestation of some alteration in consciousness which ranges from feeling dazed and confused to loss of consciousness/responsiveness, as in coma.

There are several formal definitions of TBI. Because much of the incidence data presented in this chapter comes from surveillance studies conducted by the Centers for Disease Control and Prevention (CDC), that definition will be highlighted here. The CDC defines TBI as "an occurrence of injury to the head that is documented in a medical record with one of the following conditions attributed to head injury: (1) observed or self-reported decreased level of consciousness, (2) amnesia, (3) skull fracture, or (4) objective neurological or neuropsychological abnormality or diagnosed intracranial lesion" [2]. Although skull fracture is included as evidence of TBI, skull fracture alone does not always result in direct injury to the brain [3].

More recently, in 2009, the Department of Veterans Affairs and Department of Defense (VA/DoD) [4] have proposed a new definition that addresses issues specific to TBI among service members, veterans, and civilians. The VA/ DoD defines TBI as "a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event:"

- Any period of loss of or decreased level of consciousness
- Any loss of memory for events immediately before or after the injury
- Any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.)
- Neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient
- Intracranial lesion.

This definition further specifies that external forces may include the head being struck by an object, the head striking an object, the brain experiencing acceleration/deceleration movement without external trauma to the head, a foreign body penetrating the brain, or forces generated from events such as a blast or explosion.

The severity of TBI is typically conceptualized as ranging from mild to moderate to severe. Mild TBI (mTBI) has been specifically defined by various groups, with the most commonly referenced definition coming from the American Congress of Rehabilitation Medicine (ACRM). ACRM [5] defines mTBI as a physiological disruption of brain function as a result of a traumatic event manifested by at least one of the following: alteration of mental status; loss of consciousness (LOC); or loss of memory or focal neurological deficit, that may or may not be transient where injury severity is defined as mild when the following conditions are met:

- Posttraumatic amnesia (PTA) is less than or equal to 24 h
- Glasgow Coma Scale (GCS) score obtained 30 min or more post-injury is greater than 12
- LOC is less than 30 min

Other groups, including the CDC and the World Health Organization [6, 7], also have provided definitions of mTBI. These definitions have in common that GCS scores must be at least 13, LOC and PTA are brief, and there are no abnormal findings on neuroimaging studies.

#### Classification of Injury Severity

Initial presentation of TBI varies greatly across individuals. This presentation has significant implications for later outcome, and, thus, classification of injury severity is an important indicator for prediction of immediate and long-term outcome. TBI severity is best understood as falling on a continuum from mild to severe and is typically defined by acute injury characteristics such as level or duration of impaired consciousness. Classification of injury severity should not be confused with ultimate outcome at later timepoints in the recovery course. Most commonly, injury severity is determined by GCS scores assessed at or very near the time of injury. The GCS provides a measure of depth of unconsciousness and ranks an individual's responsiveness to stimuli in the following areas: eye opening, verbalization, and motor response [8]. GCS scores range from 3 to 15, with 3 indicating no responsiveness and 15 indicating full and appropriate responsiveness in all measured areas. Typically, individuals with an initial GCS of 8 or less are categorized as having a severe TBI, individuals with a GCS of 9-12 are categorized as having a moderate TBI, and individuals with a GCS of 13 or better are categorized as having had a mTBI [9–11]. Individuals with a mTBI who also show positive findings on neuroimaging (such as a subdural hematoma, etc.) are often categorized as having a complicated mTBI and more often demonstrate longer-term outcomes similar to individuals with moderate TBI [3, 12].

TBI severity can also be determined by the duration of impaired consciousness or the length of time it takes a person to return to a conscious or responsive state. While studies define duration of impaired consciousness in different ways, it is most often defined as the length of time between injury and the point at which an individual reliably and consistently follows commands over two distinct consecutive time points. Terms used to define this transition include time to follow commands, duration of unconsciousness, coma duration, and length of coma. While these terms are often used interchangeably, one should be careful to note possible subtle distinctions in how these terms are defined across individual studies. Although there are no commonly agreed upon classification schemes for time to follow commands, a classification scheme reported by Lezak and colleagues [13, 14] classified an interval of  $\leq 20$  min coma duration as a mild injury, an interval of >20 min and  $\leq 6$  h as a moderate injury, and >6 h as a severe injury.

Similarly, the duration of confusion, most commonly defined by duration of PTA, has been used to determine injury severity. PTA refers to the phase of recovery following TBI during which the patient is alert and responsive, but is acutely confused and disoriented, with very poor attention and poor ability to retain new memories [15, 16]. Duration of PTA can be assessed retrospectively by waiting until the patient is no longer confused and asking him/her to report the first memory that he/she can recall following brain injury [17]. More commonly, duration of PTA is determined prospectively by serial assessment of the patient's attention and disorientation, using measures such as the Galveston Orientation and Amnesia Scale (GOAT) [18] or the Orientation Log [19]. The earliest and most commonly cited criteria to determine injury severity using PTA [20] classifies patients with PTA <1 h as having had slight concussion, patients with PTA of one to 24 h as having had moderate concussion, patients with PTA of 1-7 days as having had severe concussion, and patients with PTA of greater than 7 days as having had very severe concussion. However, it is not uncommon for patients in rehabilitation settings to have PTA durations well beyond 1 week post-injury. Thus, this classification system reaches ceiling levels for patients at the more severe end of the severity spectrum, resulting in great variability of outcome for these individuals and decreased prognostic value of this

Indicator	Mild	Moderate	Moderate severe	Severe	Very severe
Glasgow coma scale (GCS) [9–11]	13-15	9–12		6–8	3–5
Time to follow commands [13, 14]	≤20 min	≤ 6 h		>6 h	
Russell posttraumatic amnesia (PTA) [20]	<1 h	1–24 h		1–7 days	>7 days
Mississippi PTA classification scheme [22, 23]		0–14 days	15–28 days	29-70 days	>70 days

 Table 1
 Classification of injury severity

classification scheme. Several studies have provided newer classification systems for PTA duration that provide better prediction of later functional outcome. For instance, Walker and colleagues [21] found that PTA durations of 4 and 8 weeks were better threshold points in predicting functional outcome. Likewise, a new Mississippi PTA classification scheme examined and validated by Nakase-Richardson and colleagues [22, 23] shows improved prediction of later outcome compared with the original Russell classification system. See Table 1 for summary of injury classification schemes.

While all of these injury indicators are good predictors of later outcome, each has strengths and weaknesses. GCS scores are a good indicator of initial mortality and morbidity and can be obtained immediately after injury [24]. However, particularly if assessed too early following injury, GCS scores may overestimate injury severity in individuals with alcohol or other substance intoxication and are limited by early treatment, such as intubation or medical sedation, or in patients who are aphasic or have facial injuries that limit eye opening or verbalization. Likewise, GCS scores may underestimate injury severity in individuals who present with good alertness early on, but later show neurological deterioration, as in a rapidly developing subdural or epidural hematoma. Although time to follow commands takes into account early complications, it too can be affected by early sedation and the patient has to be monitored closely for an extended period of time to obtain this information.

The primary disadvantages of using time to follow commands and duration of PTA as injury severity indicators are that they are not immediately available for early prediction of outcome, there is no commonly agreed upon classification scheme, they often require close and extended patient monitoring, and this information is often not well documented in medical records. However, both time to follow commands and duration of PTA are generally good predictors of short- and long-term functional outcomes, such as return to work and ability to live independently [25–30].

The prognostic ability of injury severity indicators is limited for several reasons. While early injury indicators are good predictors of outcome for moderate to severe TBI, they are less useful in prediction of outcome for mTBI given that they lack variability and reach ceiling levels at the mild end of the TBI spectrum. Additionally, the type and severity of behavioral and functional outcomes can be dramatically different across individuals who were initially classified within the same level of severity, regardless of which severity indices are used. For instance, an individual with an initial GCS of 5 may have good motor recovery but severe cognitive impairment while another may have residual hemiparesis and spasticity, which can be more disabling than cognitive impairment alone. Finally, classification of injury severity using different indicators in the same patient is not always consistent. For instance, a study by Sherer and colleagues [31] revealed that individuals with TBI whose injury severity was classified by GCS scores were more likely to be classified with a severe TBI than when severity of the same injury was classified by time to follow commands or duration of PTA. For this reason, caution is needed when interpreting or comparing studies that use different classification methods to assign injury

severity, and one should not assume that different injury severity indicators will be consistent within a given individual.

#### Incidence of TBI

In its most recent report on TBI in the United States spanning the years 2002–2006, the CDC reported that an estimated 1,691,481 people (576.8 per 1,000,000) sustain a TBI annually [32]. Of this number, 1,364,797 (465.4 per 100,000), or nearly 80 %, are treated in the emergency department (ED) and released. 275,146 (93.8 per 100,000) individuals sustaining a TBI are hospitalized annually. It is further estimated that 51,538 (17.6 per 100,000) individuals sustaining a TBI annually die, and that TBI is a contributing factor in a third (30.5 %) of all injury-related deaths. These mortality rates are highly consistent with estimates in a separate report from the CDC spanning the years 1997-2007 [1], which report an annual average death rate from TBI of 53,014. Comparison of rates over time revealed increases in the incidence of TBI-related ED visits and hospitalizations [32, 33] particularly in children and older adults [32]. These increases were likely related to increases in the population over that time period and increases in falls, but may also represent an increased public awareness of TBI. TBI-related deaths decreased as much as 8.2 % over time [1, 32, 33], which may be related to increased preventive measures such as seat belt and helmet use [34, 35] and better overall treatment for severe TBI [36]. Incidence of new-onset disability from TBI annually has been estimated to be 80,000-90,000 new cases annually [37]. A more recent projected estimate indicated that incidence of new onset disability may actually be higher, at the rate of over 124,626 new cases per year or 43.3 % of all hospitalized TBI survivors [38].

Incidence rates of TBI vary widely across studies due to methodological differences. A review by Kraus and Chu [39] demonstrated that the incidence of TBI ranged from 92 cases per 100,000 persons to 618 per 100,000, with an average rate of fatal plus nonfatal hospitalized brain injuries estimated at 150 per 100,000 persons per year. Populations sampled from and case definitions of TBI differ across studies leading to variability in results. For instance, a recent study by Leibson et al. [40] found higher incidence rates (558 per 100,000) using recordreview criteria to determine TBI, compared with incidence rates determined by CDC criteria (341 per 100,000). This study demonstrated that the CDC approach may significantly underestimate the true incidence of TBI, as it only included 40 % of cases identified by record review. Although 75 % of these missing cases presented to the emergency department, they were missing the necessary CDC-specified code to be counted as a TBI. Of these, 66 % were symptomatic for TBI.

Due to difficulties with consistent data coding across available databases, the newest incidence rates from the CDC did not address injury severity. However, earlier CDC estimates from the year 2000 documented that, of patients hospitalized due to TBI, over 50 % had mild injuries, 21 % had moderate injuries and 19 % had severe injuries, resulting in approximately 102,500 moderate and severe TBIs per year [41]. These rates do not include patients who died prior to hospitalization. More recently, a Congressional report from the CDC Working Group on mTBI in 2003 estimated that mTBI makes up 75 % of the TBIs in the United States each year [6].

Overall incidence rates of TBI reported by the CDC likely underestimate the true incidence of TBI, and even more so the rates of mTBI, given that these estimates do not include individuals either treated in outpatient settings or who do not present for treatment at all [1]. Estimates suggest that up to one fourth of all persons who sustain a TBI do not seek medical care [42]. It is also possible that individuals may be treated and discharged in the ER without adequate appreciation and documentation of a mTBI injury that occurred in conjunction with other more lifethreatening injuries. This possibility is illustrated in a study by Powell and colleagues [43] that found 56 % of individuals prospectively identified to have a mTBI using a brief scripted interview and medical chart review did not have a mTBI diagnosis in their medical record and



**Fig. 1** Estimated average annual rates (per 100,000 population) of traumatic brain injury-related emergency department visits, by age group and injury mechanism in the United States over the years 2002–2006. Figure adapted with permission from 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

would have been missed by retrospective medical record review. Additionally, the most recent CDC data do not include military personnel who sustained a TBI abroad or who received care for TBI in federal, military, or Veteran Administration hospitals [32]. Given the high impact of TBI in the military, the epidemiology of TBI in military populations will be presented and discussed in detail later in this chapter. Individuals who sustain injuries as a result of sports accidents often experience injuries on the milder end of the spectrum and are most often treated in outpatient settings or do not seek medical treatment at all due to rapidly recovering symptoms. Because these individuals are typically not well-represented in hospital-based studies of incidence, TBI due to sports-related accidents will also be presented and discussed separately.

The leading causes of TBI in the US civilian population are falls (35.2 %) followed by motor vehicle-related injuries (17.3 %), a strike or blow to the head from or against objects (16.5 %), assaults (10 %), and other or unknown causes (21 %) [32]. Falls are the most common cause of TBI with an estimated number of 595,095 fallrelated TBIs annually. Fall-related TBIs are greatest at the extremes of the life span, with the highest rates seen in children less than four and in adults over 75 years of age. In contrast, motor vehiclerelated injuries are the leading cause of TBIs in late adolescence (ages 15-19) and early adulthood (ages 20-24). Assault-related TBIs are also highly represented in the 20–24 year age group [32, 33, 44]. See Fig. 1 for changes in rates of injury mechanism across age. Death from TBI is most commonly a result of firearms (34.8 %) particularly in the age ranges of 15-34 and >75 years, followed by motor-vehicle accidents (31.4 %) particularly in the age range of 15-24 years, and falls (16.7 %) particularly in the age group >75 years [1]. TBIs associated with falls (58.4 %) and firearm injuries (49.9 %) are most likely to be associated with long-term disability [38].

#### **Risk Factors**

Although a brain injury can happen to anyone, some groups are at higher risk than others. Age is an important mediating factor with regard to TBI incidence, with the highest rates of TBI occurring in children under 4 years of age, in adolescents between the ages of 15 and 19, and in adults over 65 [32]. Almost half a million emergency depart-



Fig. 2 Estimated average annual rates (per 100,000 population) of traumatic brain injury by age group in the United States over the years 2002–2006. Figure recreated with permission from 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

ment visits for TBI are made annually by children 14 years of age and younger. On the other end of the life span, adults 75 years and older have the highest rates of TBI-related hospitalization and death. While rates of TBI deaths have been decreasing in younger persons (0-44 years), they have increased significantly in persons  $\geq$ 75 years [1]. See Fig. 2 for age trends in estimated annual TBI rates. There is also evidence that elderly individuals are at risk for worse outcome from TBI than younger individuals [45, 46] and probability of long-term disability as a result of TBI has been shown to increase with age [38]. When injury mechanism is considered, children and older adults are at highest risk for fall-related TBIs, and adolescents and young adults are at highest risk for TBIs as a result of motor vehicle accidents [32].

Sex also represents a risk factor for TBI, with studies universally reporting higher rates of TBI in males than in females [32, 37, 42] at a ratio of approximately 1.6–2.8 [39]. Males account for approximately 59 % of all TBI cases in the United States and have higher rates of TBI than females in all age groups. The highest rates for all TBI-related emergency department visits, hospitalizations, and deaths combined are seen for males younger than 4 years of age [32]. Rates of injury peak in both males and females at both ends of

the lifespan and in the adolescence. Although males continue to outnumber females, injury rates were most similar between males and females for the very young and at the later end of the age range when the most common mechanism of injury was falls. Likewise, both males and females, with males outnumbering females, showed increased rates of injury between the ages of 15–19 years of age, when most TBIs are due to motor vehicle accidents [32]. See Fig. 3 for rates of TBI ED visits in males and females across the lifespan. Higher rates of TBI in males may be explained by men being more frequently exposed to high-risk situations and motor vehicle accidents than women. Although probability of long-term disability following TBI was significantly higher for females (49.5 % compared to 39.9 %) [38], death rates from TBI are reported to be three times higher among males [1]. Additionally, firearm-related TBI suicides were higher among males than females for all race/ethnic and age groups [1].

Risk for TBI appears to be mediated by socioeconomic status and racial/ethnic group. Studies document that average annual numbers of TBIs tend to be higher in families at the lowest income levels [42, 47, 48]. Minority racial/ethnic groups have been shown to sustain higher rates of TBI



**Fig. 3** Estimated average annual rates (per 100,000 population) of traumatic brain injury-related emergency department visits, by age group and sex in the United States over the years 2002–2006. Figure recreated with permission from Faul, M., Xu, L., Wald, M. M., &

[49, 50] and appear to be at higher risk for death following TBI [1]. For both sexes, American Indian/Alaska Natives (AI/AN) showed the highest annual average TBI-related death rates. The second-highest annual average rates of TBIrelated deaths were seen in Blacks, and specifically in Black men. In contrast, Hispanics had the lowest rates of TBI deaths overall for both men and women [1]. When mechanism of injury was considered, rates of firearm-related suicides were particularly high among AI/AN men aged 15-34 and among White men aged  $\geq 65$  years. Rates of firearm-related TBI homicides were also highest among Blacks between the ages of 15 and 34 [1]. AI/AN showed the highest annual average rate of motor vehicle-related TBI deaths and fall-related deaths followed by Whites in both categories. A separate surveillance study spanning the year 1997 reported that Black and AI/AN men had the highest rates of TBI attributable to assault, at approximately four times the rates of White men [51]. In addition to being overrepresented in incidence of TBI, minority racial/ethnic groups have also been reported to have poorer overall psychosocial and functional outcome following TBI

when compared with the majority group [52, 53]. Additional risk factors for TBI are history of alcohol/substance use and history of prior TBIs.

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

Specifically, alcohol consumption has been indicated as a potential risk factor for brain injury, with positive associations seen between blood alcohol concentration (BAC) and risk of injury [54, 55]. In a 14-state CDC TBI surveillance system, it was found that among those with TBI due to motor vehicle incidents, 21 % had documented alcohol use of any level and 12 % had BACs above the legal limit for intoxication. Among those with TBI due to assault, 41 % had documented alcohol use of any level and 23 % had BACs above the legal limit [51]. Prior brain injury poses a separate risk for TBI [56]. The risk of a second TBI for those with past injuries is approximately 2.8–3.0 times that of the general non-injured population with rates for a third injury given two prior injuries increasing to 7.8-9.3 times above the general population [57]. A more recent study reported that 7 % of individuals hospitalized with a TBI had at least one recurrent TBI during the follow-up period [58].

#### Sports- and Recreation-Related TBI

TBIs related to sports and recreation activities are receiving more attention resulting in better monitoring and detection. These injuries are most often mild in nature and are frequently referred to as sports-related concussion in the literature. Though mild in nature, these injuries are often graded by severity according to existing guidelines [59, 60]. Grading systems reflect increasing severity of acute symptoms and alteration of consciousness with Grade I injuries reflecting brief symptom duration, short periods of confusion, and no LOC; Grade II injuries involving brief symptom duration, longer periods of confusion, and very brief or no LOC; and Grade III injuries representing longer duration of symptoms, long periods of confusion, and sustained periods of LOC (greater than 5 min). In recent years, consensus bodies endorsed individualized assessment of concussion severity, determined by measures of symptom recovery, rather than prospective grading of injury according to acute characteristics [61, 62]. Often the term concussion is preferred over mTBI because it is more commonly associated with expectations of transient symptoms and positive recovery [63]. The terms concussion and mTBI are synonymous and will be used interchangeably for the purposes of this chapter.

It is estimated that approximately 300,000 sports-related injuries with LOC occur each year [64]. Estimates rise to between 1.6 and 3.8 million per year when milder injuries without LOC and non-medically treated injuries are considered [33]. When taking into account both organized sports and recreational activities in younger populations, a recent analysis of data from the National Electronic Injury Surveillance System documented 173,285 individuals that were less than 19 years of age were assessed and treated in the emergency department for nonfatal TBI from 2001 to 2009 [65]. This represented 6.5 % of all sports and recreation-related emergency department visits in this age group. Over the study period, the estimated number of TBIs that presented to the emergency department increased by 62 %, with the rate of visits increasing 57 % from 190 per 100,000 persons to 298 per 100,000 [65]. Of interest, though the number of visits increased over the time period, the resultant rate of hospitalizations for TBI did not increase. Activities highly represented in the

sample of injuries were bicycling, football, playground activities, basketball, and soccer. Activities with the greatest proportion of TBI injuries were, in order of risk, horseback riding (15.3 %), ice skating (11.4 %), golfing (11.0 %), all-terrain vehicle riding (10.6 %), and tobogganing/sledding (10.2 %). TBI in younger chilwas more highly associated with dren playground activities or bicycling. TBI in older children (10-19 years of age) was more likely to be associated with organized sports (football for boys; soccer or basketball for girls) or bicycling. Though rates of TBI increased over time, the authors of the study concluded that this was likely due to heightened awareness of TBI in sports and recreation, rather than increased risk or increased rates of participation in recreation or sports.

Recent prospective studies of organized sports at the high school level cite a concussion incidence level of 0.24 per 1,000 athlete exposures [66]. Athlete exposure is defined as one athlete participating in one practice or competition during which he or she is exposed to a possible athletic injury. Overall prevalence of concussive injury as a percentage of total athletic injuries at the high school level is estimated to be 9 % [33]. Concussions at the high school level occur at a higher rate during competitive play than during practice [67]. Specific sports are notable for having a higher risk for concussive injury including football, girls soccer, girls basketball, rugby, ice hockey, and lacrosse [67]. The primary cause of concussion in high school sports is contact with another player, followed by contact with the playing surface or equipment, followed by falls [66–69]. Though football represents the highest rate of injury (40.5 %) of all concussions at the high school level, studies including sports played by both sexes show that women athletes sustain a higher rate of concussions [66, 69]. In high school athletes, headache is the most prominent reported symptom (93.4 %) with amnesia noted in almost a quarter of reported concussions [70]. Gender differences have been noted in symptom presentation with male athletes reporting higher rates of confusion and amnesia and female athletes reporting drowsiness and sensitivity to noise

[68, 70]. Of athletes that suffered a concussion during the reporting period, approximately 20 % had suffered a previous concussion [69]. Concussions represent a greater proportion of sports related injuries at the high school level but happen at a lower rate overall compared with collegiate sports.

Athletes at the high school level are now assessed and monitored by surveillance programs at a higher rate than in previous years [71]. Despite this heightened surveillance, return to play decisions are generally premature compared with available guidelines [60, 72]. A recent study found that male athletes were more likely to return to play 1-2 days after sustaining a grade II concussive injury (12.6 % vs. 5.9 %) and were more likely to return less than 1 day after a grade I injury (22 % vs. 0 %). Though it is not clear from the data why men return more quickly than women to competition or practice, there was no suggestion in the data presented that it was due to more severe injury in female athletes. This trend may reflect a more cautious management of female athletes [71].

There have been reports of death due to blunt trauma to the head in youth sports, but these events were rare [73]. Of 261 youth athletes whose deaths resulted from bodily trauma and vital organ damage during sports participation taken from a 30 year retrospective study, 12 deaths were due to boxing, 10 were due to helmet-to-helmet blows in football, and 16 were due to head trauma in baseball. One hundred and thirty-eight football players were reported to have died secondary to a subdural hematoma during the study period. Of note, 12 % of those players had a previous concussion a few days to 4 weeks prior.

In collegiate sports, TBI trends are similar to high school with a slightly higher rate of injury overall. Football continues to have the highest rate of concussion in collegiate sports with a rate of 11.1 injuries per 1,000 athlete exposures [74]. Injuries were more likely during a game. Similar to the high school data, injuries were most likely to occur from helmet to helmet contact. Men's collegiate hockey has a fairly high rate of practice (5.3 %) and game-related (9 %) concussive injuries, with most concussions resulting from player contact (60.2 %) or from contact with the boards (26.3 %) [75]. Soccer at the college level demonstrates the gender difference rates of concussion in similar sports [69]. Women's soccer has a higher rate of concussive injury than men's both for overall rates of injury as well as for severe injury (6 % in women's vs. 3.9 % in men's) [76, 77]. In lacrosse women again lead men in rate of concussive injury overall (9.8 % of all injuries vs. 8.6 % injuries) [78, 79]. Most often lacrosse injuries are due to a stick to the head or contact with another player. Concussive injuries appear to have increased since 1995, mostly in men's lacrosse [79]. This increase is speculated to be due to changes in helmet design that while increasing mobility and decreasing helmet weight may have decreased dissipation of impact forces to the head [79]. Risk for concussion was not as high in women's field hockey as other sports but continued to be higher during games, with concussions representing 5.4 % of serious game injuries [80]. Concussion rates increased among many of these sports across the study period, but this was generally thought to be due to increased awareness and monitoring, rather than increases in actual injury.

Women's sports, such as gymnastics and cheerleading, are often overlooked as activities that may predispose the athlete to an injury. Rates of injury are relatively low in collegiate gymnastics (0.40 per 1,000 athlete exposures during competition; 0.14 per 1,000 athlete exposures during practice), with most of the injuries secondary to handstands or related moves [81]. For overall rates of injury in gymnastics, concussion represents only 1.7 % of injuries [82]. Concussion injury rates in cheerleading, as surveyed in high school, collegiate, and all star squads, were relatively similar to other women's sports, with concussion representing 4 % of injuries experienced in cheerleading. Injury was most often incurred during partner stunts, pyramids, or tumbling [83]. As cheerleading continues to include more gymnastics and partner stunts, and as participation rates increase, injury rates may continue to trend upward as they did in the most recent study of the sport.

In professional sports, data related to injury rates are not as widely available. The professional sports organizations that have published reports on the incidence of brain injury are the National Football League (NFL), the National Hockey League (NHL), and the Federation Internationale de Football Association (FIFA). The NFL has published a series of articles examining concussion-related data and programs. The rate of concussive injuries during games was estimated as 0.41 per NFL game [84]. The cause of most concussions involved impact from another player's helmet (67.7 %), followed by impact with other body regions of another player (20.9 %) and contact with the ground (11.4 %). The primary symptoms noted after injury were headache (55 %), dizziness (41.8 %), and blurred vision (16.3 %); LOC was noted in only a small number of cases (9.3 %). Players who were most likely to be involved in a concussive event were quarterbacks, wide receivers, tight ends, and defensive secondary players. Repeated concussive injury was noted in 160 players during the study period for this series of reports, with 51 players experiencing three or more concussions during the study [85]. Of players that experienced concussion, almost half returned to play during the same game (49.5 %) [86]. Only 8.1 % of injuries required more than 7 days to return to play [87].

Data from the National Hockey League reflect an incidence rate of 1.8 concussions per 1,000 player hours [88, 89]. Rates of injury declined over the seven season study period, likely due to increased awareness and education, but it is notable that the time lost due to concussion increased over the study period with a median time loss of 6 days per concussion [88]. This likely reflects a stricter adherence to return-to-play protocols with greater attention to adequate rest to prevent further injury.

In professional soccer, FIFA's retrospective analysis of players who suffered head and neck injuries during the course of play documented that 11 % of the 163 injuries identified were concussions [90]. The most common causes of injury were aerial challenges and use of the upper extremity, rather than heading which caused only one injury during the study period. Though incidence appears to be rising in professional sports, authors continue to note that this is likely not due to an increased injury rate but rather to an increased awareness to the issue of concussion and a need to document recovery prior to return to play.

#### TBI in the Military

With over 1.6 million individuals deployed to a combat environment since 2001, there is a significant portion of the population that is placed at high risk for incurring a TBI [91]. Prevalence rates of TBI in Service Members are estimated to be between 10 and 20 % of those who are currently serving in the military [92–94]. Surveillance from the Defense and Veterans Brain Injury Center (DVBIC) reflects 220,430 traumatic brain injuries coded in the military medical record from September of 2001 to the second quarter of 2011 [95]. Though larger numbers have been reported in the literature and in the media, these may be overestimates of the true incidence and prevalence of injury as they reflect screening data for TBI and likely include a number of false positives [96].

Screening data are usually obtained from the Post-Deployment Health Assessment/Post-Deployment Health Re-Assessment (PDHA/ PDHRA) from the DoD or the Veteran's Health Administration's (VHA) TBI Screening Questionnaire [97]. These measures are used to determine if the Service Member or veteran was involved in events that placed them at risk for TBI and if they continue to have symptoms at the time of screening; follow-up evaluations with a provider are used to determine presence of TBI and etiology of current symptoms. As the military continues to develop their care model in theater, there are now mandatory evaluations in place for those who are felt to be at risk for TBI [98], with prescribed algorithms for follow-up care. Data are forthcoming from this effort and have begun to inform military medical leadership regarding the etiology of injuries suffered in theater, time needed for recovery, and casualty rates. Screening data from the PDHA/PDHRA have not been published to date. However, multiple survey

efforts, primarily of Army personnel, find an estimated prevalence rate for mTBI of 10–20 % of Service Members surveyed [92, 99, 100]. There is some question as to whether the finding of continued symptoms associated with an injury event with either loss of, or alteration of, consciousness truly reflects mTBI and not another associated disorder (e.g., PTSD, pain-related disorder, or depression) [92].

Though combat-related or weapon-related TBI is seen as a signature injury in the cohort of Service Members who have served in Iraq and Afghanistan, TBI is actually a significant cause of hospitalization for Service Members prior to the current conflicts and remains a significant cause of hospitalization in the non-deployed population with the rate exceeding that of combat-related TBI (74.6 vs. 50.3 per 100,000 service members) [101].

VHA has screened for TBI in approximately 518,775 veterans of the current conflicts from April of 2007 to March of 2011 who have presented to VA medical facilities [102]. Of that number, 97,000 individuals have screened positive and were referred to secondary level evaluation. Of those, who screened positive, 72,623 individuals were referred for a secondary evaluation during which their symptoms were examined in more detail and full clinical evaluation was performed. Following that secondary evaluation, 40,154, or 7 % of the total of those screened, were found to have a symptom presentation and history consistent with mTBI.

The majority of TBIs diagnosed in the military and in VHA are consistent with mTBI (76 %), with the primary etiology being blastrelated. A blast TBI results from the Service Member being proximal to an explosive, such as an improvised explosive device (IED), rocket propelled grenade, land mine, or other artillery or bombs [103]. There are different levels of blastrelated injury defined in the literature: (1) primary blast is a result of the rapid overpressurization and underpressurization of surrounding air as a result of the shock wave, (2) secondary injury results from blast-related fragments or shrapnel, (3) tertiary injury incurred either from falling debris or the Service Member being propelled into an object or their vehicle being propelled by the explosive, (4) quarternary injury from the associated physical processes that result from detonation such as heat injury or toxic detonation products, and (5) quinary injury, resulting from environmental hazards that may remain after the bomb detonates [103–106]. Following blast-related TBI, the other major causes of combat-related TBI are consistent with the major causes of TBI in the civilian population with motor vehicle accidents or land transport accidents, falls, and sports and recreational injuries rounding out the major causes of TBI within the military population. In those with severe and penetrating TBI, the four most common etiologies are blast, motor vehicle accident, falls, and gunshots to the head or neck [107].

Investigation of TBI in theater-based military treatment facilities (MTFs) is beginning to emerge in the literature [108]. Review of records from OIF by the Naval Health Research Center for the period of March through September 2003 noted ICD-9 codes consistent with TBI-related diagnoses in 115 personnel. Most of the injuries were due to combat activities (71 %) with a smaller proportion related to non-battle injuries (16 %). Seven percent of the injuries were secondary to vehicle accidents. Thirteen percent of TBI patients were killed in action or died of their wounds. Concussion was the most common injury code, especially among the non-battle injuries (94 %). Skull fractures and other head wounds were prominently noted in those wounded in action or killed in action (26–33 %). The majority of injuries were caused by IED (52 %); in those who died, gunshots and mortar rounds made up a larger proportion of this group (40 %). The leading causes of non-combat injuries were blunt trauma and motor vehicle accidents. Most often those who were wounded in action had a higher percentage of other bodily injury with face (50 %) and extremity injuries (31 %) representing the majority of other areas of injury. Two thirds of those in the study were wearing protective equipment, generally a helmet and body armor, most of those injured reported a mean of 2.5 types of protective gear worn at the time of injury. Return to duty rates were relatively high in the population, with 46 % of those wounded in action returning to duty and 67 % of those with non-battle injuries returning to duty. Of those with a mTBI who were discharged from service, 29 % of the discharges were disability-related (not specific necessarily to TBI). Of those with moderate to severe injury, 100 % of discharges from service were disability-related.

In a 10-year study of TBI hospitalizations in the Continental United States (CONUS) or European MTF's conducted with records from 1997 to 2006, 110,392 Service Members had at least one medical encounter for TBI and there were 15,372 hospitalizations for TBI, with falls and land transport accidents representing the primary etiology for injury. Hospitalization rates have increased over the course of the conflict, vary by Service branch and phase of the conflict, and reflect a higher rate of weaponsrelated injuries. The Service Member hospitalized with TBI is generally a younger man, who is at the rank of junior enlisted or non-commissioned officer (E1-E5), and tends to serve in the Army or Marines. In examining the early stages of the conflict, Heltemes and colleagues [109] determined an incident inpatient hospitalization rate for TBI of 10.4 Service Members per 10,000 troop strength (1,213 personnel in total) in either Landstuhl (German-based medical center) or in CONUS Regional Medical Centers. This study found that of the sample, only 3 % died of their injuries during hospitalization. The majority of the diagnoses were intracranial injury without skull fracture (59.7 %), with 39.3 % suffering a fracture of the skull. These data likely represent an underestimate of incident TBI hospitalizations as they did not account for intheater deaths or hospitalization.

More recently, a study of all TBI hospitalizations in the US Army for the period of September 2001 to September of 2007 documented that 46 % of the hospitalizations were for severe TBI, 54 % for moderate, and less than 1 % were for mild [110]. Though 65 % of the severe injuries were related to explosions, almost half of the injuries were related to non-combat causes. Overall about 0.14 % of service members in OEF and 0.31 % of those serving in OIF had TBIrelated hospitalizations [110]. In a separate study of the Army hospitalization rates, Ivins and others [101] found a 105 % increase in TBI hospitalizations in the Army from 2000 to 2006, with a 60-fold increase in those injuries attributed to weapons. Of the 2,959 cases that presented to an Army medical treatment facility, the majority of cases was mild in severity and was associated with extracranial injuries. Finally, studies that have assessed for TBI in those who were hospitalized for other conditions that warranted inpatient treatment noted about 20–30 % have TBI in addition to their other injuries [91, 111].

Data from the VA reflect a high incidence of TBI in addition to other, significant bodily injury [112] in their sub-acute rehabilitation facilities. Of 188 consecutive patients admitted to a VA Polytrauma Rehabilitation Center (PRC) between 2001 and 2006, 93 % were diagnosed with a TBI in addition to their other injuries. In addition, pain disorders and mental health conditions were noted to have a high rate of co-occurrence (100 % and 39 %, respectively).

The continuing challenge for clinicians working with military Service Members with TBI is the high level of co-occurring disorders that are noted in the population, especially mental health difficulties, including posttraumatic stress disorder (PTSD) and pain-related disorders. For example, in a sample of veterans who screened positive for TBI within the VHA, those with clinically confirmed diagnosis of TBI were more likely than those without confirmed TBI to have clinical diagnoses of PTSD, other anxiety disorders, and adjustment disorders [113]. Co-occurring mental health and TBI diagnoses in the VA setting varied by sex. Of those veterans with a confirmed TBI diagnosis, PTSD was the most common co-occurring psychiatric diagnosis, with men more likely to have a PTSD diagnosis than women [114]. Women were two times more likely to have a depression diagnosis and 1.5 times more likely to have PTSD with co-occurring depression. In addition, women were noted to report more severe neurobehavioral symptoms than men. Screening-based survey data reflects a high overlap of TBI and mental health difficulties, especially PTSD, with overlap between the two estimated to be about 30 % of all those who screen positive for TBI [92, 115]. The noted

co-occurrence of PTSD tends to be associated with longer symptom duration following injury, especially in those with mTBI [92, 93].

With respect to other mental health diagnoses, there are fewer empirical studies. Veterans who have a history of clinically diagnosed TBI are 1.55 times more likely to die of suicide than those without TBI history and this rate was further increased in those with milder injuries (1.95) [97]. Concurrent diagnosis of Major Depression was greater in those who died of suicide regardless of severity. Only one study specifically examined the rate of alcohol abuse following TBI in a military cohort and found that there was no relationship between alcohol abuse and TBI when other comorbid psychological health difficulties and demographics were controlled in a comparative model [109]. Finally, pain is a major complaint in this population, with a prevalence rate of pain disorders at 43.1 % based on a meta-analysis of veterans. Though PTSD was thought to potentially mediate the relationship between TBI and pain, TBI continued to demonstrate an independent correlation with pain disorder diagnosis when mental health diagnoses were controlled for in the comparative model [116]. These challenging patients should continue to be approached as individual cases, with an understanding that TBI, in addition to co-occurring mental health, pain, and potential sensory dysfunction, may contribute to the clinical presentation.

#### **Prevalence and Societal Impact**

TBI is the leading cause of death for persons under the age of 44 in the United States [117]. It is estimated that between 3.2 and 5.3 million Americans (1.1-1.7 % of the US population) live with disability due to TBI [33, 118]. Similar to estimates of incidence, these numbers are likely an underestimate because they do not include individuals that did not seek medical care, were treated and released from the ER, or were not treated in hospital settings. Additionally, these estimates do not take into account prevalence of long-term disability in military populations. When considering medical costs and lost productivity, the lifetime cost of TBI in the United States was estimated to be 60 billion dollars annually based on information for the year 2000 [119]. When this estimate is converted to 2009 dollars, the estimated total lifetime cost of TBI is \$221 billion with \$14.6 billion for medical costs, \$69.2 billion for work loss costs, and \$137 billion for value of lost quality of life. Total lifetime costs were highest for males age 25–44 years [63]. These estimates do not take into account the emotional cost of TBI or the indirect impact on families, caregivers, and the community.

The societal impact of TBI is immense and difficult to quantify. TBI frequently results in long-term disability and can adversely affect the lives of TBI survivors, their families, friends, and society as a whole. In addition to medical expenses and economic burden, long-term TBIrelated disability frequently results in major changes in interpersonal roles and relationships, as well as decreased engagement in community activities such as work, school, driving, and leisure activities [120]. This impact is particularly acute for the high proportion of young adult TBI survivors, who may be just beginning to establish social, vocational, and family roles.

TBI survivors commonly face levels of disability that limit their ability to return to previous levels of employment and other productive endeavors. Return to employment is consistently identified as among the poorest areas of psychosocial outcome following TBI [121], with post-injury unemployment estimates ranging from 10 to 70 % [122–124]. It has been estimated that nearly one of five hospitalized TBI survivors has not returned to work 1 year following injury as a result of persisting disability, resulting in total lifetime productivity losses of \$51.2 billion (based on information from the year 2000) [119]. Physical deficits limit return to work less than the presence of cognitive, behavioral, and personality changes [125]. Dikmen and colleagues [28] found that age, education, stability of pre-injury work history, and injury severity were all strongly related to the amount of time it took patients to return to work. More specifically, individuals over the age of 50, those with less than high school education, and those with an unstable pre-injury work history were less likely to return to work and took longer to go back to work than

those in other groups. As expected, individuals with milder injuries went back to work more frequently and sooner than those with more severe injuries.

In addition to diminished productive activity, a large percentage of TBI survivors face loss of or decreased personal independence. Most commonly, individuals who are unable to return to independent living situations following TBI will ultimately reside with a family member or significant other post-injury. It has been documented that almost half of persons with moderate and severe TBI who were living independently prior to injury were living with parents at 1 year post-injury [126]. Individuals with moderate and severe TBI also have a markedly increased risk for institutional (usually nursing home) placement as compared to non-injured controls [126, 127].

Personal and family roles are frequently strained by personality and behavioral changes after TBI. Aggression and disinhibition, as well as impaired social skills, poor self-awareness, and impaired social problem solving, can negatively affect interpersonal relationships. Caregiver subjective burden has been reported to have higher associations with behavioral disruption, than with physical impairments resulting from TBI [128]. Adjustment to the caregiver role is often more difficult for spouses than for parents, given the specific strains that are placed on marriages due to personality changes, increasing dependence, and increasing social isolation. Subsequently, rates of divorce are higher following TBI than in the general population [128]. Attrition of friendships and leisure activities is common following TBI [129], which often results in TBI survivors becoming increasingly dependent on their primary caregivers to meet their social needs. This pattern increases family burden further, as caretakers have less time to restore their social networks leading to increases in their own social isolation [128].

#### Prevention

Awareness of public statistics is particularly helpful for targeting and planning intervention programs. Prevention programs typically seek to prevent or reduce brain injuries by providing public education, implementing specific public health programs, and affecting public policy. Many public health education programs have focused on vehicular safety (e.g., wearing seatbelts, wearing approved helmets, obeying traffic laws) and firearms safety [130], and there is good evidence that bicycle and motorcycle helmets, seatbelts, and airbags reduce severity of brain injury following accidents [131–133]. Such programs may partly account for the declining death rates from TBI due to vehicle-related and firearm injuries [1]. One of the largest prevention programs, the ThinkFirst National Injury Prevention Foundation (http://www.thinkfirst.org) in conjunction with the CDC, provides a wealth of information on reducing risk of TBI from multiple potential causes. Additionally, the CDC Heads Up Program provides pamphlets, fact sheets, and toolkits to increase awareness of TBI, particularly mTBI, with the goal of preventing or mitigating poor outcome from TBI (http://www. cdc.gov/concussion/headsup).

Despite these programs, fall-related deaths have continued to increase [1, 32], especially among older adults. Falls and fall-related TBI can seriously affect the quality of life in older adults and place a large burden on the US Health Care System. The CDC has several campaigns to reduce fall-related TBI and to build awareness of early signs of fall-related TBI in older adults. Modifiable fall risk factors include muscle weakness, gait and balance problems, vision problems, psychoactive medications, and home hazards. Effective fall-prevention programs typically focus on medication management, exercise, vision correction, and home modification, and the CDC provides a publiavailable document reviewing cally and describing evidence-based fall-prevention programs [134].

#### Conclusions

In closing, this chapter illustrates that mortality and morbidity due to TBI are major public health problems in the United States. Given the likelihood of persistent impairments, individuals who are diagnosed with TBI often require ongoing rehabilitation and mental health support. Neuropsychologists often play an important role in the evaluation and treatment of persons with TBI in both of these settings. Understanding the specific population-based statistics for TBI, statistics related to common outcomes, and the overall effect of TBI on the person's quality of life and community functioning are vital to successful evaluation, education, and care of the individual with TBI. In addition, understanding the risk factors for TBI and efforts aimed at prevention allows for a broader role of the neuropsychologist in the community, as an educator and as a resource for special populations and advocacy groups.

Understanding the risks in specific at-risk populations of individuals, as presented in this chapter, allows the neuropsychologist to understand the unique causes of injury and factors that influence outcome from injury. As neuropsychologists increasingly play a role in the evaluation of the injured athlete, appreciation of the rates of injury, common causes, and effect of adherence to return to play guidelines on outcome are paramount to successful decision making. As more and more military service members return from deployment and re-enter the community, the neuropsychologist will likely see increasing numbers of these individuals who suffer with persisting cognitive and neurobehavioral complaints. The appreciation of the specific types of injury these individuals incurred, as well as the likelihood that other, co-occurring disorders may complicate the evaluation or treatment of the individual veteran or service member will allow for the adoption of more effective and efficacious strategies.

Finally, population statistics reflect the challenges that the individual with TBI and their family and caregivers face as they reenter their communities and attempt to resume their work and home life. Understanding the effects on the individual, their caregivers, and their social environment provides a context in which to place recommendations and to plan strategies that optimize the potential for successful reintegration.

#### References

- Coronado, V. G., Xu, L., Basavaraju, S. V., McGuire, L. C., Wald, M. M., Faul, M. D., et al. (2011). Surveillance for traumatic brain injury-related deaths—United States, 1997–2007. *Centers for Disease Control and Prevention Morbidity and Mortality Weekly Report*, 60(5), 1–32.
- Marr, A., & Coronado, V. (Eds.). (2004). Central nervous system injury surveillance data submission standards—2002. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery*, 27(3), 422–428.
- Department of Veterans Affairs/Department of Defense. (2009). VA/DoD clinical practice guideline for management of concussion/mild traumatic brain injury (mTBI), Version 1.0 2009.
- American Congress of Rehabilitation Medicine. (1993). Definition of mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 8, 86–87.
- National Center for Injury Prevention and Control. (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.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., et al. (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(Suppl), 84–105.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81–84.
- Clifton, G. L., Hayes, R. L., Levin, H. S., Michel, M. E., & Choi, S. C. (1992). Outcome measures for clinical trials involving traumatically brain-injured patients: Report of a conference. *Neurosurgery*, *31*(5), 975–978.
- Hannay, H. J., & Sherer, M. (1996). Assessment of outcome from head injury. In R. K. Narayan, J. E. Wilberger, & J. T. Povlishock (Eds.), *Neurotrauma* (pp. 723–747). New York: McGraw-Hill.
- Levin, H. S., & Eisenberg, H. M. (1991). Management of head injury. Neurobehavioral outcome. *Neurosurgery Clinics of North America*, 2(2), 457–472.
- Dikmen, S., Machamer, J. E., Powell, J. M., & Temkin, N. R. (2003). Outcome 3 to 5 years after moderate to severe traumatic brain injury. *Archives* of *Physical Medicine and Rehabilitation*, 84(10), 1449–1457.
- Lezak, M. D. (1995). Neuropsychological Assessment (3rd ed.). New York: Oxford University Press.
- Lezak, M. D., Howieson, D. B., Loring, D. W., Hannay, H. J., & Fischer, J. S. (2004). *Neuropsychological*

Assessment (4th ed.). New York: Oxford University Press.

- Russell, W. R. (1932). Cerebral involvement in head injury. A study on the examination of two hundred cases. *Brain*, 55, 549–603.
- Symonds, C. P. (1937). Mental disorder following head injury. *Proceedings of the Royal Society of Medicine*, 30, 1081–1094.
- Symonds, C. P., & Russell, W. R. (1943). Accidental head injuries: Prognosis in service patients. *Lancet*, *1*, 7–10.
- 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.
- Jackson, W. T., Novack, T. A., & Dowler, R. N. (1998). Effective serial measurement of cognitive orientation in rehabilitation: The orientation log. *Archives of Physical Medicine and Rehabilitation*, 79(6), 718–720.
- Russell, W. R., & Smith, A. (1961). Post-traumatic amnesia in closed head injury. *Archives of Neurology*, 5, 4–17.
- Walker, W. C., Ketchum, J. M., Marwitz, J. H., Chen, T., Hammond, F., Sherer, M., et al. (2010). A multicentre study on the clinical utility of post-traumatic amnesia duration in predicting global outcome after moderate-severe traumatic brain injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 81*(1), 87–89.
- Nakase-Richardson, R., Sherer, M., Seel, R. T., Hart, T., Hanks, R., Arango-Lasprilla, J. C., et al. (2011). Utility of post-traumatic amnesia in predicting 1-year productivity following traumatic brain injury: Comparison of the Russell and Mississippi PTA classification intervals. *Journal of Neurology, Neurosurgery, and Psychiatry, 82*(5), 494–499.
- 23. Nakase-Richardson, R., Sepehri, A., Sherer, M., Yablon, S. A., Evans, C., & Mani, T. (2009). Classification schema of posttraumatic amnesia duration-based injury severity relative to 1-year outcome: Analysis of individuals with moderate and severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 90(1), 17–19.
- Wardlaw, J. M., Easton, V. J., & Statham, P. (2002). Which CT features help predict outcome after head injury? *Journal of Neurology, Neurosurgery, and Psychiatry*, 72(2), 188–192; Discussion 51.
- 25. Sherer, M., Sander, A. M., Nick, T. G., High, W. M., Jr., Malec, J. F., & Rosenthal, M. (2002). Early cognitive status and productivity outcome after traumatic brain injury: Findings from the TBI model systems. *Archives of Physical Medicine and Rehabilitation*, 83(2), 183–192.
- 26. Dikmen, S., McLean, A., Jr., Temkin, N. R., & Wyler, A. R. (1986). Neuropsychologic outcome at one-month postinjury. *Archives of Physical Medicine* and Rehabilitation, 67(8), 507–513.

- Dikmen, S., & Machamer, J. (1995). Neurobehavioral outcomes and their determinants. *The Journal of Head Trauma Rehabilitation*, 10, 74–86.
- Dikmen, 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.
- Ellenberg, J. H., Levin, H. S., & Saydjari, C. (1996). Posttraumatic Amnesia as a predictor of outcome after severe closed head injury. Prospective assessment. Archives of Neurology, 53(8), 782–791.
- Dikmen, S., Ross, B. L., Machamer, J. E., & Temkin, N. R. (1995). One year psychosocial outcome in head injury. *Journal of International Neuropsychological Society*, 1(1), 67–77.
- Sherer, M., Struchen, M. A., Nakase-Thompson, R., & Yablon, S. A. (2005). Comparison of indices of severity of traumatic brain injury. *Journal of International Neuropsychological Society*, 11(S1), 48.
- 32. 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.
- 33. Langlois, J. A., Rutland-Brown, W., & Wald, M. M. (2006). The epidemiology and impact of traumatic brain injury: A brief overview. *The Journal of Head Trauma Rehabilitation*, 21(5), 375–378.
- 34. Braver, E. R., Ferguson, S. A., Greene, M. A., & Lund, A. K. (1997). Reductions in deaths in frontal crashes among right front passengers in vehicles equipped with passenger air bags. *Journal of the American Medical Association*, 278(17), 1437–1439.
- Thompson, R. S., Rivara, F. P., & Thompson, D. C. (1989). A case–control study of the effectiveness of bicycle safety helmets. *New England Journal of Medicine*, 320(21), 1361–1367.
- 36. Faul, M., Wald, M. M., Rutland-Brown, W., Sullivent, E. E., & Sattin, R. W. (2007). Using a costbenefit analysis to estimate outcomes of a clinical treatment guideline: Testing the Brain Trauma Foundation guidelines for the treatment of severe traumatic brain injury. *Journal of Trauma*, 63(6), 1271–1278.
- Thurman, D. J., Alverson, C., Dunn, K. A., Guerrero, J., & Sniezek, J. E. (1999). Traumatic brain injury in the United States: A public health perspective. *The Journal of Head Trauma Rehabilitation*, 14(6), 602–615.
- 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. *The Journal of Head Trauma Rehabilitation*, 23(2), 123–131.
- Kraus, J. F., & Chu, L. D. (2005). Epidemiology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 3–26). Washington, DC: American Psychiatric Publishing, Inc.

- 40. Leibson, C. L., Brown, A. W., Ransom, J. E., Diehl, N. N., Perkins, P. K., Mandrekar, J., et al. (2011). Incidence of traumatic brain injury across the full disease spectrum: A population-based medical record review study. *Epidemiology*, 22(6), 836–844.
- Thurman, D. J., & Guerrero, J. (1999). Trends in hospitalization associated with traumatic brain injury. *Journal of the American Medical Association*, 282(10), 954–957.
- Sosin, D. M., Sniezek, J. E., & Thurman, D. J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury*, 10(1), 47–54.
- Powell, J. M., Ferraro, J. V., Dikmen, S. S., Temkin, N. R., & Bell, K. R. (2008). Accuracy of mild traumatic brain injury diagnosis. *Archives of Physical Medicine and Rehabilitation*, 89(8), 1550–1555.
- Rutland-Brown, W., Langlois, J. A., Thomas, K. E., & Xi, Y. L. (2006). Incidence of traumatic brain injury in the United States, 2003. *The Journal of Head Trauma Rehabilitation*, 21(6), 544–548.
- Howard, M. A., 3rd, Gross, A. S., Dacey, R. G., Jr., & Winn, H. R. (1989). Acute subdural hematomas: An age-dependent clinical entity. *Journal of Neurosurgery*, 71(6), 858–863.
- 46. Rothweiler, B., Temkin, N. R., & Dikmen, S. S. (1998). Aging effect on psychosocial outcome in traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 79(8), 881–887.
- 47. Kraus, J. F., Fife, D., Ramstein, K., Conroy, C., & Cox, P. (1986). The relationship of family income to the incidence, external causes, and outcomes of serious brain injury, San Diego County, California. *American Journal of Public Health*, *76*(11), 1345–1347.
- Whitman, S., Coonley-Hoganson, R., & Desai, B. T. (1984). Comparative head trauma experiences in two socioeconomically different Chicago-area communities: A population study. *American Journal of Epidemiology*, 119(4), 570–580.
- Cooper, K. D., Tabaddor, K., Hauser, W. A., Shulman, K., Feiner, C., & Factor, P. R. (1983). The epidemiology of head injury in the Bronx. *Neuroepidemiology*, 2, 70–88.
- 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, National Center for Injury Prevention and Control.
- Langlois, J. A., Kegler, S. R., Butler, J. A., Gotsch, K. E., Johnson, R. L., Reichard, A. A., et al. (2003). Traumatic brain injury-related hospital discharges. Results from a 14-state surveillance system, 1997. *MMWR Surveillance Summaries*, 52(4), 1–20.
- Williams Gary, K., Arango-Lasprilla, J. C., & Stevens, L. F. (2009). Do racial/ethnic differences exist in post-injury outcomes after TBI? A comprehensive review of the literature. *Brain Injury*, 23(10), 775–789.
- Arango-Lasprilla, J. C., & Kreutzer, J. S. (2010). Racial and ethnic disparities in functional, psychoso-

cial, and neurobehavioral outcomes after brain injury. *The Journal of Head Trauma Rehabilitation*, 25(2), 128–136.

- 54. Smith, G. S., & Kraus, J. F. (1988). Alcohol and residential, recreational, and occupational injuries: A review of the epidemiologic evidence. *Annual Review of Public Health*, 9, 99–121.
- 55. Waller, P. F., Stewart, J. R., Hansen, A. R., Stutts, J. C., Popkin, C. L., & Rodgman, E. A. (1986). The potentiating effects of alcohol on driver injury. *Journal of the American Medical Association*, 256(11), 1461–1466.
- Salcido, R., & Costich, J. F. (1992). Recurrent traumatic brain injury. *Brain Injury*, 6(3), 293–298.
- Annegers, J. F., Grabow, J. D., Kurland, L. T., & Laws, E. R., Jr. (1980). The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota, 1935–1974. *Neurology*, 30(9), 912–919.
- 58. Saunders, L. L., Selassie, A. W., Hill, E. G., Nicholas, J. S., Horner, M. D., Corrigan, J. D., et al. (2009). A population-based study of repetitive traumatic brain injury among persons with traumatic brain injury. *Brain Injury*, 23(11), 866–872.
- Cantu, R. C. (1998). Return to play guidelines after a head injury. *Clinics in Sports Medicine*, 17(1), 45–60.
- American Academy of Neurology. (1997). Practice parameter: The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology*, 48(3), 581–5.
- 61. Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Johnston, K., Kelly, J., et al. (2002). Summary and agreement statement of the First International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. *British Journal of Sports Medicine*, 36(1), 6–10.
- 62. McCrory, P., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J., et al. (2005). Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *British Journal of Sports Medicine*, 39(4), 196–204.
- Langlois Orman, J. A., Kraus, J. F., Zaloshnja, E., & Miller, T. (2011). Epidemiology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury*. Washington, DC: American Psychiatric Publishing.
- 64. Thurman, D. J., Branche, C. M., & Sniezek, J. E. (1998). The epidemiology of sports-related traumatic brain injuries in the United States: Recent developments. *The Journal of Head Trauma Rehabilitation*, 13(2), 1–8.
- 65. Centers for Disease Control and Prevention. (2011). Nonfatal traumatic brain injuries related to sports and recreation activities among persons aged ≤19 Years— United States, 2001–2009. Morbidity and Mortality Weekly Report (MMWR), 60(39), 1337–1342.
- 66. Lincoln, A. E., Caswell, S. V., Almquist, J. L., Dunn, R. E., Norris, J. B., & Hinton, R. Y. (2011). Trends in concussion incidence in high school

sports: A prospective 11-year study. American Journal of Sports Medicine, 39(5), 958–963.

- Halstead, M. E., & Walter, K. D. (2010). American Academy of Pediatrics. Clinical report—Sportrelated concussion in children and adolescents. *Pediatrics*, 126(3), 597–615.
- Frommer, L. J., Gurka, K. K., Cross, K. M., Ingersoll, C. D., Comstock, R. D., & Saliba, S. A. (2011). Sex differences in concussion symptoms of high school athletes. *Journal of Athletic Training*, 46(1), 76–84.
- Gessel, L. M., Fields, S. K., Collins, C. L., Dick, R. W., & Comstock, R. D. (2007). Concussions among United States high school and collegiate athletes. *Journal of Athletic Training*, 42(4), 495–503.
- Meehan, W. P., 3rd, d'Hemecourt, P., & Comstock, R. D. (2010). High school concussions in the 2008– 2009 academic year: Mechanism, symptoms, and management. *American Journal of Sports Medicine*, 38(12), 2405–2409.
- Yard, E. E., & Comstock, R. D. (2009). Compliance with return to play guidelines following concussion in US high school athletes, 2005–2008. *Brain Injury*, 23(11), 888–898.
- McCrory, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M., et al. (2009). Consensus statement on concussion in sport—the Third International Conference on Concussion in Sport held in Zurich, November 2008. *The Physician and Sportsmedicine*, 37(2), 141–159.
- Thomas, M., Haas, T. S., Doerer, J. J., Hodges, J. S., Aicher, B. O., Garberich, R. F., et al. (2011). Epidemiology of sudden death in young, competitive athletes due to blunt trauma. *Pediatrics*, *128*(1), e1–e8.
- 74. Dick, R., Ferrara, M. S., Agel, J., Courson, R., Marshall, S. W., Hanley, M. J., et al. (2007). Descriptive epidemiology of collegiate men's football injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *Journal of Athletic Training*, 42(2), 221–233.
- Agel, J., Dompier, T. P., Dick, R., & Marshall, S. W. (2007). Descriptive epidemiology of collegiate men's ice hockey injuries: National collegiate athletic association injury surveillance system, 1988– 1989 through 2003–2004. *Journal of Athletic Training*, 42(2), 241–248.
- 76. Agel, J., Evans, T. A., Dick, R., Putukian, M., & Marshall, S. W. (2007). Descriptive epidemiology of collegiate men's soccer injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2002–2003. *Journal of Athletic Training*, 42(2), 270–277.
- 77. Dick, R., Putukian, M., Agel, J., Evans, T. A., & Marshall, S. W. (2007). Descriptive epidemiology of collegiate women's soccer injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2002–2003. *Journal of Athletic Training*, 42(2), 278–285.
- Dick, R., Lincoln, A. E., Agel, J., Carter, E. A., Marshall, S. W., & Hinton, R. Y. (2007). Descriptive

epidemiology of collegiate women's lacrosse injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2003– 2004. *Journal of Athletic Training*, 42(2), 262–269.

- Dick, R., Romani, W. A., Agel, J., Case, J. G., & Marshall, S. W. (2007). Descriptive epidemiology of collegiate men's lacrosse injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2003–2004. *Journal of Athletic Training*, 42(2), 255–261.
- Dick, R., Hootman, J. M., Agel, J., Vela, L., Marshall, S. W., & Messina, R. (2007). Descriptive epidemiology of collegiate women's field hockey injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2002–2003. *Journal of Athletic Training*, 42(2), 211–220.
- Marshall, S. W., Covassin, T., Dick, R., Nassar, L. G., & Agel, J. (2007). Descriptive epidemiology of collegiate women's gymnastics injuries: National collegiate athletic association injury surveillance system, 1988–1989 through 2003–2004. *Journal of Athletic Training*, 42(2), 234–240.
- Singh, S., Smith, G. A., Fields, S. K., & McKenzie, L. B. (2008). Gymnastics-related injuries to children treated in emergency departments in the United States, 1990–2005. *Pediatrics*, 121(4), e954–e960.
- Shields, B. J., & Smith, G. A. (2009). Cheerleadingrelated injuries in the United States: A prospective surveillance study. *Journal of Athletic Training*, 44(6), 567–577.
- Pellman, E. J., Powell, J. W., Viano, D. C., Casson, I. R., Tucker, A. M., Feuer, H., et al. (2004). Concussion in professional football: Epidemiological features of game injuries and review of the literature—part 3. *Neurosurgery*, 54(1), 81–94; Discussion 6.
- Pellman, E. J., Viano, D. C., Casson, I. R., Tucker, A. M., Waeckerle, J. F., Powell, J. W., et al. (2004). Concussion in professional football: Repeat injuries—part 4. *Neurosurgery*, 55(4), 860–873; Discussion 73–6.
- Pellman, E. J., Viano, D. C., Casson, I. R., Arfken, C., & Feuer, H. (2005). Concussion in professional football: Players returning to the same game—part 7. *Neurosurgery*, 56(1), 79–90; Discussion 2.
- Pellman, E. J., Viano, D. C., Casson, I. R., Arfken, C., & Powell, J. (2004). Concussion in professional football: Injuries involving 7 or more days out—Part 5. *Neurosurgery*, 55(5), 1100–1119.
- Benson, B. W., Meeuwisse, W. H., Rizos, J., Kang, J., & Burke, C. J. (2011). A prospective study of concussions among National Hockey League players during regular season games: The NHL-NHLPA Concussion Program. *Canadian Medical Association Journal*, 183(8), 905–911.
- Wennberg, R. A., & Tator, C. H. (2008). Concussion incidence and time lost from play in the NHL during the past ten years. *Canadian Journal of Neurological Sciences*, 35(5), 647–651.
- Fuller, C. W., Junge, A., & Dvorak, J. (2005). A six year prospective study of the incidence and causes of

head and neck injuries in international football. *British Journal of Sports Medicine*, 39(Suppl 1), i3–i9.

- French, L. M. (2009). TBI in the military. Preface. *The Journal of Head Trauma Rehabilitation*, 24(1), 1–3.
- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. Soldiers returning from Iraq. *New England Journal of Medicine*, 358(5), 453–463.
- 93. Schneiderman, A. I., Braver, E. R., & Kang, H. K. (2008). Understanding sequelae of injury mechanisms and mild traumatic brain injury incurred during the conflicts in Iraq and Afghanistan: Persistent postconcussive symptoms and posttraumatic stress disorder. *American Journal of Epidemiology*, 167(12), 1446–1452.
- 94. Tanielian, T., & Jaycox, L. H. (Eds.). (2008). Invisible wounds of war: Psychological and cognitive unjuries, their consequences, and services to assist recovery. Santa Monica, CA: RAND Corporation.
- Department of Defense: Traumatic brain Injury numbers. DoD numbers for traumatic brain injury '00–11' Q2 totals. [October 13, 2011]. http://dvbic. org/TBI-Numbers.aspx.
- Iverson, G. L., Langlois, J. A., McCrea, M. A., & Kelly, J. P. (2009). Challenges associated with postdeployment screening for mild traumatic brain injury in military personnel. *Clinical Neuropsychology*, 23(8), 1299–1314.
- Brenner, L. A., Vanderploeg, R. D., & Terrio, H. (2009). Assessment and diagnosis of mild traumatic brain injury, posttraumatic stress disorder, and other polytrauma conditions: Burden of adversity hypothesis. *Rehabilitation Psychology*, 54(3), 239–246.
- Ling, G. S., & Ecklund, J. M. (2011). Traumatic brain injury in modern war. *Current Opinion in Anaesthesiology*, 24(2), 124–130.
- 99. Terrio, H., Brenner, L. A., Ivins, B. J., Cho, J. M., Helmick, K., Schwab, K., et al. (2009). Traumatic brain injury screening: Preliminary findings in a US Army Brigade Combat Team. *The Journal of Head Trauma Rehabilitation*, 24(1), 14–23.
- 100. Wilk, J. E., Thomas, J. L., McGurk, D. M., Riviere, L. A., Castro, C. A., & Hoge, C. W. (2010). Mild traumatic brain injury (concussion) during combat: Lack of association of blast mechanism with persistent postconcussive symptoms. *The Journal of Head Trauma Rehabilitation*, 25(1), 9–14.
- Ivins, B. J. (2010). Hospitalization associated with traumatic brain injury in the active duty US Army: 2000–2006. *NeuroRehabilitation*, 26(3), 199–212.
- 102. DePalma, R. G., Cross, G. M., et al. (2011). Epidemiology of mTBI (Mild Traumatic Brain Injury) due to blast: History, DOD/VA data bases: Challenges and opportunities. *Papers presented at the RTO Human Factors and Medicine Panel (HFM) Halifax*, Canada on 3-5 October 2011.

- Okie, S. (2005). Traumatic brain injury in the war zone. New England Journal of Medicine, 352(20), 2043–2047.
- 104. Moore, D. F., & Jaffee, M. S. (2010). Military traumatic brain injury and blast. *NeuroRehabilitation*, 26(3), 179–181.
- 105. Warden, D. (2006). Military TBI, during the Iraq and Afghanistan wars. *The Journal of Head Trauma Rehabilitation*, 21(5), 398–402.
- 106. Taber, K. H., Warden, D. L., & Hurley, R. A. (2006). Blast-related traumatic brain injury: What is known? *Journal of Neuropsychiatry and Clinical Neurosciences*, 18(2), 141–145.
- 107. Meyer, K., Helmick, K., Doncevic, S., & Park, R. (2008). Severe and penetrating traumatic brain injury in the context of war. *Journal of Trauma Nursing*, 15(4), 185–189; quiz 90–1.
- 108. Galarneau, M. R., Woodruff, S. I., Dye, J. L., Mohrle, C. R., & Wade, A. L. (2008). Traumatic brain injury during Operation Iraqi Freedom: Findings from the United States Navy-Marine Corps Combat Trauma Registry. *Journal of Neurosurgery*, 108(5), 950–957.
- 109. Heltemes, K. J., Dougherty, A. L., MacGregor, A. J., & Galarneau, M. R. (2011). Inpatient hospitalizations of U.S. military personnel medically evacuated from Iraq and Afghanistan with combat-related traumatic brain injury. *Military Medicine*, 176(2), 132–5.
- 110. Wojcik, B. E., Stein, C. R., Bagg, K., Humphrey, R. J., & Orosco, J. (2010). Traumatic brain injury hospitalizations of U.S. army soldiers deployed to Afghanistan and Iraq. *American Journal of Preventive Medicine*, 38(1 Suppl), S108–S116.
- 111. Gaylord, K. M., Cooper, D. B., Mercado, J. M., Kennedy, J. E., Yoder, L. H., & Holcomb, J. B. (2008). Incidence of posttraumatic stress disorder and mild traumatic brain injury in burned service members: Preliminary report. *Journal of Trauma*, 64(Suppl 2), S200–S205; Discussion S5–S6.
- 112. Sayer, N. A., Cifu, D. X., McNamee, S., Chiros, C. E., Sigford, B. J., Scott, S., et al. (2009). Rehabilitation needs of combat-injured service members admitted to the VA Polytrauma Rehabilitation Centers: The role of PM&R in the care of wounded warriors. *PM & R: The Journal of Injury, function, and Rehabilitation, 1*(1), 23–28.
- 113. Carlson, K. F., Nelson, D., Orazem, R. J., Nugent, S., Cifu, D. X., & Sayer, N. A. (2010). Psychiatric diagnoses among Iraq and Afghanistan war veterans screened for deployment-related traumatic brain injury. *Journal of Traumatic Stress*, 23(1), 17–24.
- 114. Iverson, K. M., Hendricks, A. M., Kimerling, R., Krengel, M., Meterko, M., Stolzmann, K. L., et al. (2011). Psychiatric diagnoses and neurobehavioral symptom severity among OEF/OIF VA patients with deployment-related traumatic brain injury: A gender comparison. *Women's Health Issues*, 21(4 Suppl), S210–S217.
- 115. Polusny, M. A., Kehle, S. M., Nelson, N. W., Erbes, C. R., Arbisi, P. A., & Thuras, P. (2011). Longitudinal effects of mild traumatic brain injury and posttraumatic stress disorder comorbidity on postdeployment outcomes in national guard soldiers deployed to Iraq. Archives of General Psychiatry, 68(1), 79–89.
- 116. Nampiaparampil, D. E. (2008). Prevalence of chronic pain after traumatic brain injury: A systematic review. *Journal of the American Medical Association*, 300(6), 711–719.
- 117. Web-based Injury Statistics Query and Reporting System (WISQARS) [database on the Internet] 2011 [cited October 14, 2011]. http://www.cdc.gov/ injury/wisqars.
- 118. Zaloshnja, E., Miller, T., Langlois, J. A., & Selassie, A. W. (2008). Prevalence of long-term disability from traumatic brain injury in the civilian population of the United States, 2005. *The Journal of Head Trauma Rehabilitation*, 23(6), 394–400.
- 119. Finkelstein, E., Corso, P., & Miller, T. (2006). The incidence and economic burden of injuries in the United States. New York: Oxford University Press.
- 120. Roebuck-Spencer, T. M., Banos, J., Sherer, M., & Novack, T. (2010). Neurobehavioral aspects of traumatic brain injury sustained in adulthood. In S. Hunter & J. Donders (Eds.), *Principles and practice* of lifespan developmental neuropsychology (pp. 329–344). Cambridge, England: Cambridge University Press.
- 121. Draper, K., Ponsford, J., & Schonberger, M. (2007). Psychosocial and emotional outcomes 10 years following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 22(5), 278–287.
- 122. Ezrachi, O., Ben-Yishay, Y., Kay, T., Diller, L., & Rattock, J. (1991). Predicting employment in traumatic brain injury following neuropsychological rehabilitation. *The Journal of Head Trauma Rehabilitation*, 6(3), 71–84.
- 123. Gollaher, K., High, W., Sherer, M., Bergloff, P., Boake, C., Young, M. E., et al. (1998). Prediction of employment outcome one to three years following traumatic brain injury (TBI). *Brain Injury*, 12(4), 255–263.

- 124. Engberg, A. W., & Teasdale, T. W. (2004). Psychosocial outcome following traumatic brain injury in adults: A long-term population-based follow-up. *Brain Injury*, 18(6), 533–545.
- 125. Brooks, N., McKinlay, W., Symington, C., Beattie, A., & Campsie, L. (1987). Return to work within the first seven years of severe head injury. *Brain Injury*, *1*(1), 5–19.
- 126. Dikmen, S., Machamer, J., & Temkin, N. (1993). Psychosocial outcome in patients with moderate to severe head injury: 2-year follow-up. *Brain Injury*, 7(2), 113–124.
- 127. Kersel, D. A., Marsh, N. V., Havill, J. H., & Sleigh, J. W. (2001). Psychosocial functioning during the year following severe traumatic brain injury. *Brain Injury*, 15(8), 683–696.
- Liss, M., & Willer, B. (1990). Traumatic brain injury and marital relationships: A literature review. *International Journal of Rehabilitation Research*, 13(4), 309–320.
- 129. Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: A literature review and recommendations. *Brain Injury*, 9(1), 81–92.
- 130. Think First National Brain Injury Prevention Foundation [Online]. Fast facts: Traumatic brain injury. 2008 [cited 2008 July 1]. http://www.thinkfirst.org/Documents/FastFacts/TFbrain368.pdf.
- 131. Attewell, R. G., Glase, K., & McFadden, M. (2001). Bicycle helmet efficacy: A meta-analysis. Accident Analysis and Prevention, 33(3), 345–352.
- 132. Pintar, F. A., Yoganandan, N., & Gennarelli, T. A. (2000). Airbag effectiveness on brain trauma in frontal crashes. Annual Proceedings of the Association for the Advancement of Automotive Medicine, 44, 149–169.
- 133. Hillary, F. G., Schatz, P., Moelter, S. T., Lowry, J. B., Ricker, J. H., & Chute, D. L. (2002). Motor vehicle collision factors influence severity and type of TBI. *Brain Injury*, 16(8), 729–741.
- 134. Stevens, J. A. (2010). A CDC compendium of effective fall interventions: What works for communitydwelling older adults (2nd ed.). Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

# Cognitive and Behavioral Outcomes from Traumatic Brain Injury

# Julie Griffen and Robin Hanks

#### Abstract

Outcome following traumatic brain injury (TBI) depends on many factors, including severity of injury. Research has demonstrated a dose–response relationship between TBI severity and cognitive outcomes, with more severe deficits associated with increasingly severe injuries. Additionally, changes in psychological functioning, behavior, and participation in the community may be sequelae of TBI. This chapter focuses on recovery and outcomes from TBI, including mild, mild-complicated, and moderate-to-severe, as well as mitigating factors, such as demographics and premorbid factors, psychological comorbidities, type of injury, repeated concussions, and financial incentives. Outcome prediction is essential for planning rehabilitation goals, patient and family education about long-term changes, and identifying necessity of future assistance. This task is made challenging by the additional influence of factors such as premorbid functioning, type and severity of injury, and other situational factors.

#### Keywords

Outcomes • Cognition • Behavior • Psychosocial • Participation • Mitigating factors

J. Griffen, Ph.D. • R. Hanks, Ph.D., ABPP (⊠) Wayne State University School of Medicine, Rehabilitation Institute of Michigan, Detroit, MI, USA e-mail: rhanks@med.wayne.edu A variety of factors affect recovery from traumatic brain injury (TBI). The extent of cognitive recovery from TBI largely depends on the severity of the injury, as illustrated by the well-established dose–response relationship between TBI severity and cognitive outcome [1, 2]. This finding demonstrates that neuropsychological performance correlates with severity of injury with the most severe deficits following severe injuries. Furthermore, changes in behavior, psychological functioning, and participation in the community may also occur subsequent to TBI. This chapter will address recovery and outcomes following TBI, including mild, mild-complicated, and moderate-to-severe, as well as mitigating factors.

#### Mild TBI

Mild TBI (mTBI) has traditionally been defined by a Glasgow Coma Scale (GCS) score of 13-15, loss of consciousness (LOC) less than 30 min, and posttraumatic amnesia (PTA) less than 24 h [3]. Brief LOC may also be associated with mTBI; however, its usefulness in predicting symptoms subsequent to mTBI is questionable. Research has shown that LOC is not associated with total number of symptoms or duration of symptoms among athletes who sustained concussions [4]. Some persons report experiencing symptoms consistent with post-concussion syndrome (PCS), which can be grouped into three clusters: somatic, affective, and cognitive [5]. Somatic symptoms typically include headache, fatigue, and dizziness. The affective cluster often includes irritability, anxiety, depressed mood, and sleep difficulties. The most commonly reported cognitive changes include slowed processing speed, poor attention, and difficulty with short-term memory [5-7].

It is noteworthy, however, that PCS symptoms are not specific to mTBI [8, 9] and some individuals have questioned the construct validity of this diagnosis, including the authors of the Diagnostic and Statistical Manual of Mental Disorders—IV-Text Revision [10]. mTBI is listed as a diagnosis under the category of Criteria Sets and Axes Provided for Further Study, due to the limited empirical evidence for this disorder as a distinct entity. Furthermore, in prospective studies, the presence of mTBI did not predict PCS symptoms at 5 days post-injury, rather the presence of a depressive or anxiety disorder and female gender were most strongly related to PCS symptom reporting [6, 8, 11]. Rates of PCS symptoms have been found to be roughly equivalent among mTBI patients and trauma controls, which indicates that mTBI is not specifically predictive of PCS [12]. Other studies have found similar rates of PCS symptoms among normal controls and outpatients [13, 14]. Furthermore, PCS symptoms have not been found to be related to objectively measured cognitive functioning [12]. Rather subjective complaints of deteriorating cognitive functioning are likely related to non-brain injury factors [15, 16]. For studies that have noted PCS symptoms, symptoms resolved within 16 days of injury [4] and poor medical health, mental health, and marital status at baseline (being widowed, divorced, or separated) were more predictive of complaints such as fatigue at 12 months than was injury [17]. Persons with history of mTBI may attribute fatigue, as well as other PCS symptoms, to their injuries but the symptoms may actually be associated with premorbid factors, psychological status, or other health-related factors. Thus, PCS symptoms appear to reflect psychological distress related to the events of the injury, pre-injury psychological distress, or non-clinical factors and are likely unrelated to the actual head injury especially when they occur after the first month.

Mild cognitive deficits may occur within the first few days after mTBI, often including difficulty with recall, slowed processing speed, and reduced attention. These symptoms may be due to the actual brain injury, but other causes such as injury-related pain and psychological distress cannot be ruled out [8]. Research has suggested that athletes sustaining concussions demonstrate full recovery on cognitive measures within 7–10 days (e.g., return to personal baseline) [18, 19]. In community samples of persons with mTBI deficits in verbal learning and attention as compared to controls were evident up to 1-6 months post-injury in some individuals, but many individuals did not show any cognitive impairment during this time period [20, 21]. Although subtle neuropsychological changes may be evident following mTBI, research has shown that these changes are often resolved within 3 months [8, 19].

Regarding functional outcomes, assessed with the GOS, research has shown that persons who have sustained mTBI have good outcomes in both the short and long terms [8]. Poor outcomes and lingering PCS symptoms appear to be unrelated to brain injury and likely related to psychological variables or litigation. For example, subjective cognitive and somatic complaints may be related to depression or anxiety [8, 22]. The literature also indicates a tendency for persons sustaining injuries to recall themselves as being healthier and happier prior to the injury. For example, persons who had sustained concussions 6 months previously underestimated their pre-concussion symptoms by 97 % [23]. This tendency has been referred to as the "good old days" bias, such that people overestimate the actual degree of change that has occurred since injury by underestimating premorbid symptoms [24–26]. Iverson et al. [25] found that preinjury ratings of persons with mTBI were higher than ratings of control participants. As PCS symptoms are nonspecific, a person may associate negative events and feelings to an injury rather than normal emotional fluctuations, events, etc. The effects of litigation on recovery from mTBI are discussed later in this chapter in the mitigating factors section. Thus, consistent findings indicate full recovery following an uncomplicated mTBI in the vast majority of injured persons.

#### Repeated mTBI

Research has also focused on the cumulative effects of sustaining repeated mTBIs, especially among athletes. Whereas some correlational studies have suggested a dose-response relationship between number of injuries and poorer neuropsychological status, a meta-analysis on multiple mTBIs by Belanger et al. [27] indicated that the literature revealed no overall significant effect on neurocognitive functioning or symptom complaints. Their results showed that sustaining multiple mTBIs has been associated with cognitive deficits in executive functioning and memory, but the effect size was small and the clinical significance was unclear. The authors concluded that sustaining two or more mTBIs has little relationship with cognitive performance several months later.

Some researchers have speculated that repeated trauma to the brain may accelerate degenerative processes later in life, such as mild cognitive impairment, Parkinson's, Alzheimer's, or some other type of neurodegenerative disease [28]. Findings concerning whether the length of recovery following subsequent mTBIs is longer than the first mTBI have been inconclusive [27, 28]. Among professional boxers, a review of the literature by Loosemore et al. [29] revealed that 10-20 % develop measurable long-term brain injury as indicated by clinical, radiologic, or histopathologic examination, but they found that amateur boxing was not associated with neurocognitive deterioration. A recent study on diffuse axonal injury (DAI) as measured by MRI showed no significant differences in frequencies of microhemorrhages between amateur boxers and controls [30]. A possible explanation for this discrepancy is that a head guard is not permitted in professional boxing, but is required in amateur boxing. Although the concepts of dementia pugilistica and chronic traumatic encephalopathy have been discussed in the media, there are very few well-controlled studies published in the neurology literature on these conditions and the studies have not controlled for other factors that could contribute to cognitive impairment such as premorbid learning disabilities, ADHD, and substance abuse.

The risk of "second-impact syndrome" has been identified, especially among athletes. This syndrome may occur when someone sustains a second concussion prior to complete healing from a previous concussion, usually within a few days to a week, which has been thought to lead to acute brain swelling and the possibility of fatality [19], but the probability of such an injury occurring is extremely low. Randolph and Kirkwood's [31] review of the risks of sport-related concussion included an examination of the Catastrophic Sports Injury Database for 10 years of American football at all levels. These authors found that there was only one case of diffuse cerebral swelling at all levels of play over this time period, even though it was estimated that 40 % of players were returned to play when symptomatic [32]. Given the overall high rate of concussion, one would expect that if second impact syndrome was a common medical event, one would see more than one case in a 10-year span in this database. Additionally, given the return to play guidelines currently in place in the sports community, individuals are at a lower risk for this kind of event given that it already has a very low base rate. Research has indicated that when diffuse cerebral swelling is seen, it is most often in children and adolescents and represents a calcium subchannel mutation that is related to familial hemiplegic migraine [33, 34].

#### Complicated mTBI

Complicated mTBI is a classification of TBI for cases in which GCS scores were between 13 and 15 with evidence of an intracranial bleed or lesion [35, 36]. Research has shown that a subgroup of persons with GCS scores in this range accompanied by intracranial bleeds had poorer outcomes, including neuropsychological status, than those who had sustained an uncomplicated injury [35]. Similarly, persons with GCS of 15 after TBI who required surgery after developing intracranial hematoma were also at risk for worse outcomes, such that only 65 % had a good outcome [37]. Research has shown an increased level of disability among persons with complicated mTBI at 6 months and 1 year as compared to those sustaining uncomplicated injuries [35, 36, 38, 39]. Persons sustaining complicated mTBIs have also been found to have significantly worse cognitive performances than control participants at 1 month post-injury, whereas those with uncomplicated mTBI had performances comparable to controls [40]. Furthermore, based on group data, persons sustaining complicated mTBIs have not been found to experience complete recovery of cognitive function to unimpaired levels by 1 year postinjury, which contrasts the 3 month recovery of neuropsychological status that is typical with mTBI [36].

Outcomes at 1 year post-injury among persons with complicated mTBI are actually more consistent with the outcomes following moderate TBI [36]. Functional outcomes were equivalent for those with complicated mTBI and moderate TBI, including the following domains: physical (FIM motor domain scores), cognitive (FIM cognitive domain), independence, employability (e.g., Disability Rating Scale), and community integration (Community Integration Questionnaire (CIQ)). The most common areas of cognitive impairment were processing speed, learning and memory, and word generation [35, 36].

Kashluba et al. [36] highlighted the importance of these findings in understanding the limitations of exclusively relying on GCS scores in the classification of TBI severity, as many of these individuals have length of unconsciousness or length of posttraumatic confusion (PTC) that are more consistent with moderate injuries. Persons with similar GCS scores in the mild range can have strikingly different outcomes if the injury was further complicated by an intracranial bleed. The authors argue for greater consideration of additional factors beyond GCS to predict outcome and determine severity of injury, including LOC, length of PTA, and time to follow commands. As the complicated mTBI group has been found to have similar outcomes to those sustaining moderate TBIs, more information regarding their outcomes is presented in the moderate-severe section of this chapter.

#### Moderate-Severe TBI

Moderate to severe TBI has typically been defined as a LOC longer than 30 min, PTA longer than 24 h, or a GCS of 9–12 for moderate severity and 3-8 for severe severity [3]. Following injury, persons with severe TBI may proceed through a series of stages, including coma, vegetative state, minimally conscious state, confused state (PTA), and recovery. While all will, by definition, have some period of coma (typically described as a GCS of 8 or below), most will never be in a vegetative state and it is unknown how many will be in a minimally conscious state at some point during recovery. For patients who are in vegetative and/or minimally conscious states, the duration of these states varies greatly [41]. Many people with moderate TBI, however, may never have been in a coma and none are in coma at hospital admission. Vegetative and minimally conscious states are not seen in moderate TBI unless there is deterioration due to some late occurring complication such as intracranial bleeding. However the confused state is seen in all patients with moderate or severe TBI. Incidence of persistent vegetative state (complete unawareness of self and environment lasting longer than 30 days with some preserved brainstem functioning such as eye opening) at 1 year post-injury is rare (e.g., 1 %; [41]). During a minimally conscious state, a person demonstrates inconsistent awareness of the environment and is inconsistent in following commands. The minimally conscious state is typically a temporary phase of recovery [42]. Subsequently, persons with TBI become responsive but confused. They often experience retrograde amnesia, such that they cannot recall events for a period of time prior to the head injury. In severe injuries, the last memory that can be recalled may have occurred days, months, or even longer before the injury. As recovery proceeds, the extent of retrograde amnesia typically shortens but the events surrounding the injury are not typically fully recalled. PTA is memory loss for events following the injury, including time in coma [43]. As other neurobehavioral impairments (e.g., disrupted sleep-wake cycle, disturbed consciousness, altered psychomotor activity, agitation) often accompany the memory and cognitive impairments seen in PTA, the broader term PTC is now often used to describe the early period of recovery following TBI [44]. PTC may be brief, such as several days, or longlasting, such as longer than several weeks. PTA/ PTC is typically considered to be resolved when a person demonstrates orientation and the ability to create and retain new memories [42]. PTA duration is best determined by serial administration of an orientation measure such as the Orientation Log or the Galveston Orientation and Amnesia Test. Agitated or restless behaviors during PTC have been associated with better outcomes than initial sluggishness or immobility [45]. Sherer et al. [46] found that patients who were more severely confused had worse outcomes in employability and productivity.

Neurobehavioral symptoms are common among those with moderate to severe TBI and typically include problems with irritability, temper, dizziness, sensitivity to noise, and blurred vision. Additionally, they may experience apathy or lack of initiative, as well as extreme fatigue or tiring easily [43]. The most common cognitive deficits following moderate to severe TBI include impairments in attention, processing speed, and learning and memory. Attentional difficulties can include distractibility, poor concentration, and reduced divided attention. Information processing speed is reduced, which can affect numerous other cognitive domains. Complex attention and executive functioning have frequently been found to be impaired following moderate to severe TBI [47, 48]. Some have argued that these deficits are the consequence of deficits in processing speed and attention [49]. Executive dysfunction may present as poor self-awareness, insight, or selfcontrol, as well as impairments in organization, planning, and judgment. Memory problems typically consist of difficulties with learning new information, retaining it, and subsequently retrieving it. These difficulties may also be due to impairments in attention and executive functioning. Deficits in language and visuospatial constructional skills may also be present in more severe injuries [1]. Classic aphasia is relatively rare following TBI, unless there is a focal lesion [50]. Communication problems may be present due to word finding problems, organization, and executive functioning (e.g., social judgment, impulsivity). At 1 month post-injury, persons with moderate or severe TBI performed significantly worse on all cognitive measures than did controls, and those with severe injuries continued to perform worse than controls at 1 year post-injury [51].

Significant, long-term deficits are consistently found among persons with moderate to severe TBI. Moderate TBI appears to be associated with cognitive deficits for 6 months or longer postinjury; it is noteworthy that in a meta-analysis on cognitive outcome, Dikmen et al. [1] indicated that due to inconsistent use of cut points in severity labels, findings had to be tempered accordingly. Consistent with the dose–response theory, severe TBI is often associated with more extensive cognitive deficits than moderate TBI. For example, Novack et al. [52] found that the severely injured group had worse cognitive outcomes than did the moderately injured group at 12 months post-injury, and both groups were below normative cognitive performance levels.

Research on the timeline of cognitive recovery from moderate to severe TBI has generally indicated that the most rapid recovery occurs during the first 5 months post-injury [53]. Recovery typically continues between 6 months to 2 years post-injury [54] with the steepest rates of improvement between 2 and 5 months [53]. Millis et al. [55] found that further improvement can still be evident at 5 years post-injury. Rate and extent of recovery varies across cognitive domains, such that there is slower improvement for more complex functioning [56]. Consistent with this, Millis et al. [55] found that a greater proportion of persons with TBI demonstrated improvement on simpler memory tasks than on complex memory tasks. Recovery of reasoning and problem-solving skills was also variable, such that some people demonstrate sparing or improvement and others exhibit marked impairment up to several years post-injury. Their results also showed that the greatest improvements during years 1–5 post-injury occurred on measures of visuoconstruction (Block Design), problemsolving (Wisconsin Card Sorting Test), and complex attention (Trails B). Motor and visuospatial domains continue to show steep improvement throughout the first year post-injury [53]. Millis et al. [55] reported that tasks that require complex constructional skills and speed may be the most sensitive to late recovery (e.g., 1-5 years post-injury).

Residual cognitive deficits are common 2 years post-injury [54] and differences between persons with TBI and controls in attention, processing speed, memory, and executive functioning have been found up to 10 years post-injury [57]. Millis et al. [55] found that at 5 years post-injury (complicated mild to severe) cognitive functioning ranged from no measurable impairment to severe impairment. The course of cognitive recovery varies greatly among individuals. Millis et al. [55]

found that 22.2 % of persons in the Traumatic Brain Injury Model Systems (TBIMS) national database demonstrated cognitive improvement during 1-5 years post-injury, while 62.6 % remained stable and 15.2 % deteriorated. The further decline of cognitive functioning during the recovery stage of TBI has also been reported by Till et al. [58]. Millis et al. [55] noted that the subgroup showing continued decline in cognitive functioning was the oldest group (e.g., middle age), with the next lower age group remaining stable, and the youngest group showing cognitive improvement. The declines were most evident on measures requiring cognitive flexibility and speeded performance and were of a great enough magnitude to be associated with functional declines. Bercaw et al. [59] suggested that possible reasons for this decline include medical comorbidities or emotional distress.

Cognitive performance measured during acute rehabilitation is useful in predicting 1-year outcomes, even above and beyond functional and injury severity factors. For example, neuropsychological status within the first month postinjury assessed during inpatient rehabilitation with a brief standard battery was predictive of handicap, functional outcomes, need for supervision, and employability at 1 year post-injury. An estimate of premorbid cognitive functioning (WTAR) was most predictive of outcomes at 1 year post-injury individually. A measure of executive functioning (TMT-B) also showed unique predictive power of functional independence (FIM) and general outcomes (GOS-E) [60].

#### **Productivity Outcomes**

Moderate to severe TBI has been associated with significant functional impairments at 3–5 years post-injury. Specifically, Dikmen et al. [61] noted that 60 % of their sample of persons with TBI reported having long-lasting cognitive difficulties that affected their abilities to complete daily tasks independently and 30 % were unable to return to school or work. Bercaw et al. [59] found that improvements in processing speed and learning were predictive of ratings of

disability and functional independence at 2 years post-injury, even after controlling for effects of age and injury severity.

Cognitive and behavioral sequelae after TBI can interfere with returning to or gaining employment. Estimates for the rate of unemployment following TBI range from 60 to 80 % [62, 63]. A strong dose–response relationship has been shown between severity of TBI and return to work rates, with lower rates of return for more severe injuries [64, 65]. The early indicators of TBI severity (e.g., GCS, length of PTA, time to follow commands) and functional indicators of injury severity (e.g., measures of disability and handicap such as DRS or FIM) have been associated with rates of returning to work at 1 year post-injury [66].

Doctor et al. [67] reported unemployment rates at 1 year post-injury of 31 % among persons with mTBI, 46.4 % among those with moderate TBI, and 62.1 % among those with severe TBI, all of whom were working prior to their injuries. These rates were significantly higher than those of the general population, ranging from 8 to 10 %. Dikmen et al. [21] had similar findings, such that persons with more severe TBIs had lower return rates than those with mild or moderate injuries. Additionally, they reported that persons with TBI had a lower return to work rate than trauma controls (49 %vs. 63 %) at 1 year post-injury. The greatest rate of returning to work post-TBI occurs between 1 and 6 months postinjury; however, those with more severe TBIs may continue to return to work over a longer period of time as compared to those with mTBIs who reach an asymptote earlier. It is noteworthy that only a very small percentage of those with the most severe TBIs return to work within 2 years of injury [64].

Neuropsychological status is also an important factor in returning to work. Dikmen et al. [64] found that neuropsychological functioning at 1 month post-injury was associated with employment rates. Specifically, 96 % of those with "excellent" cognitive abilities were working; these people likely had milder injuries and good premorbid functioning. Intact executive functioning, attention, and memory skills are critical for adequate performance and job success. In a meta-analysis of the predictive power of neuropsychological assessment in vocational outcomes, Crepeau and Scherzer [68] found that executive functioning was highly correlated with employment outcomes and measures of language, visuospatial abilities, and global cognitive status was moderately correlated with employment. Cifu et al. [66] reported that neuropsychological assessment occurring in the acute phase of rehabilitation was not predictive of employment outcomes at 1 year. They hypothesized that this finding may be related to the fact that a minimum threshold of functioning must be present to complete neuropsychological testing, which reduces the range of assessment by removing the most severe injuries from the analyses. They also suggested that the specific domains of neuropsychological assessment may not adequately map onto functional skills and abilities; whereas, cognitive assessment on a more functional measure (e.g., DRS) was predictive of return to work. Sherer et al. [69], however, found that early cognitive assessment was predictive of later employment outcomes. This review study also indicated inconclusive findings regarding the relationship between late or concurrent neuropsychological assessment with employment outcomes. Consistent with this, Putnam and Fichtenberg [70] indicated that the predictive power of neuropsychological assessment attenuates as time increases between time of injury and time of neuropsychological testing. They argued that comprehensive assessments, especially approximately 1 month post-injury, are more informative than later assessments.

Demographic variables have also been associated with rates of returning to work post-TBI. Dikmen et al. [64] found that age, education, and pre-injury work history were predictive of postinjury employment. Specifically, they found that persons over age 50, persons with less than a high school education, and those with unstable work histories were less likely to return to work and if they did return, the time to do so was longer. Having no history of alcohol and drug abuse or premorbid psychiatric diagnosis has also been associated with more productive activity at 1 year post-injury [71]. Lower scores on measures of productivity in community integration have been associated with older age, premorbid behavioral problems, less education, and being unmarried at the time of the injury [72].

Unfortunately, the literature on return to work often consists exclusively of measurement of employment status, without further assessment of other job-related variables. Cifu et al. [66] argued that a measurement of percentage of employment among persons with TBI at various time points post-injury provides a distorted picture of the overall adjustment to employment among this group. The literature is lacking in description of job retention and changes in level of employment (e.g., less complex tasks, fewer responsibilities, fewer hours) [65]. However, several studies have looked at job stability rates. For example, Machamer et al. [73] found that premorbid characteristics, including extent of preinjury work history and earnings, and lower neuropsychological functioning post-injury were significantly related to difficulty maintaining stable employment 3-5 years post-injury. In their study, 46 % of those who returned to work mainstable, uninterrupted employment. tained Kreutzer et al. [74] also indicated relationships between demographic variables and stable employment after TBI, including minority status, marital status, and educational background. They also noted that driving independence was strongly related to post-injury work stability.

## Independent Living and Community Integration Outcomes

Dikmen et al. [75] reported that 76 % of persons with TBI returned to independent living, in contrast to 93 % of trauma controls at 1 year postinjury. Again, increased injury severity (as measured by length of coma) was associated with reduced likelihood of returning to independent living; specifically, the rates were 89 % for less than 1 h and 1–24 h of coma, 74 % for 1–6 days, 49 % for 7–13 days, 55 % for 14–28 days, and 23 % for more than 29 days. Temkin et al. [65] noted that many persons with TBI who had previously lived independently resided with their parents for the first few years post-injury. Hart et al. [76] reported that neuropsychological assessment, particularly of executive functioning, was predictive of need for supervision after TBI.

Social functioning and community integration are also often affected by moderate to severe TBI. Problems in these areas have been reported to be greater than those in basic ADLs at 1 year post-injury and also exhibit a dose-response relationship with injury severity [65, 77]. Participation in leisure activities also appears to be reduced among those with moderate to severe TBI, even many years post-injury [65]. Relative stability of social functioning, albeit lower than community norms, has been reported for at least 5 years postrehabilitation. Evidence for some improvement in the subdomains of home integration and occupation was reported [78]. Female gender tended to predict better outcomes in community integration overall and specifically in home integration [72].

# Life Satisfaction

Reductions in life satisfaction and quality of life post-TBI have also been reported [65]. A study examining changes in life satisfaction over time following TBI showed that a decline in the second year postdischarge was followed by steady improvement. The authors reported that this pattern could represent a "honeymoon period" in which persons with TBI do not fully appreciate the consequences of their injuries until sometime after discharge and then satisfaction increases as people begin to adapt and use accommodations for long-lasting sequelae [78]. Those who have better awareness of their deficits secondary to TBI have been found to report less life satisfaction as compared to persons with impaired awareness of deficits [79]. Unlike other domains described in this section, life satisfaction does not appear to have a strong dose-response relationship with injury severity, with research showing either no effect [80] or increased life satisfaction with greater severity of injury [81, 82]. Pierce and Hanks [83] reported that this counterintuitive finding that enhanced life satisfaction follows more severe injuries may be explained by oversensitivity to symptoms seen in persons with mild injuries. They found that the strongest predictor of life satisfaction was level of participation or extent of involvement in activities; execution and ability to do various activities was also predictive of satisfaction but to a lesser extent.

#### Psychological Sequelae of TBI

TBI has been associated with increased risk of psychological disorders, both organically and situationally, with regard to reactions to injury and disability. This section primarily focuses on organically based changes which may occur following moderate to severe TBI, with some highlights of other causes and rates of disorders. Possible biological causes for psychological changes following TBI include the possibility that hypoxia leads to the release of free radicals and excitotoxic neurotransmitters. Additionally, neurochemical changes can occur subsequent to TBI, including effects on norepinephrine, serotonin, dopamine, and acetylcholine [84]. Anxiety and depression have been shown to have the strongest association with TBI and are perhaps the most commonly studied psychological changes. A meta-analysis by van Reekum et al. [85] revealed that the highest relative risks (RR) following TBI were for major depression (RR 7.5), bipolar disorder (RR 5.3), anxiety disorders (RR 2.0), and panic disorder (RR 5.8). Interestingly, although there are many pharmacologic approaches to dealing with these disorders in the medical and psychiatric literature, there are no approved medications for treating these disorders in those with TBI and medications are used off-label. For a review see Waldron-Perrine, Hanks, and Perrine [86].

#### **Mood Disorders**

*Depression*. Research has shown compelling evidence for TBI causing major depression [85].

Estimates for the occurrence of depression during the first year post moderate to severe TBI range from 14 to 42 %; rates for future time points, up to 50 years post-injury, were 11-61 %. The rates of moderate to severe depression as measured by the Center for Epidemiological Studies Depression Scale (CES-D) following complicated mild to severe TBI ranged from 31 % at 1 month to 17 % at 3–5 years post-injury [87]. Major Depression occurred in 44.3 % of persons with TBI in the time up to 7.5 years post-injury [85]. These estimates are much higher than the lifetime prevalence of depression in the general community [88]. Dikmen et al. [87] suggest that the elevated rates of depression post-TBI cannot be fully explained by the presence of somatic symptoms associated with brain injury, as depressed affect and lack of positive affect are also increased in persons with TBI.

The development of depression post-TBI is secondary to numerous factors, including premorbid personality, psychiatric history, social support, reaction to injury and disability, and organic changes [85]. Dikmen et al. [87] reported that the rates of depression were largely unassociated with severity of the brain injury (e.g., complicated mild, moderate, and severe). Increased depressive symptoms have, however, also been reported to be associated with less severe TBIs [85, 87]. Dikmen et al. [87] explain that the literature assessing the relationship between TBI severity and depression is mixed, likely because the relationship is complex and mediated by many factors, including awareness of deficits, injury-related disabilities, etc. Symptoms of depression may increase as a person becomes more aware of his or her deficits. Dikmen et al. [87] also found that premorbid factors, such as unstable work history, less than high school education, and alcohol abuse, predicted the presence of depression after TBI. Premorbid psychiatric disorders may also contribute to post-TBI depression [89]. Research has generated mixed findings for gender differences in the development of depression post-TBI, several showing no relationship [90–92] and some showing an increased risk for females [93, 94].

Robinson and Jorge [89] argue that although depression may be related to changes in social functioning, it is "not simply a psychological response to the severity of physical or intellectual impairment" (p. 237). Lesion location(s) and changes in neurotransmitters have been considered organic causes of depression post-TBI of moderate to severe severity [85, 95–97]. Associations between lesions in the left dorsolateral frontal cortex, left basal ganglia, and to a lesser extent, right hemisphere and depression have been reported [78, 97, 98]. Depressive symptoms post-TBI may also be related to hypometabolism in the lateral and dorsal frontal cortex, specifically the cingulate gyrus, and increased activation in the ventral limbic structures, such as the amygdala, medial thalamus, and prelimbic cortex [99–101].

Although researchers have noted that our understanding of the neurobiological underpinnings of depression is limited, Rosenthal et al. [102] hypothesized that lesions/contusions to the frontal pole where noradrenergic and serotonergic projections enter the cortex could greatly disrupt cortical aminergic functions, possibly leading to depression. Kelly et al. [103] found that 18 % of persons with TBI had growth hormone deficiency/ insufficiency at 6–9 months post-injury and also were 3.7 times more likely to report depression, suggesting a possible relationship between hormone changes and depression subsequent to TBI.

Bipolar disorder. Bipolar affective disorder was present in 4.2 % of persons with TBI over 7.5 years post-injury in the van Reekum et al. [85] meta-analysis, which is higher than the general lifetime prevalence rate of 0.8 % [88]. Males were found to have an increased likelihood of bipolar or cyclothymia in a small sample size (e.g., 1 of 10 females and 4 of 8 males; [94]). Shukla et al. [104] found that 50 % of persons developing mania post-TBI also had seizures, suggesting a possible link of mania secondary to seizure. Pope et al. [105] reported that persons with mania post-TBI responded preferentially to valproate rather than the typical drug of choice, lithium, which further supports a seizure hypothesis. Mania may also be associated with right hemisphere lesions, limbic system

lesions [106], and temporal basal polar lesions [91]. Jorge et al. [91] reported that mania has not been associated with severity of TBI, level of cognitive, physical, or social impairments, or personal/ familial history of psychiatric disorder.

#### Anxiety

Generalized anxiety disorder (GAD). In a metaanalysis, van Reekum et al. [85] reported that 9.1 % of persons with TBI had GAD over the 7.5 years post-injury. This is approximately two times the community prevalence rates. GAD is often comorbid with depression following TBI, just as it is in community samples [94]. Comorbid anxiety and depression were associated with a longer duration of mood disturbance as compared to non-anxious, depressed persons with TBI. Research has been mixed as to the association between severity of injury and risk of GAD. Epstein and Ursano [107] described the interrelationships between anxiety and TBI as multifactorial, and at that time the relationship to specific tissue damage was unclear; however, some studies have pointed to certain typical lesion occasions. Jorge et al. [91] reported that right hemisphere lesions were associated with anxious depression, and left anterior lesions were associated with major depression without anxiety. Right orbito-frontal lesions have also been associated with anxiety and depression [108].

*Posttraumatic stress disorder* (PTSD). PTSD was reported to occur in 14.1 % of a large sample in van Reekum's [85] meta-analysis over 7.5 years post-TBI with a relative risk of 1.8. PTSD appears to be more common in persons with less severe TBI, as it is most likely that those with mild injuries have memories related to their accident (e.g., motor vehicle accident, assault) whereas those with severe injuries are less likely to have any recall of these stressful events secondary to LOC or amnesia [109]. Among persons with mTBI who experienced acute stress disorder at 1 month post-injury, 82 % later developed PTSD, whereas it only occurred in 11 % of persons with mTBI who did not have acute stress disorder [110]. Many persons who experience PTSD post-TBI have also suffered other physical traumas, which can also cause or contribute to PTSD [111]. In a small sample, Ohrey et al. [112] found higher rates of PTSD among females post-TBI.

*Panic disorder*. van Reekum et al. [85] reported that 9.2 % of persons with TBI had panic disorder over the 7.5 years post-injury in a meta-analysis, which has a relative risk of approximately 5.8 as compared to the community prevalence rates. Typical neuroanatomical areas that have been implicated in panic attacks include orbitofrontal, cingulate, and medial temporal cortical areas, which are also frequent locations for TBI-related lesions [111]. Yet, as van Reekum et al. [85] notes, there is no available data at this point to support a pathophysiologic hypothesis of panic disorder in TBI.

Obsessive compulsive disorder (OCD). With regard to obsessive and compulsive symptoms, van Reekum [85] noted in the meta-analysis of articles spanning a 7.5 year period following TBI, 6.4 % of people had OCD, which indicates a relative risk of 2.6 as compared to a community sample. It was thought that OCD associated with TBI may be relatively brief and could be very effectively treated with antidepressants, as compared to the generally more chronic OCD [111, 113]. Based on available research, Kant et al. [114] have concluded that there is a possible causative involvement of the frontal systems impairment in developing OCD post-TBI. Drummond and Gravestock [115] have also suggested that the limbic system, which has been implicated as a checking system, may become oversensitive post-TBI. The organic relationship between TBI and OCD is yet unclear, but areas that may be implicated include damage to the right orbital cortex, right orbitofrontal cortex, frontal cortex, occipital cortex, basal ganglia (especially caudate), and the limbic system [111].

#### **Personality Disorders**

Several studies have reported an increase in rates of personality disorders following TBI [85, 116].

Koponen et al. [116] reported that roughly one third of persons with TBI in their sample had personality disorders, noting that personality disorders are also prevalent in persons prone to TBI and may reflect premorbid factors rather than postinjury conditions exclusively. The specific diagnoses reported vary by research study, including avoidant, borderline, narcissistic [94], antisocial, and obsessive-compulsive [116]. Such authors have noted that changes in personality are likely more common than developing symptoms consistent with an identified personality disorder and that these changes are associated with damage or changes to subcortical and frontal systems [85]. Moreover, if one looks at the nature of the behavioral disturbances after moderate to severe TBI, the clinical presentation is more reflective of neurobehavioral disturbance rather than true personality change or personality disorder.

#### Schizophrenia and Psychosis

Schizophrenia has not been found to be strongly associated with TBI, with a minor, if any, relative risk of schizophrenia post-TBI, 0.5 as compared to community rates of 0.7 % [85]. Several authors have concluded that there is no evidence and that it is very unlikely that TBI causes or increases the risk of schizophrenia [85, 117, 118]. Mixed evidence has been reported regarding a relationship between TBI before age 10 and increased risk of schizophrenia and/or earlier age of onset [117, 119, 120]. Kim [118] explained that the research evidence provides contradictory results concerning a possible stress-diathesis model in which TBI may affect the risk of expression of schizophrenia in persons at risk for the disorder, highlighting the need for additional research to be conducted.

TBI has been associated with a small, but significant, risk of non-schizophrenic psychosis that is unrelated to age of injury and family history [117]. This relationship could be related to prodromal symptoms, such that preexisting cognitive impairments or behaviors put persons with early psychotic or pre-schizophrenic symptoms at risk for TBI [118, 121].

### **Substance Abuse Disorders**

Although many individuals who sustain a brain injury may have been using substances at the time of their injury, and those with TBI who consumed alcohol prior to their injuries drank at a higher rather than their peers [122], new onset substance abuse has not been strongly associated with TBI [85, 123]. Substance abuse is not uncommon post-TBI, but rates are typically lower than in the general community [85], although there is some literature to indicate that there is a significant percentage of individuals who had premorbid histories of substance abuse that return to using at pre-injury levels relatively quickly [78]. Kreutzer et al. [124] reported that alcohol consumption was infrequent in the first year post-TBI, but approximately 25 % of the sample increased consumption over the second year. Illicit drug use did not show the same increases over time and remained dramatically decreased relative to pre-injury rates [125]. Although there is evidence for infrequent new onset substance abuse post-TBI [123], some studies have shown that 15-20 % of abstinent or infrequent users of alcohol significantly increased their consumption, such that they used substance abuse treatment or were considered moderate to heavy drinkers post-TBI [126, 127].

With regard to the effects of substance abuse on outcomes, Carroll et al. [8] demonstrated that substance use was associated with worse outcomes in persons with TBI. Interestingly, the effects of pre-injury substance abuse in those with TBI have been closely linked to reduced employment [128, 129] and lower reported subjective well-being [82]. Persons with moderate to severe TBI who returned to work and were on average 16 months post-injury reported consuming more alcohol than those who were unemployed post-injury, which may be related to increased income to purchase alcohol and to use of alcohol as a coping strategy for increased work-related stress [130]. Additionally, higher risk of developing problematic alcohol consumption post-injury has been associated with being male, being single or divorced, and less than high school education [131].

## Mitigating Factors

#### **Penetrating Head Injuries**

Penetrating head injuries, often representing gunshot wounds (GSW), have been studied in comparison to closed head injuries (e.g., as the result of a motor vehicle collision). Survival rates of GSWs to the head are poor, with a majority of persons dying at the scene of the injury or within 48 h [132]. For those who survive, having a GCS of less than eight has been associated with especially poor outcomes, such as death or severe injury [73]. For example, Selden et al. [133] reported that among 67 persons with self-inflicted GSWs, 98 % who had initial GCS scores of 8 or less died, whereas 91 % with GCS scores higher than eight survived. In a more recent study, Wertheimer et al. [67] reported that those who survived penetrating injuries, even with low initial GCS, showed improvement in functioning during rehabilitation.

Several predictors have been identified for post-injury cognitive functioning, including brain-volume-loss, location of penetration, and pre-injury factors. Dikmen et al. [1] reported that total brain-volume loss was strongly associated with cognitive outcomes. Additionally, persons with penetrating injuries were at risk of accelerated age-related changes. The location of the penetrating injury predicted the cognitive domains that were adversely affected, but was least predictive of post-injury intelligence. The authors reported that pre-injury intelligence and size of lesion were most predictive.

A body of research has provided evidence that the prognoses for penetrating head injuries are similar to those of motor vehicle-related injuries, if the individual survives the penetrating injury [134]. Severe injuries, whether penetrating or closed head, have been found to have similar courses of recovery in functional and psychosocial domains [67]. Additionally, cognitive outcomes at 12–14 years post-injury have also been found to be similar among Vietnam veterans with penetrating and non-penetrating injuries [135]. Ylioja et al. [136] reported a similar degree of cognitive recovery and generally comparable cognitive sequelae at 1–2 years post-injury, even without accounting for injury location, noting a few differences in performance across cognitive domains. Specifically, those with penetrating TBIs were found to have better verbal learning and fine motor dexterity, but possibly worse attention within a few weeks after injury; at 1–2 years post-injury, they again had better fine motor dexterity but worse verbal generativity. Attentional and verbal learning abilities were comparable at the follow-up assessments.

Some studies have, however, described statistically worse outcomes, including decreased functionality, community integration, and employment, for violent injuries (e.g., penetrating and blunt head trauma) as compared to a nonviolent comparison group (e.g., MVC) [137, 138]. Wertheimer et al. [67] noted that although statistically significant differences have been identified, the actual differences were small and not clinically meaningful, which is consistent with the information noted above.

#### **APOE-4 Allele**

The presence of the APOE-4 allele has been studied in its relationship to TBI. Smith et al. [139] reported that among fatal cases of TBI, APOE4 was associated with severe secondary ischemic brain damage and more frequent moderate to severe contusions. They have hypothesized that APOE4 may also be associated with worse initial severity, possibly affecting GCS scores. In a meta-analysis, Zhou et al. [140] found, however, that the APOE4 allele was not associated with initial TBI severity. The APOE4 allele was associated with increased risk of worse outcomes at 6 months post-injury in their meta-analysis. For example, in a prospective cohort study, Teasdale et al. [141] found that persons with TBI and the APOE4 allele were more than two times as likely to have poor outcomes (e.g., Glascow Outcome Scale) at 6 months. Possible mechanisms for this association have been hypothesized, including amyloid deposits, neurofibrillary tangles, oxidative stress, disruption of cholinergic transmission, and changes in CNS degeneration and regeneration; however, the mechanism behind this association is currently unknown [142]. In animal models of subarachnoid hemorrhage [143] and nonpenetrating brain injury [144], animals carrying APOE4 have more neuroinflammation, glial activation, and neuronal injury than those without. The literature is mixed in regard to the presence of differences in neuropsychological performances among persons with and without the APOE4 allele after TBI [60, 145–148]. In summary, in individuals with moderate to severe TBI, there is some evidence that the presence of the APOE4 allele is associated with poorer outcome, but additional research needs to be conducted in order to elucidate the mechanisms and impact of functional outcomes.

#### **Caregivers and Family Functioning**

The outcomes of persons with TBI may be affected by the well-being of their caregivers and families. Among persons with moderate to severe TBI, outcomes, such as functionality, life satisfaction, and well-being, have been shown to be associated with characteristics of the caregiver [149]. Specifically, caregivers' level of distress and life satisfaction was predictive of functional outcome, even after controlling for severity of injury, age, education, and cognitive functioning of persons with TBI. Additionally, persons with TBI were found to be more distressed and less satisfied with life when their caregivers had less perceived support and greater behavioral dyscontrol in their families. Interestingly, the strongest individual predictor of emotional distress among persons with TBI was their caregivers' level of emotional distress; likewise, the strongest predictor of life satisfaction among persons with TBI was their caregivers' perceived level of social support. Sady et al. [150] reported that better outcomes in productivity and social integration were associated with more caregivers' perceived social support among persons with severe TBIs. Among persons with complicated mild or moderate injuries, they found better social integration was associated with less caregiver distress, as well as more home integration with greater family functioning.

#### **Ethnicity and Minority Status**

Ethnicity and minority status has been associated with poorer outcomes following TBI (for a review, see Gary et al. [151]). Specifically in regard to productivity and employment outcomes, ethnic minorities have been found to be twice as likely to be nonproductive or unemployed [152, 153]. Sherer et al. [152] reported that decreased productivity was found even after controlling for preinjury productivity, education, and cause of injury. These authors argued that race and ethnicity may be confounded with a number of factors such as access to and utilization of services making it impossible to determine the direct effect, if any, of race and ethnicity on outcome.

Sander et al. [154] also found that race/ethnicity was related to reduced community integration even after accounting for age, education, injury severity, and income, such that African Americans and Hispanics had lower scores than Caucasians. Low income, however, was also found to be related to reduced aspects of community integration, above and beyond race/ethnicity.

Similarly, Arango-Lasprilla et al. [155] found worse functional outcomes at 1-year, as measured by the Disability Rating Scale (DRS), Functional Independence Measure (FIM), CIQ, and Extended Glasgow Outcome Scale (GOS-E), post-injury for minority TBI survivors, even after controlling for sociodemographic and injury-related factors. They suggest these differences may be attributed to a direct or indirect result of therapy services which may be obtained with less frequency and lower intensity by persons from minority groups. Burnett et al. [156] noted that minorities in their study received significantly fewer minutes of therapies and less intense treatment as compared to those in the majority group. In contrast, Rosenthal et al. [157] did not find significant differences in functional outcomes associated with minority status at admission, discharge, or 1-year postinjury assessments.

Regarding neuropsychological assessment outcomes, Kennepohl et al. [158] found that lower levels of acculturation were associated with reduced performances on neuropsychological tests among African Americans with TBI, even after controlling for demographic variables and injury characteristics. This study found that even orientation testing was affected by acculturation. As such, the consideration of cultural factors is indicated even in basic assessment.

#### **Environmental Factors**

Another mitigating influence on outcomes after TBI is environmental factors, such as access to care, neighborhoods, and transportation. These factors have the potential to facilitate or impede outcomes. The Craig Hospital Inventory of Environmental Factors (CHIEF) has been developed to assess the effects of perceived barriers [159]. Subdomains of the CHIEF include physical and structural barriers, attitudinal and support barriers, barriers to services and assistance, policy barriers, and barriers at work and school. Whiteneck et al. [160] found that persons 1-year post-TBI reported that the five barriers affecting them most were availability of transportation, situational environment (e.g., noise, lighting, crowding), government policies, the attitudes of others at home, and the natural environment. They also found that perception of more barriers was associated with reduced participation as measured by the CHART (e.g., mobility, productivity, mobility), as well as lower life satisfaction.

Corrigan and Bogner [161] found that neighborhood of residence explained variance in overall outcome and satisfaction above and beyond individual characteristics. Findings regarding outcomes in various settings (e.g., rural vs. urban) have been variable (e.g., [162–165]). Corrigan and Bogner [161] suggested that this dichotomy is likely too broad and complex and, as a result, is a poor index of environmental influence.

Devitt et al. [72] reported that poorer access to transportation was predictive of reduced self-care and productivity/occupation outcomes. As mentioned previously, Kreutzer et al. [74] found that driving independence was strongly related to work stability after TBI. More research is needed to facilitate better understanding of the relationship between environmental factors and outcomes.

#### Litigation and Financial Incentives

As mentioned in the introduction to this chapter, outcomes in TBI typically demonstrate a doseresponse relationship with more severe injuries resulting in more extensive impairments. It is noteworthy that an inverse dose-response relationship has been identified as well, such that persons with minor compensable injuries exhibit more "pseudo-abnormalities," such as cognitive, motor, and postconcussion symptoms, than those with more severe injuries, especially when involved in litigation [152, 166, 167]. Carroll et al. [8] reported that compensation and/or litigation was the only consistent predictor of continued symptoms among those with mTBI in their meta-analysis. As discussed earlier, symptoms typically resolve quickly following mild injuries. Additionally, Paniak et al. [7] found that persons seeking compensation reported significantly more symptoms than those uninvolved in that type of process. Greiffenstein and Baker [152] conclude that persistent postconcussive symptoms following mTBI are strongly associated with simulation or exaggeration of symptoms, although this explanation may not have been the initial cause of symptoms or the only reason for their continuation (e.g., biopsychosocial factors). Binder and Rohling [166] reported that late onset of symptoms post-TBI was also much more likely among persons seeking compensation. Interestingly, they also found that persons with more mTBIs are more likely to seek monetary compensation for their injuries (e.g., workers' compensation) than are those with moderate to severe TBIs. From their meta-analysis, they concluded that if financial incentives were eradicated, symptoms would be reduced or even eliminated among some people with TBI.

Among persons with moderate to severe TBI, the effects of litigation on symptom reporting have been mixed. Tsanadis et al. [167] found that those involved in litigation reported more postconcussion symptoms than did non-litigating individuals. In contrast, Wood and Rutterford [74] reported no difference in outcomes regarding litigation status among people with more severe injuries.

# Conclusion

Although outcome prediction is a challenging task in TBI rehabilitation, it is essential for planning current and long-term rehabilitation goals, identifying the necessity of future assistance and educating patients and families about possible long-term psychosocial changes. As has been discussed in this chapter, outcome prediction needs to take into account injury characteristics such as severity and etiology, but even more important are factors related to premorbid functioning, situational factors, emotional functioning, and caregiver/family issues. Brain injury characteristics can be helpful in gauging needs for resources and treatment planning, especially when delineating needed services for those with mild versus moderate or severe injuries, but these characteristics are only modestly related to many of the outcomes noted in this chapter. Instead, clinicians need to assess contextual factors, comorbidities, and even non-neurologic issues when developing plans for post-acute rehabilitation and contemplating outcomes with medical professionals, family members, and persons with TBI.

Acknowledgment Preparation of this chapter was partially supported by U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grant H133A080044.

#### References

- Dikmen, S. S., et al. (2009). Cognitive outcome following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 24(6), 430–438.
- Rohling, M. L., Meyers, J. E., & Millis, S. R. (2003). Neuropsychological impairment following traumatic brain injury: A dose–response analysis. *Clinical Neuropsychology*, 17(3), 289–302.
- Malec, J. F., et al. (2007). The mayo classification system for traumatic brain injury severity. *Journal of Neurotrauma*, 24(9), 1417–1424.
- 4. Erlanger, D., et al. (2003). Symptom-based assessment of the severity of a concussion. *Journal of Neurosurgery*, 98(3), 477–484.
- Levin, H. S., et al. (1987). Neurobehavioral outcome following minor head injury: A three-center study. *Journal of Neurosurgery*, 66(2), 234–243.

- Meares, S., et al. (2008). Mild traumatic brain injury does not predict acute postconcussion syndrome. *Journal of Neurology, Neurosurgery, and Psychiatry*, 79(3), 300–306.
- Paniak, C., et al. (2002). Patient complaints within 1 month of mild traumatic brain injury: A controlled study. *Archives of Clinical Neuropsychology*, 17(4), 319–334.
- Carroll, L. J., et al. (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(Suppl), 84–105.
- Larrabee, G. J. (2005). Mild traumatic brain injury. In G. J. Larrabee (Ed.), *Forensic neuropsychology : A scientific approach* (pp. 209–236). New York: Oxford University Press.
- American Psychiatric Association. (2000). *Diagnostic* criteria from DSM-IV-TR2000 (Vol. xii). Washington, DC: American Psychiatric Association. 370p.
- 11. Fear, N. T., et al. (2009). Symptoms of postconcussional syndrome are non-specifically related to mild traumatic brain injury in UK Armed Forces personnel on return from deployment in Iraq: An analysis of self-reported data. *Psychological Medicine*, 39(8), 1379–1387.
- 12. Meares, S., et al. (2011). The prospective course of postconcussion syndrome: The role of mild traumatic brain injury. *Neuropsychology*, *25*(4), 454–465.
- Iverson, G. L., & Lange, R. T. (2003). Examination of "postconcussion-like" symptoms in a healthy sample. *Applied Neuropsychology*, 10(3), 137–144.
- Lees-Haley, P. R., & Brown, R. S. (1993). Neuropsychological complaint base rates of 170 personal injury claimants. *Archives of Clinical Neuropsychology*, 8(3), 203–209.
- Dikmen, S., et al. (2010). Rates of symptom reporting following traumatic brain injury. *Journal of the International Neuropsychological Society*, 16(3), 401–411.
- Iverson, G., Zasler, N. D., & Lange, R. T. (2007). Postconcussive disorder. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles* and practice (pp. 373–405). New York: Demos.
- de Leon, M. B., et al. (2009). Baseline predictors of fatigue 1 year after mild head injury. Archives of Physical Medicine and Rehabilitation, 90(6), 956–965.
- Belanger, H. G., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concussion: A meta-analysis. *Journal of International Neuropsychological Society*, 11(4), 345–357.
- McCrea, M., et al. (2003). Acute effects and recovery time following concussion in collegiate football players—The NCAA Concussion Study. *Journal of the American Medical Association*, 290(19), 2556–2563.
- Heitger, M. H. (2006). Motor deficits and recovery during the first year following mild closed head injury. *Brain Injury*, 20(8), 807–824.
- Dikmen, S. S., et al. (1995). Neuropsychological outcome at 1-year post head-injury. *Neuropsychology*, 9(1), 80–90.

- Suhr, J. A., & Gunstad, J. (2002). Postconcussive symptom report: The relative influence of head injury and depression. *Journal of Clinical and Experimental Neuropsychology*, 24(8), 981–993.
- Ferguson, R. J., et al. (1999). Postconcussion syndrome following sports-related head injury: Expectation as etiology. *Neuropsychology*, 13(4), 582–589.
- Gunstad, J., & Suhr, J. A. (2004). Cognitive factors in postconcussion syndrome symptom report. *Archives* of *Clinical Neuropsychology*, 19(3), 391–405.
- Iverson, G. L., et al. (2010). "Good old days" bias following mild traumatic brain injury. *Clinical Neuropsychology*, 24(1), 17–37.
- Mittenberg, W., et al. (1992). Symptoms following mild head-injury—Expectation as etiology. *Journal of Neurology, Neurosurgery, and Psychiatry*, 55(3), 200–204.
- Belanger, H. G., Spiegel, E., & Vanderploeg, R. D. (2010). Neuropsychological performance following a history of multiple self-reported concussions: A meta-analysis. *Journal of International Neuropsychological Society*, *16*(2), 262–267.
- McCrea, M. (2007). Sports neuropsychology: Assessment and management of traumatic brain injury. *Clinical Neuropsychologist*, 21(4), 717–722.
- 29. Loosemore, M., Knowles, C. H., & Whyte, G. P. (2008). Amateur boxing and risk of chronic traumatic brain injury: Systematic review of observational studies. *British Journal of Sports Medicine*, 42(11), 864–867.
- Hahnel, S., et al. (2008). Prevalence of cerebral microhemorrhages in amateur boxers as detected by 3TMR imaging. *American Journal of Neuroradiology*, 29(2), 388–391.
- Randolph, C., & Kirkwood, M. W. (2009). What are the real risks of sport-related concussion, and are they modifiable? *Journal of the International Neuropsychological Society*, 15(4), 512–520.
- McCrea, M., et al. (2009). Effects of a symptom-free waiting period on clinical outcome and risk of reinjury after sport-related concussion. *Neurosurgery*, 65(5), 876–882. discussion 882–3.
- 33. Kors, E. E., et al. (2001). Delayed cerebral edema and fatal coma after minor head trauma: Role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. *Annals* of *Neurology*, 49(6), 753–760.
- 34. Stam, A. H., et al. (2009). Early seizures and cerebral oedema after trivial head trauma associated with the CACNA1A S218L mutation. *Journal of Neurology, Neurosurgery, and Psychiatry, 80*(10), 1125–1129.
- Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery*, 27(3), 422–428.
- 36. Kashluba, S., et al. (2008). Neuropsychologic and functional outcome after complicated mild traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(5), 904–911.

- Miller, J. D., Murray, L. S., & Teasdale, G. M. (1990). Development of a traumatic intracranial hematoma after a minor head-injury. *Neurosurgery*, 27(5), 669–673.
- Hsiang, J. N., et al. (1997). High-risk mild head injury. *Journal of Neurosurgery*, 87(2), 234–238.
- 39. van der Naalt, J., et al. (1999). One year outcome in mild to moderate head injury: The predictive value of acute injury characteristics related to complaints and return to work. *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 66(2), 207–213.
- Borgaro, S. R., et al. (2003). Cognitive and affective sequelae in complicated and uncomplicated mild traumatic brain injury. *Brain Injury*, 17(3), 189–198.
- Jiang, J. Y., et al. (2002). Early indicators of prognosis in 846 cases of severe traumatic brain injury. *Journal of Neurotrauma*, 19(7), 869–874.
- 42. Roebuck-Spencer, T., & Sherer, M. (2008). Moderate and severe traumatic brain injury. In J. E. Morgan & J. H. Ricker (Eds.), *Textbook of clinical neuropsychology* (pp. 411–429). New York: Taylor & Francis.
- McKinley, W. W., & Watkins, A. J. (1999). Cognitive and behavioral effects of brain injury. In M. Rosenthal et al. (Eds.), *Rehabilitation of the adult* and child with traumatic brain injury (pp. 74–86). Philadelphia, PA: Davis.
- 44. Stuss, D. T., et al. (1999). The acute period of recovery from traumatic brain injury: Posttraumatic amnesia or posttraumatic confusional state? *Journal* of *Neurosurgery*, 90(4), 635–643.
- 45. Reyes, R. L., Bhattacharyya, A. K., & Heller, D. (1981). Traumatic head injury: Restlessness and agitation as prognosticators of physical and psychologic improvement in patients. *Archives of Physical Medicine and Rehabilitation*, 62(1), 20–23.
- 46. Sherer, M., et al. (2008). Effect of severity of posttraumatic confusion and its constituent symptoms on outcome after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(1), 42–47.
- Fork, M., et al. (2005). Neuropsychological sequelae of diffuse traumatic brain injury. *Brain Injury*, 19(2), 101–108.
- Mathias, J. L., & Wheaton, P. (2007). Changes in attention and information-processing speed following severe traumatic brain injury: A meta-analytic review. *Neuropsychology*, 21(2), 212–223.
- Felmingham, K. L., Baguley, I. J., & Green, A. M. (2004). Effects of diffuse axonal injury on speed of information processing following severe traumatic brain injury. *Neuropsychology*, 18(3), 564–571.
- Sohlberg, M. M., & Mateer, C. A. (1990). Evaluation and treatment of communication skills. In J. S. Kreutzer & P. Wehman (Eds.), *Community integration following traumatic brain injury* (pp. 67–84). Baltimore, MD: Brookes.
- Dikmen, S., et al. (1987). Memory and head-injury severity. *Journal of Neurology, Neurosurgery, and Psychiatry*, 50(12), 1613–1618.

- Novack, T. A., et al. (2000). Cognitive and functional recovery at 6 and 12 months post-TBI. *Brain Injury*, 14(11), 987–996.
- Christensen, B. K., et al. (2008). Recovery of cognitive function after traumatic brain injury: A multilevel modeling analysis of Canadian outcomes. *Archives of Physical Medicine and Rehabilitation*, 89(12 Suppl), S3–S15.
- Schretlen, D. J., & Shapiro, A. M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15(4), 341–349.
- Millis, S. R., et al. (2001). Long-term neuropsychological outcome after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 16(4), 343–355.
- Kersel, D. A., et al. (2001). Neuropsychological functioning during the year following severe traumatic brain injury. *Brain Injury*, 15(4), 283–296.
- Draper, K., & Ponsford, J. (2008). Cognitive functioning ten years following traumatic brain injury and rehabilitation. *Neuropsychology*, 22(5), 618–625.
- Till, C., et al. (2008). Postrecovery cognitive decline in adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(12), S25–S34.
- Bercaw, E. L., et al. (2011). Changes in neuropsychological performance after traumatic brain injury from inpatient rehabilitation to 1-year follow-up in predicting 2-year functional outcomes. *Clinical Neuropsychology*, 25(1), 72–89.
- 60. Hanks, R. A., et al. (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.
- Dikmen, S. S., et al. (2003). Outcome 3 to 5 years after moderate to severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(10), 1449–1457.
- van Velzen, J. M., et al. (2009). How many people return to work after acquired brain injury? A systematic review. *Brain Injury*, 23(6), 473–488.
- 63. Wehman, P., et al. (1993). Return to work: Supported employment strategies, costs, and outcome data. In D. F. Thomas, F. E. Menz, & D. C. McAlees (Eds.), *Community-based employment following traumatic brain injury*. Menomonie, WI: University of Wisconsin-Stout.
- Dikmen, S. S., et al. (1994). Employment following traumatic head injuries. Archives of Neurology, 51(2), 177–186.
- Temkin, N. R., et al. (2009). Social functioning after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 24(6), 460–467.
- 66. Cifu, D. X., et al. (1997). Acute predictors of successful return to work 1 year after traumatic brain injury: A multicenter analysis. *Archives of Physical Medicine and Rehabilitation*, 78(2), 125–131.
- Doctor, J. N., et al. (2005). Workers' risk of unemployment after traumatic brain injury: A normed

comparison. Journal of International Neuropsychological Society, 11(6), 747–752.

- Crepeau, F., & Sherzer, P. (1993). Predictors and indicators of work status after traumatic brain injury: A meta-analysis. *Neuropsychological Rehabilitation*, 3, 5–35.
- Sherer, M., et al. (2002). Neuropsychological assessment and employment outcome after traumatic brain injury: A review. *Clinical Neuropsychology*, 16(2), 157–178.
- Putnam, S. H., & Fichtenberg, N. L. (1999). Neuropsychological examination of the patient with traumatic brain injury. In M. Rosenthal et al. (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 147–166). Philadelphia, PA: F.A. Davis Company.
- 71. Wagner, A. K., et al. (2002). Return to productive activity after traumatic brain injury: Relationship with measures of disability, handicap, and community integration. *Archives of Physical Medicine and Rehabilitation*, 83(1), 107–114.
- Devitt, R., et al. (2006). Prediction of long-term occupational performance outcomes for adults after moderate to severe traumatic brain injury. *Disability* and *Rehabilitation*, 28(9), 547–559.
- Machamer, J., et al. (2005). Stability of employment after traumatic brain injury. *Journal of International Neuropsychological Society*, 11(7), 807–816.
- Kreutzer, J. S., et al. (2003). Moderating factors in return to work and job stability after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 18(2), 128–138.
- Dikmen, S. S., et al. (1995). One year psychosocial outcome in head injury. *Journal of International Neuropsychological Society*, 1(1), 67–77.
- Hart, T., et al. (2003). The relationship between neuropsychologic function and level of caregiver supervision at 1 year after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 221–230.
- Oddy, M., Humphrey, M., & Uttley, D. (1978). Subjective impairment and social recovery after closed head injury. *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 41(7), 611–616.
- Corrigan, J. D., Smith-Knapp, K., & Granger, C. V. (1998). Outcomes in the first 5 years after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79(3), 298–305.
- Evans, C. C., et al. (2005). Early impaired selfawareness, depression, and subjective well-being following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20(6), 488–500.
- Smith, J. L., Magill-Evans, J., & Brintnell, S. (1998). Life satisfaction following traumatic brain injury. *Canadian Journal of Rehabilitation*, 11, 131–140.
- Brown, M., & Vandergoot, D. (1998). Quality of life for individuals with traumatic brain injury: Comparison with others living in the community. *The Journal of Head Trauma Rehabilitation*, 13(4), 1–23.

- Corrigan, J. D., et al. (2001). Life satisfaction after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 16(6), 543–555.
- Pierce, C. A., & Hanks, R. A. (2006). Life satisfaction after traumatic brain injury and the World Health Organization model of disability. *American Journal of Physical Medicine and Rehabilitation*, 85(11), 889–898.
- 84. Meythaler, J. M., et al. (2007). Neuropharmacology: A rehabilitation perspective. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (pp. 1023–1036). New York: Demos Medical Publishing.
- van Reekum, R., Cohen, T., & Wong, J. (2000). Can traumatic brain injury cause psychiatric disorders? *Journal of Neuropsychiatry and Clinical Neurosciences*, 12(3), 316–327.
- 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.
- Dikmen, S. S., et al. (2004). Natural history of depression in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 85(9), 1457–1464.
- Bourdon, K. H., et al. (1992). Estimating the prevalence of mental disorders in U.S. adults from the epidemiologic catchment area survey. *Public Health Reports*, 107(6), 663–668.
- Robinson, R. G., & Jorge, R. (1994). Mood disorders. In J. M. Silver, S. C. Yudofsky, & R. E. Hales (Eds.), *Neuropsychiatry of traumatic brain injury* (pp. 219– 250). Washington, DC: American Psychiatric Press.
- Jorge, R. E., et al. (2004). Major depression following traumatic brain injury. *Archives of General Psychiatry*, 61(1), 42–50.
- Jorge, R. E., et al. (1993). Depression and anxiety following traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 5(4), 369–374.
- 92. Seel, R. T., et al. (2003). Depression after traumatic brain injury: A National Institute on Disability and Rehabilitation Research Model Systems multicenter investigation. Archives of Physical Medicine and Rehabilitation, 84(2), 177–184.
- Ashman, T. A., et al. (2004). Psychiatric challenges in the first 6 years after traumatic brain injury: Crosssequential analyses of Axis I disorders. *Archives of Physical Medicine and Rehabilitation*, 85(4 Suppl 2), S36–S42.
- van Reekum, R., et al. (1996). Psychiatric disorders after traumatic brain injury. *Brain Injury*, 10(5), 319–327.
- Fann, J. R. (1997). Traumatic brain injury and psychiatry. *Journal of Psychosomatic Research*, 43(4), 335–343.
- Seel, R. T., Macciocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major

depression after moderate to severe TBI. *The Journal* of *Head Trauma Rehabilitation*, 25(2), 99–112.

- 97. Silver, J. M., Hales, R. E., & Yudofsky, S. C. (1992). Neuropsychiatric aspects of traumatic brain injury. In S. C. Yudofsky & R. E. Hales (Eds.), *The American Psychiatric Press textbook of neuropsychiatry* (pp. 363–395). Washington, DC: American Psychiatric Press.
- Koenigs, M., et al. (2008). Distinct regions of prefrontal cortex mediate resistance and vulnerability to depression. *Journal of Neuroscience*, 28(47), 12341–12348.
- Davidson, R. J., et al. (2002). Neural and behavioral substrates of mood and mood regulation. *Biological Psychiatry*, 52(6), 478–502.
- 100. Jorge, R. E., & Starkstein, S. E. (2005). Pathophysiologic aspects of major depression following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20(6), 475–487.
- 101. 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.
- 102. Rosenthal, M., Christensen, B. K., & Ross, T. P. (1998). Depression following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79(1), 90–103.
- 103. Kelly, D. F., et al. (2006). Neurobehavioral and quality of life changes associated with growth hormone insufficiency after complicated mild, moderate, or severe traumatic brain injury. *Journal of Neurotrauma*, 23(6), 928–942.
- 104. Shukla, S., et al. (1987). Mania following head trauma. *The American Journal of Psychiatry*, 144(1), 93–96.
- 105. Pope, H. G., Jr., et al. (1988). Head injury, bipolar disorder, and response to valproate. *Comprehensive Psychiatry*, 29(1), 34–38.
- 106. Starkstein, S. E., et al. (1987). Mania after brain injury. A controlled study of causative factors. *Archives of Neurology*, 44(10), 1069–1073.
- 107. Epstein, R. S., & Ursano, R. J. (1994). Anxiety disorders. In J. M. Silver, S. C. Yudofsky, & R. E. Hales (Eds.), *Neuropsychiatry of traumatic brain injury* (pp. 285– 311). Washington DC: American Psychiatric Press.
- Grafman, J., et al. (1986). The effects of lateralized frontal lesions on mood regulation. *Brain*, 109(Pt 6), 1127–1148.
- 109. Mayou, R., Bryant, B., & Duthie, R. (1993). Psychiatric consequences of road traffic accidents. *British Medical Journal*, 307(6905), 647–651.
- Bryant, R. A., & Harvey, A. G. (1998). Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury. *The American Journal of Psychiatry*, 155(5), 625–629.
- 111. Hiott, D. W., & Labbate, L. (2002). Anxiety disorders associated with traumatic brain injuries. *NeuroRehabilitation*, 17(4), 345–355.
- Ohry, A., Rattok, J., & Solomon, Z. (1996). Posttraumatic stress disorder in brain injury patients. *Brain Injury*, 10(9), 687–695.

- Childers, M. K., et al. (1998). Obsessional disorders during recovery from severe head injury: Report of four cases. *Brain Injury*, 12(7), 613–616.
- 114. Daar, E. S., et al. (2003). Improving adherence to antiretroviral therapy. *The AIDS Reader*, 13(2), 81–82. 85–6, 88–90.
- 115. Drummond, L. M., & Gravestock, S. (1988). Delayed emergence of obsessive-compulsive neurosis following head injury. Case report and review of its theoretical implications. *British Journal of Psychiatry*, 153, 839–842.
- 116. Koponen, S., et al. (2011). Axis I and II psychiatric disorders in patients with traumatic brain injury: A 12-month follow-up study. *Brain Injury*, 25(11), 1029–1034.
- 117. Harrison, G., et al. (2006). Risk of schizophrenia and other non-affective psychosis among individuals exposed to head injury: Case control study. *Schizophrenia Research*, 88(1–3), 119–126.
- Kim, E. (2008). Does traumatic brain injury predispose individuals to develop schizophrenia? *Current Opinion in Psychiatry*, 21(3), 286–289.
- AbdelMalik, P., et al. (2003). Childhood head injury and expression of schizophrenia in multiply affected families. *Archives of General Psychiatry*, 60(3), 231–236.
- Wilcox, J. A., & Nasrallah, H. A. (1987). Childhood head trauma and psychosis. *Psychiatry Research*, 21(4), 303–306.
- 121. Fann, J. R., et al. (2004). Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. *Archives of General Psychiatry*, 61(1), 53–61.
- 122. Kreutzer, J. S., et al. (1990). Alcohol use among persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 5(3), 9–20.
- 123. Whelan-Goodinson, R., et al. (2009). Psychiatric disorders following traumatic brain injury: Their nature and frequency. *The Journal of Head Trauma Rehabilitation*, 24(5), 324–332.
- 124. Kreutzer, J. S., et al. (1996). A prospective longitudinal multicenter analysis of alcohol use patterns among persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *11*(5), 58–69.
- 125. Kreutzer, J. S., Witol, A. D., & Marwitz, J. H. (1996). Alcohol and drug use among young persons with traumatic brain injury. *Journal of Learning Disabilities*, 29(6), 643–651.
- 126. Bombardier, C. H., et al. (2003). The natural history of drinking and alcohol-related problems after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 185–191.
- 127. Corrigan, J. D., Rust, E., & Lamb-Hart, G. L. (1995). The nature and extent of substance abuse problems among persons with traumatic brain injuries. *Journal of Head Trauma Rehabilitation*, 10(3), 29–45.
- Corrigan, J. D., et al. (1997). Systematic bias in outcome studies of persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 78(2), 132–137.

- 129. Sherer, M., et al. (1999). Contribution of functional ratings to prediction of longterm employment outcome after traumatic brain injury. *Brain Injury*, 13(12), 973–981.
- 130. Sander, A. M., Kreutzer, J. S., & Fernandez, C. C. (1997). Neurobehavioral functioning substance abuse, and employment after brain injury: implications for vocational rehabilitation. *Journal of Head Trauma Rehabilitation*, 12(5), 28–41.
- 131. Bombardier, C. H., Rimmele, C. T., & Zintel, H. (2002). The magnitude and correlates of alcohol and drug use before traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 83(12), 1765–1773.
- 132. Rolston, R., et al. (2002). New cytogenetic variant, insertion (15;17)(q22;q12q21), in an adolescent with acute promyelocytic leukemia. *Cancer Genetics and Cytogenetics*, 134(1), 55–59.
- 133. Pendleton, A. R., & Machamer, C. E. (2006). Differential localization and turnover of infectious bronchitis virus 3b protein in mammalian versus avian cells. *Virology*, 345(2), 337–345.
- Krishnaraju, R. K., Hart, T. C., & Schleyer, T. K. (2003). Comparative genomics and structure prediction of dental matrix proteins. *Advances in Dental Research*, 17, 100–103.
- 135. Kreutzer, C., et al. (2003). A new method for reliable fenestration in extracardiac conduit Fontan operations. *Annals of Thoracic Surgery*, 75(5), 1657–1659.
- 136. Hart, T. C., et al. (2003). Novel ENAM mutation responsible for autosomal recessive amelogenesis imperfecta and localised enamel defects. *Journal of Medical Genetics*, 40(12), 900–906.
- 137. Kinane, D. F., & Hart, T. C. (2003). Genes and gene polymorphisms associated with periodontal disease. *Critical Reviews in Oral Biology and Medicine*, 14(6), 430–449.
- 138. Stall, R., et al. (2003). Association of co-occurring psychosocial health problems and increased vulnerability to HIV/AIDS among urban men who have sex with men. *American Journal of Public Health*, 93(6), 939–942.
- 139. Sherer, M., Hart, T., & Nick, T. G. (2003). Measurement of impaired self-awareness after traumatic brain injury: A comparison of the patient competency rating scale and the awareness questionnaire. *Brain Injury*, 17(1), 25–37.
- 140. Fodrie, T. Y., et al. (2009). Evaluation of a real-time polymerase chain reaction assay using analytespecific reagents for detection of methicillinresistant *Staphylococcus aureus* nasal carriage. *Analytical and Quantitative Cytology and Histology*, 31(6), 410–416.
- 141. Scorpio, D. G., et al. (2009). Anaplasma phagocytophilum propagation is enhanced in human complementcontaining medium. *Clinical Microbiology and Infection, 15*(Suppl 2), 48–49.
- 142. Aubert, B., et al. (2009). Search for di-muon decays of a light scalar boson in radiative transitions Upsilon → gammaA0. *Physical Review Letters*, 103(8), 081803.

- 143. Adamson, P., et al. (2008). Measurement of neutrino oscillations with the MINOS detectors in the NuMI beam. *Physical Review Letters*, 101(13), 131802.
- 144. Sherer, E. C., et al. (2008). Efficient and accurate characterization of the Bergman cyclization for several enediynes including an expanded substructure of esperamicin A1. *The Journal of Physical Chemistry B*, 112(51), 16917–16934.
- 145. Evans, C. C., et al. (2008). Evaluation of an interdisciplinary team intervention to improve therapeutic alliance in post-acute brain injury rehabilitation. *The Journal of Head Trauma Rehabilitation*, 23(5), 329–338.
- 146. Sherer, D. M., et al. (2008). Nomograms of the axial transverse diameter of the fetal foramen magnum between 14 and 40 weeks' gestation. *Journal of Ultrasound in Medicine*, 27(9), 1297–1303.
- 147. Chan, W. T., et al. (2008). Murine leukemia virus spreading in mice impaired in the biogenesis of secretory lysosomes and Ca<sup>2+</sup>-regulated exocytosis. *PLoS One*, *3*(7), e2713.
- 148. Raad, I. I., et al. (2008). The role of chelators in preventing biofilm formation and catheter-related bloodstream infections. *Current Opinion in Infectious Diseases*, 21(4), 385–392.
- 149. Sherer, E., et al. (2008). Identification of agestructured models: Cell cycle phase transitions. *Biotechnology and Bioengineering*, 99(4), 960–974.
- 150. Cook, J. R., et al. (2003). T(14;18)(q32;q21) involving MALT1 and IGH genes in an extranodal diffuse large B-cell lymphoma. *Human Pathology*, 34(11), 1212–1215.
- 151. Gary, K. W., et al. (2009). Racial differences in employment outcome after traumatic brain injury at 1, 2, and 5 years postinjury. *Archives of Physical Medicine and Rehabilitation*, 90(10), 1699–1707.
- 152. Sherer, M., et al. (2003). Race and productivity outcome after traumatic brain injury: Influence of confounding factors. *The Journal of Head Trauma Rehabilitation*, 18(5), 408–424.
- 153. Arango-Lasprilla, J. C., et al. (2008). Racial differences in employment outcomes after traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 89(5), 988–995.
- 154. Sander, A. M., et al. (2009). Relationship of race/ ethnicity and income to community integration following traumatic brain injury: Investigation in a nonrehabilitation trauma sample. *NeuroRehabilitation*, 24(1), 15–27.
- 155. Arango-Lasprilla, J. C., et al. (2007). Traumatic brain injury and functional outcomes: Does minority status matter? *Brain Injury*, 21(7), 701–708.
- 156. Burnett, D. M., et al. (2003). Ethnographic analysis of traumatic brain injury patients in the national model systems database. *Archives of Physical Medicine and Rehabilitation*, 84(2), 263–267.
- 157. Rosenthal, M., et al. (1996). Impact of minority status on functional outcome and community integration following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 11, 40–57.

- Kennepohl, S., et al. (2004). African American acculturation and neuropsychological test performance following traumatic brain injury. *Journal of International Neuropsychological Society*, *10*(4), 566–577.
- 159. Department, C.H.R. (2001). Craig Hospital inventory of environmental factors (CHIEF) manual, v.3.0. Eaglewood, Co: Craig Hospital.
- 160. Whiteneck, G. G., Gerhart, K. A., & Cusick, C. P. (2004). Identifying environmental factors that influence the outcomes of people with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 19(3), 191–204.
- 161. Corrigan, J. D., & Bogner, J. A. (2008). Neighborhood characteristics and outcomes after traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 89(5), 912–921.
- 162. Johnstone, B., et al. (2003). Rural/urban differences in vocational outcomes for state vocational rehabilitation clients with TBI. *NeuroRehabilitation*, 18(3), 197–203.

- 163. Gontkovsky, S. T., et al. (2006). Effect of urbanicity of residence on TBI outcome at one year post-injury. *Brain Injury*, 20(7), 701–709.
- 164. Harradine, P. G., et al. (2004). Severe traumatic brain injury in New South Wales: Comparable outcomes for rural and urban residents. *The Medical Journal of Australia*, 181(3), 130–134.
- 165. Farmer, J. E., Clark, M. J., & Sherman, A. K. (2003). Rural versus urban social support seeking as a moderating variable in traumatic brain injury outcome. *The Journal of Head Trauma Rehabilitation*, 18(2), 116–127.
- 166. Levy, Y., et al. (2003). Intravenous immunoglobulins in peripheral neuropathy associated with vasculitis. Annals of the Rheumatic Diseases, 62(12), 1221–1223.
- 167. Sherer, M., et al. (2003). Use of the WCST and the WCST-64 in the assessment of traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 25(4), 512–520.

Part II

Assessment

# **Bedside Evaluations**

# Mark Sherer, Joseph T. Giacino, Matthew J. Doiron, Allison LaRussa, and Sabrina R. Taylor

#### Abstract

Comprehensive neuropsychological evaluation will often not be the best approach to early assessments of persons with TBI on the inpatient acute care or rehabilitation unit. A number of key issues for such patients can be addressed with brief bedside evaluations. These issues include level of consciousness, posttraumatic confusion, language functioning, overall cognitive functioning, and emotional distress. Brief assessments of these issues that can be completed at bedside and are amenable to repeated administration to track clinical course can make several contributions to clinical management. These contributions include feedback to family members regarding patient status, feedback to the patient to improve selfawareness and facilitate active participation in therapies, feedback to caregivers to inform approaches to treatment, documentation of the course of recovery and detection of unexpected worsening, assessment of effects of medication and other interventions, determination of decision making capacity, determination of safety judgment, and others.

#### Keywords

Evaluation • Bedside • Brief • Minimally conscious • Confusion • Aphasia • Cognition • Depression • Anxiety

M. Sherer, Ph.D., ABPP, FACRM (⊠) TIRR Memorial Hermann, Houston, TX, USA

University of Texas Medical School at Houston, Baylor College of Medicine, Houston, TX, USA e-mail: Mark.Sherer@memorialhermann.org

Department of Physical Medicine and Rehabilitation, Massachusetts General Hospital, Boston, MA, USA Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA, USA

MGH Institute for Healthcare Professionals, Boston, MA, USA

M.J. Doiron, B.A. • A. LaRussa • S.R. Taylor, Ph.D. Neuropsychology Laboratory, Spaulding Rehabilitation Hospital, Charlestown, MA, USA

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_3, © Springer Science+Business Media, LLC 2014

J.T. Giacino, Ph.D. Spaulding Rehabilitation Hospital, Charlestown, MA, USA

# Brief Evaluations for Persons with TBI

# Rationale for Brief Evaluations for Persons in Early Recovery from TBI

Neuropsychological assessment often involves assessment of a broad range of cognitive and emotional issues using multiple tests and questionnaires that require hours to administer. However, persons in early recovery from TBI are often unable to complete such extensive evaluations. In addition, the critical clinical questions for such patients (e.g., is he/she aware of his/her surroundings; can he/she consistently indicate yes or no; is he/she oriented; is he/she depressed, etc.) may be answerable with much briefer assessments. Brief evaluations that address important clinical questions can inform patient care without overly fatiguing the patient or unduly interfering with the patient's participation in rehabilitation therapies. These brief assessments can contribute to many of the same goals as comprehensive assessments that are typically completed later in the course of recovery. These goals include feedback to family members, feedback to improve patient self-awareness, feedback to caregivers including guidance for treatment, documentation of the course of recovery and detection of unexpected worsening, assessment of effectiveness of medication and other interventions, determination of decision making capacity, determination of safety judgment, and others [1].

# Issues Assessed with Bedside Evaluations

Bedside evaluations are best suited to assessment of high impact patient issues that have direct implications for patient management and/ or provide information that is important to the patient and caregivers. Such measures should be conducive to rapid and repeated administration at the bedside or in the therapy gym. While a large number of possible issues may be amenable to bedside evaluation, this chapter will focus on (1) disorders of consciousness (DOC), (2) posttraumatic confusion including constituent symptoms such as disorientation, restlessness (agitation), and impaired attentional functioning, (3) language abilities including yes/no responding, (4) cognitive abilities such as memory and reasoning that affect safely judgment and decision making capacity, and (5) emotional status.

The importance of these issues is largely selfevident. Accurate diagnosis of a patient's level of consciousness can literally have life or death consequences. Physicians and family members may be more likely to withhold heroic interventions from vegetative than from minimally conscious patients. Appropriate management for and interactions with locked in or akinetic patients differ from vegetative or minimally conscious patients. For responsive but confused patients, assessment of recovery of orientation and resolution of restlessness, cognitive impairment, and decreased level of arousal yields important information regarding the rate of recovery and likely outcome. Since restlessness is a key issue in early patient management in the rehabilitation setting, it is important to be able to detect the effects of interventions intended to reduce agitation.

While persistent aphasia is not common following blunt head trauma, it does occur and, of course, it can certainly be present in patients with penetrating injuries. Confused patients may show language forms generally associated with aphasia such as paraphasias that resolve as their confusion resolves. It is important for family members and clinicians alike to have an understanding of the patient's ability to process and express language. Structured assessments are crucial in this arena as even experienced clinicians may overestimate a patient's language comprehension ability. This is especially common for patients who respond appropriately to social cues and nonverbals in casual interactions. It is of particular importance to have a careful assessment of the patient's ability to give accurate yes/no responses as this is generally the initial manner in which the patient reports pain or expresses preferences such as the desire to go to the bathroom.

Early assessment of cognitive functioning should focus on ability to attend to nursing

and therapy interventions and ability to retain information such as safety instructions. These issues are crucial to ongoing patient management. More detailed assessment of cognitive abilities can often wait to just before discharge or shortly after discharge when this information can inform supervision needs at home, therapy goals for outpatient cognitive rehabilitation services, and eventual plans for community activities such as driving, return to work, etc.

New onset of depression and anxiety is common after TBI. Often these disorders are manifested during the post-acute period of recovery, but they may become evident during inpatient rehabilitation as well. Alternatively, some patients may have labile affect with episodes of crying that are thought by rehabilitation staff and family members to reflect depression even though the patient remains confused and does not have the ability to consistently recall information regarding his or her current situation. In either case, early assessment of emotional function can provide important information to the family and rehabilitation team as well as guide interventions for the patient.

# Characteristics of Useful Bedside Evaluations

There are a number of characteristics that are desirable in bedside evaluations. Ideally such assessments should be brief, taking no more than 30 min of direct assessment time with the patient. Longer assessments may be overly taxing for many persons in early recovery from TBI and the time involved in obtaining the assessment will bump up against other clinical care that the patient is receiving. We have often encountered patients who fatigued within 5 min of initiation of an assessment. In such cases, performance can rapidly deteriorate so that the assessment reveals only that which was already known, that the patient fatigues rapidly.

Assessments should address issues of key importance to patient management and ongoing treatment. As noted above, rehabilitation caregivers need to know if a patient can give reliable yes/no responses, can process and retain safety instructions, is imminently suicidal, etc. A detailed assessment of specific cognitive impairments such as executive functions can usually wait till a later point in recovery when this information may guide post-discharge planning.

Bedside assessments should be repeatable. Since a key goal of brief assessments is to document the patient's progress or detect an unexpected decline or leveling in recovery, these assessments may be repeated often. For many patients in early recovery who have not recovered the ability to consistently form and later recall new memories, repeating assessment of orientation or simple cognitive abilities is not problematic. These patients are unlikely to show practice effects due to their continued anterograde amnesia. However, note that even patients who have not regained orientation can be sensitized by repeated questioning. Some patients become aware that they are not oriented and may be irritated or embarrassed by repeated questioning. This difficulty can usually be resolved by taking some time to talk with the patient to express interest and build rapport before launching into yet another barrage of questions about what day it is.

Bedside assessments should require few or no testing materials. If materials are required they should be compact. It is highly desirable that any required materials can be reproduced by drawing or listing them on a sheet of paper. While patients can and should be scheduled for these brief evaluations, the clinician often encounters a patient that he/she has wanted to assess serendipitously while on the unit to see another patient. If assessment materials are not readily at hand, such opportunities may be missed resulting in lost information for the treatment team and lost time for the neuropsychologist.

#### Measures

#### Coma Recovery Scale-Revised [2]

(i) *Background and Purpose*. The original Coma Recovery Scale (CRS) was published in 1991 by Giacino and colleagues [3] to detect and monitor subtle but important neurobehavioral signs of conscious awareness in patients with DOC, specifically, coma, the vegetative state (VS), and the minimally conscious state (MCS). The original 25-point scale was comprised of six subscales designed to assess arousal, auditory, visual, motor, oromotor-verbal, and communication functions. Indications for use were aimed at improving diagnostic accuracy and outcome prediction, early identification of medical complications. facilitating treatment planning, and monitoring treatment effectiveness. The CRS was revised in 2004 and re-named, the Coma Recovery Scale-Revised (CRS-R) [2]. The revised version consists of 23 hierarchically arranged items organized into six subscales designed to assess audition, receptive and expressive language, visuoperception, communication ability, motor functions, and arousal level. Modifications to the original scale were prompted by new developments in the diagnostic criteria for DOC [4], the results of a Rasch analysis [5] completed in 2000, and feedback from clinicians and researchers. The psychometric integrity of the CRS-R has been extensively investigated and shown to be a valid, reliable, and clinically useful measurement tool for aiding differential diagnosis, monitoring recovery of consciousness, and evaluating response to treatment (see below for further discussion of the psychometric properties) [2, 6-8]. The scale is currently available in 13 languages, including English, Italian, French, German, Dutch, Spanish, Portuguese, Swedish, Norwegian, Danish, Greek, Chinese, and Korean. The DOC Task Force of the American Congress of Rehabilitation Medicine recommended the CRS-R with "minor reservations" for use in clinical practice and it was selected as the measure of choice for monitoring recovery of consciousness in TBI research by the Traumatic Brain Injury Common Data Elements (CDE) consortium, cosponsored by the National Institute of Neurological Disorders and Stroke (NINDS), Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, U.S. Department of Veterans Affairs and the National Institute on Disability and Rehabilitation Research [9].

(ii) Administration and Scoring. The CRS-R Administration and Scoring Manual provides specific instructions for administering the 23 items on the scale and offers operational definitions for scorable behavioral responses. The examination is comprised of a brief baseline observation period, assessment of brain stem reflexes, an Arousal Facilitation Protocol (AFP), and administration of the six CRS-R subscales. The baseline observation period is conducted to document the nature and frequency of behaviors that occur at "rest." These findings are used to help determine whether specific behaviors observed during assessment of command-following represent volitional responses or episodes of random movement. Brain stem reflexes are assessed for prognostic purposes and to assist with interpretation of responses to specific items on the CRS-R subscales. For example, on the Visual Subscale, the inability to elicit visual pursuit may be secondary to cranial nerve injury and unrelated to level of consciousness. The AFP is administered at the outset of the examination when patients appear sleepy or underaroused. Deep pressure is applied to specific muscle groups, particularly those linked to the vestibular system (e.g., sternocleidomastoid, trapezius), to increase alertness. CRS-R subscale items are administered in the order they appear on the Record Form. Each subscale is structured so that items representing the most complex behaviors are administered first and are assigned the highest score. The remaining subscale items reflect progressively decreasing levels of neurologic function and are assigned lower scores, accordingly. When an item is scored positively (i.e., the behavior observed meets the specified response criteria), the examiner proceeds to the next subscale, avoiding the need to administer lower-level items requiring more intrusive stimulation (e.g., application of noxious stimulation).

The first CRS-R subscale administered is the Auditory subscale. This 4-item subscale assesses language comprehension at the upper limit (i.e., consistent or reproducible command-following), and the ability to detect sound at the lower limit (i.e., localization, startle). The 5-item Visual subscale examines visuoperceptual functions, including the ability to recognize and localize objects in space, visual pursuit, sustained fixation, and response to visual threat. The 6-item Motor subscale tests instrumental praxis (i.e., functional object use), purposeful movement (i.e., automatic motor responses, object manipulation), and response to noxious stimulation (i.e., localization, flexion withdrawal, abnormal posturing). The 3-item Oromotor/Verbal subscale extracts evidence of intelligible speech, unintelligible vocalization (and other forms of active oral movement), and oral reflexive movement. The 2-item Communication subscale uses a series of standardized personal and situational orientation questions to elicit discernible verbal or gestural yes-no responses. Communicative responses are characterized as "functional" if they are consistently accurate and "nonfunctional" if they are recognizable but inaccurate. The 3-item Arousal subscale assesses level of alertness and ability to sustain attention. Attention is assessed by counting the number of times the patient fails to respond to a verbal prompt over the course of the examination (cutoff = 3). Response accuracy is not considered in scoring this item. Level of alertness is ascertained by monitoring for episodes of sustained eyelid closure that occur *during* the examination (i.e., sustained eye-opening with or without stimulation). The CRS-R also includes a supplementary item designed to capture behaviors that occur in response to a specific triggering stimulus (e.g., smiling, crying when presented with a family photo). This item is not scored but can be used to detect non-reflexive behaviors that occur in contingent relation to specific environmental events, one of the diagnostic criteria associated with the MCS. Subscale scores are summed to obtain a total score ranging between 0 and 23.

Subscale scores are recorded on the CRS-R Record Form along with the total score. The CRS-R total scores can also be entered on the CRS-R Progress Tracking Chart, which provides information concerning the trajectory and rate of recovery. CRS-R items that correspond to specific diagnostic criteria for MCS and emergence from MCS are denoted with an asterisk or a cross, respectively. The diagnostic impression, therefore, is based on the profile of scores attained on the six subscales. Figure 1 shows the trajectory of change in CRS-R total (Panel A) and subscale (Panel B) scores for a patient with disturbance in consciousness who was eventually found to have underlying aphasia. Administration time for the CRS-R ranges from 15 to 30 min, depending on the patient's level of consciousness. The CRS-R Administration and Scoring Manual and other relevant information can be found online at the following websites:

- National Institute on Neurologic Disorders and Stroke—TBI CDE: www.commondataelements.ninds.nih.gov
- Center for Outcome Measurement in Brain Injury: www.COMBI.org
- Spaulding-Harvard TBI Model System
  Program: www.SH-TBIMS.org
- A training DVD is available by request at the following website sponsored by the Coma Science Group: www.coma.ulg.ac.be

(iii) Recommendations for Use in Clinical Care. The CRS-R has been widely used in both clinical practice and research. In the clinical domain, common indications include differential diagnosis (e.g., Is the patient in a vegetative or MCS?), establishing prognosis (e.g., Is the rate of recovery above or below average for patients with DOC?), gauging functional outcome (e.g., Is the patient able to communicate reliably?), treatment planning (e.g., Are yes-no responses consistent enough to support use of an augmentative communication system?), and assessing response to treatment (e.g., Did the frequency/accuracy of commandfollowing improve following introduction of a neurostimulant?). In research applications, the CRS-R has served as a prognostic indicator in outcome prediction studies [2, 7], as an outcome measure in clinical trials [10, 11], and as a reference standard in diagnostic neuroimaging and electrophysiology validation studies [12–14].

The CRS-R is the only standardized neurobehavioral assessment instrument that directly incorporates all of the diagnostic criteria required to distinguish VS from MCS, and MCS from emergence from MCS [6]. There is evidence that this feature improves diagnostic sensitivity as the



Fig. 1 Trajectory of patient performance on the CRS-R across a 10-week course of inpatient rehabilitation. Panel A shows the change in total CRS-R scores over time. During weeks 1 and 2, scores consistently remained in the vegetative range. In week 3, the total score sharply increased and then slowly transitioned into the MCS range during weeks 4 through 6. Although variable, the total score progressively improved through week 8 and was near ceiling, signaling emergence from MCS. Panel B compares recovery curves across the six CRS-R subscales. In week 3, a marked disparity emerged between motor (triangles) and language (diamonds, x's and asterisks) performance. Despite the absence of any evidence of language comprehension or expression during weeks 4-6 (Auditory subscale scores <3; Oromotor-Verbal subscale scores <3; Communication subscale scores <2), there was

scale has been shown to outperform clinician consensus in detecting signs of MCS [7].

Serial CRS-R assessments may also alert the examiner to subclinical changes in medical status. For example, a sharp decrease in the total CRS-R score may signal the onset of occult illness. We have observed situations in which the total CRS-R score has precipitously declined by

clear and consistent evidence of complex automatic motor behavior (Motor subscale scores >4), including picking up objects and returning social gestures. This pattern of findings raised the possibility that an underlying aphasia may have been contributing to the absence of commandfollowing and expressive speech. Following week 7, reproducible command-following was noted (Auditory subscale=3), providing some evidence of language comprehension. During week 8, functional object use was demonstrated indicating emergence from MCS, however, command-following remained inconsistent, yes–no communication attempts remained unreliable and paraphasic verbalizations were noted. Taken together, the early dissociation between language and motor performance was likely related to an underlying aphasia

more than five points prior to detection of systemic infection. Conversely, we have had cases in which a chronically low CRS-R total score (i.e., <10) demonstrates a sharp increase after initiating antibiotics for suspected respiratory illness. The rate of recovery, as reflected by the change in the total CRS-R score over a 4-week period (e.g., week 4-week 1), can also help predict outcome and disposition needs (e.g., outpatient program, inpatient rehabilitation hospital, skilled nursing facility).

(iv) Key Research. Psychometric Properties: The psychometric properties of the CRS-R have been extensively studied by investigators in the U.S. and Europe. In their 2004 standardization study, Giacino and colleagues administered the CRS-R to 80 patients, all of whom were unable to follow commands or communicate reliably [2]. Inter-rater reliability was high for the total CRS-R score, high for the auditory, motor, oromotor/verbal, and communication subscale scores, and moderate for the visual subscale. Similarly, test-retest reliability was high for the total score, and for the auditory, motor, communication, and visual subscale scores. Tests of internal consistency showed a significant relationship between the total CRS-R score and individual subscale scores, demonstrating the measure's homogeneity. Subscale inter-correlations were moderate with the exception of a low inter-correlation between the visual and oromotor/verbal subscale. The CRS-R also showed adequate concurrent validity with the original version of the CRS and with the DRS. In a separate analysis of 20 subjects focusing on diagnostic agreement, inter-rater agreement for diagnosis was consistent in 16 of 20 cases assessed by two different examiners on the same day, and diagnosis remained stable across two examinations completed by the same examiner on consecutive days (i.e., test-retest reliability) in 18 out of 20 cases.

In 2010, an expert panel was convened by the American Congress of Rehabilitation Medicine to conduct a systematic review of the literature on the psychometric properties of behavioral rating scales designed for patients with DOC [6]. The review found the standardized administration, scoring and interpretive guidelines user-friendly and the psychometric properties (i.e., construct validity, internal consistency, inter-rater reliability, test-retest reliability) robust. The CRS-R was the only measure of the 13 scales examined to be recommended with "minor" reservations for use in patients with DOC.

Employing an Italian version of the CRS-R, Sacco and colleagues found good inter-rater and

test-retest reliability for both total and subscale scores when experienced raters administered the scale [15]. A Norwegian study completed by Løvstad and colleagues explored the influence of rater experience on the reliability and diagnostic validity of the CRS-R. Raters from six different hospitals with three levels of experience established a diagnosis after independently administering and scoring the CRS-R twice over 3 days. Results again showed adequate reliability and validity; however, more experienced raters were more accurate in distinguishing VS and MCS than less experienced raters [8]. Inter-rater reliability for CRS-R scores was greater when highly experienced raters were paired with moderately experienced raters than when highly experienced raters were paired with newly trained raters. Testretest reliability was greater among moderately experienced raters relative to newly trained raters, and there were no significant correlations in scores between the newly trained raters. Regarding diagnostic agreement, inter-rater and test-retest reliability were better among highly experienced raters than among less experienced raters (e.g., test-retest: 88 % for highly and moderately experienced raters vs. 50 % agreement between highly experienced and newly trained raters).

Schnakers et al. investigated the psychometric properties of a French translation of the CRS-R in a cohort of patients in VS and MCS [16]. Interrater and test-retest reliability were found to be satisfactory, and validity analyses showed significant correlations between total scores on the French CRS-R and scores on three other measures of level of consciousness-the Glasgow Coma Scale, Full Outline of UnResponsiveness, and Wessex Head Injury Matrix. The authors also found strong diagnostic agreement between raters. A fourth validation study conducted by Simões et al. using a Portuguese version of the CRS-R showed high inter-rater and test-retest reliability for both the total score and the six subscale scores [17].

A recently-published study by La Porta and colleagues in Italy applied Rasch analysis to further explore the measurement properties of the CRS-R [18]. Rasch analysis iteratively examines the underlying constructs assessed by rating scales and determines the "fit" between the individual items and the scale in its entirety. Scales with high degrees of "fit" are capable of interval measurement, allowing the examiner to gauge the distance between items using a measurement constant referred to as a "logit." Within and between-subject comparisons can also be performed. La Porta, et al. collected CRS-R data from 129 participants across five hospitals using an Italian translation of the scale. Twelve experienced raters administered the CRS-R twice per patient extracting a total of 258 scores. Rasch analysis demonstrated excellent internal construct validity and satisfied all the principles required for interval measurement. The ordering of the scoring categories for the six subscales remained stable across different settings and raters with variable levels of experience, and scores were invariant regardless of the length of time post-injury, setting, age, or sex of the patients. The authors concluded that the CRS-R provides a linear measure of ability and, thus, is appropriate for use at the level of the individual patient.

## **Clinical Applications**

#### **Differential Diagnosis**

Giacino et al. compared the CRS-R to the DRS in differentiating MCS from VS in a subsample of subjects included in their 2004 standardization study [2]. While the rate of diagnostic agreement between the CRS-R and the DRS was 87.5 %, the CRS-R detected evidence of conscious awareness in 10 cases (of 80) in which the DRS yielded a diagnosis of VS. Most of the cases missed by the DRS demonstrated evidence of visual pursuit on the CRS-R, a key diagnostic criterion not investigated by the DRS.

In 2009, Schnakers et al. compared diagnoses established by the CRS-R with those made based on clinical consensus of the rehabilitation team [7]. Data were collected from 103 patients with mixed injury etiologies who were followed by specialized neurorehabilitation teams. The investigators reported that 41 % of patients diagnosed with VS by team consensus (via qualitative observational assessment) had at least one sign of conscious awareness based on CRS-R examination. Similarly, 10 % of patients diagnosed with MCS by team consensus met criteria for emergence from MCS when examined with the CRS-R. Failure to detect purposeful eye movements accounted for most of the cases missed by simple observational assessment.

The CRS-R includes an unscored supplementary scale designed to detect behaviors that occur selectively in response to specific stimuli as this type of contingent relationship between a triggering stimulus and a specific response is associated with conscious awareness. Formisano et al. introduced the Post-Coma Scale (PCS) to incorporate emotional responsiveness into the assessment of patients with DOC [19]. The PCS seeks to supplement the assessment of features of MCS by incorporating a measure of emotional responsiveness. In this study, patients were diagnosed based on scores on the CRS-R and the PCS, both of which were administered by a single professional or by a professional in the presence of a caregiver. Results showed a significant positive correlation between PCS and CRS-R scores. Of note, patients at higher levels of consciousness demonstrated more emotional responsiveness. In addition, patients scored higher on both the CRS-R and PCS when caregivers were present during the exam, suggesting that the presence of caregivers may induce sufficient emotional stimulation to drive volitional behaviors.

#### **Outcome Assessment**

The CRS-R can be used to assess outcome either by monitoring "difference scores" (i.e., change in total score from time 1 to time 2) or by tracking changes in diagnosis (e.g., transition from VS to MCS or MCS to confusional state). Diagnosis has been shown to be of prognostic importance, as patients in MCS generally have more favorable outcomes than those in VS after controlling for chronicity. Katz and coworkers retrospectively examined the recovery trajectories of 36 patients diagnosed with VS or MCS on the CRS-R at admission to rehabilitation [20]. Patients were evaluated across the inpatient rehabilitation course and followed at least once between 1 and 4 years post-injury. Of the 11 patients admitted with a diagnosis of VS, 8 transitioned to MCS after spending an average of 8 weeks in VS. Approximately 70 % of patients admitted to rehabilitation in either VS or MCS emerged from MCS. However, when patients were segregated by admitting diagnosis (VS vs. MCS), almost twice as many patients in MCS emerged (80 %) relative to those in VS (45 %).

Noé et al. prospectively followed 32 patients with DOC on the CRS-R for at least 6 months or until emergence from MCS [21]. Approximately 25 % of the sample emerged from MCS within 6 months of injury. The rate of emergence was higher in the group admitted in MCS (35 % vs. 8 %). MCS subjects who did not emerge after the 6 month follow-up period (n=13) were reassessed an average of 15 months after injury. No significant changes in neurological status were observed in any of the subjects followed.

Estraneo and colleagues employed the CRS-R to prospectively monitor outcome in 50 patients who remained in VS for at least 6 months after severe acquired brain injury. Patients were followed for an average of 26 months after injury [22]. At the final follow-up, 24 % had transitioned to MCS and, of these, 20 % recovered consciousness after 12 months post-injury. Six of the 10 patients who recovered consciousness late did so between 18 and 26 months post-injury. Of note, four patients in non-traumatic VS (three with anoxia and one with hemorrhagic stroke) recovered signs of consciousness on the CRS-R well beyond the 3-month cutoff established for "permanent VS" by the Multi-Society Task Force on PVS [23].

#### Monitoring Treatment Effectiveness

The standardized administration format and quantitative approach to assessment championed

by the CRS-R suggest it is a suitable instrument for monitoring the effectiveness of treatment interventions applied in patients with DOC. The CRS-R has been used to monitor rate of recovery in patients diagnosed with VS and MCS who were exposed to amantadine hydrochloride vs. placebo [10] and to detect changes in alertness, motor function, and communication ability in a patient with chronic posttraumatic MCS following deep brain stimulation of the central thalamus [11].

The CRS-R has also been used in program evaluation. Seel et al. employed the CRS-R to investigate the effectiveness of a specialized early neurorehabilitation program for patients with DOC [24]. The investigators found that patients improved from admission to discharge on all six subscales of the CRS-R and 53 % had emerged from MCS by discharge.

# Use of the CRS-R as a Reference Standard

In view of the absence of a gold standard for detection of conscious awareness, investigators have employed the CRS-R as a "reference standard" to compare the results of neuroimaging and electrophysiologic, behavioral studies. Rodriguez-Moreno and colleagues developed an fMRI paradigm in which ten subjects diagnosed with VS, MCS, EMCS, or Locked-In Syndrome (LIS) were shown pictures of common objects and instructed to silently name each object presented [14]. The authors found that the degree of activation of the language network observed during the task correlated with the subjects' CRS-R score, and that activation patterns in subjects who attained high CRS-R scores (i.e., LIS, EMCS) approximated healthy controls while those with low CRS-R scores showed little to no activation.

Similarly, a series of recent studies have shown that CRS-R total scores correlate with metabolic activity in critical cortical networks [25–27]. Total CRS-R scores have been found to be higher in patients who retain activity in frontoparietal midline structures thought to mediate internal, stimulus-independent "self" consciousness [25, 28], while scores on the Auditory, Oromotor-Verbal and Communication subscales have been shown to be lower in patients with metabolic dysfunction in the dominant language hemisphere [26] and with white matter connectivity measured with diffusion tensor imaging [29].

Bekinschtein and others explored the relationship between level of consciousness and electromyographic (EMG) changes in response to movement commands presented to ten patients diagnosed with VS or MCS on the CRS-R [30]. Subjects received verbal commands to move their right or left hands or to remain still while EMG and video recordings monitored spontaneous muscle activity. Two patients (one MCS and one VS) who failed to produce any observable motor activity showed significantly more EMG activity following administration of the movement command than when instructed to simply rest. These findings suggest that bedside EMG monitoring may improve diagnostic accuracy in patients who evidence little to no active movement.

Electrophsiologic studies employing the CRS-R have shown that measures of EEG entropy in the left frontotemporal region correlate with CRS-R scores obtained in the acute but not chronic stage of recovery [12] and with resting state EEG measures of cortical connectivity [31].

Recent experimental studies have demonstrated an association between cortical excitability induced by transcranial magnetic stimulation (TMS) and level of consciousness gauged by the CRS-R. Lapitskaya and colleagues found a significant correlation between short-latency afferent inhibition values and CRS-R scores in 47 patients with DOC (24 VS and 23 MCS) and 14 healthy controls [32]. Similarly, Casali et al. found a graded relationship between CRS-R total scores and the duration and complexity of EEG activity induced by TMS pulses in patients diagnosed with coma, VS, MCS, emergence from MCS, and LIS [33]. Using the CRS-R as a benchmark, the investigators were able to distinguish all subjects who were alert from those who were completely unconscious based on the complexity of the EEG signal.

#### Confusion Assessment Protocol [34]

(i) Background and Purpose. TBI is synonymous with disordered consciousness. Persons with severe injuries have a period of complete loss of consciousness called coma that persists for hours after the injury. Persons who survive severe TBI generally recover to a state of, at least, partial consciousness, though a few remain in an unconscious, vegetative state. Persons with moderate injuries have briefer periods of unconsciousness, and have, by definition, recovered partial consciousness by the time of presentation to the emergency department. Persons with mild TBI often have no period of complete loss of consciousness, but rather have a period of altered consciousness that is characterized by inability to form and later recall new memories. For patients who have recovered the ability to respond to the environment in a relatively consistent, meaningful way, the next period of recovery is characterized by confusion [35]. Some refer to this confusional state as delirium and yet others use the term posttraumatic encephalopathy. We prefer the term posttraumatic confusional state (PTCS). While PTCS is a subtype of delirium, delirium can be caused by a wide range of medical conditions ranging from substance withdrawal to end-stage organ failure. The courses, prognoses, and likely underlying neuropathologies of these diverse conditions vary greatly making delirium an imprecise term [36]. Posttraumatic encephalopathy is a similarly imprecise term as encephalopathy refers to any disease or disruption of brain function.

Our research and clinical experience indicate that PTCS is likely ubiquitous in moderate and severe TBI. Persons with mild TBI may not show a full confusional state though many do. PTCS is characterized by disorientation, cognitive impairment, fluctuation in presentation, agitation, decreased daytime arousal, nighttime sleep disturbance, and psychotic-type symptoms [34]. Our research has shown that assessment of PTCS informs judgments about long-term prognosis and that PTCS is characterized by a particular pattern of recovery with decreased daytime arousal, nighttime sleep disturbance, and psychotic-type symptoms recovering earliest post-injury and fluctuation and cognitive impairment persisting longest post-injury [37].

PTCS has clear implications for early clinical management including rehabilitation. Confused patients pose increased risk of injury to self and others. Confusional states are associated with increased duration and cost of care [38]. Confused patients have poorer cooperation with therapy activities. Family members/significant others are distressed to see their loved ones confused and may over-interpret words and actions that flow from this confusion. The Confusion Assessment Protocol (CAP) [34] was developed to provide a standardized procedure for diagnosing persons as confused or non-confused and to facilitate serial tracking of patient recovery over time. The CAP was designed to be easy to administer, score, and interpret.

(ii) Administration and Scoring. The CAP is a collection of scales and items developed by a number of TBI researchers. These scales and items were selected for inclusion in the CAP based on their ability to distinguish patients who met diagnostic criteria for delirium for those who did not and based on coverage of the seven key symptoms of PTCS identified by the authors of the CAP. In developing the CAP, new scoring rules were created for some of the items, some items were modified, and a methodology was created for combining scores from items to determine whether a patient is confused or not.

Cognitive impairment and orientation are measured with performance measures. Cognitive impairment is measured by items taken from the Cognitive Test of Delirium [39] and the Toronto Test of Acute Recovery after TBI [35]. These items measure basic attentional and mental control functions. Patients are asked to count to 20 forwards and backwards and recite the months forwards and backwards. On a simple vigilance task, patients indicate when a target letter is spoken and inhibit any response to non-target letters. Patients also answer simple reasoning questions. Finally, patients are shown five pictures that they must identify from a field of ten pictures after a delay. In selecting these items, the authors found that more demanding tasks such as word list learning were failed by virtually all persons in early recovery from TBI so that such items did not assist in distinguishing confused patients from non-confused patients.

Orientation is assessed using the Galveston Orientation and Amnesia Test [40]. This test requires the patient to give personal information (name, date of birth, residence), current circumstances (current location, date of admission to the hospital, means of conveyance to the hospital), first memory after injury, last memory before injury, time, day, and date.

Agitation, fluctuation, nighttime sleep disturbance, decreased daytime arousal, and psychotic-type symptoms are rated using scales or items selected from scales. Ratings for each of these symptoms are based on observations over a 24 h period. The rater should base these scores on his/her own interactions with the patient as well as reports from nurses, therapists, family members, and others are appropriate. Agitation is measured with the Agitated Behavior Scale [41]. Fluctuation and psychotictype symptoms are measured using items from the Delirium Rating Scale-R-98 [42]. An item from the Delirium Rating Scale-R-98 was modified to measure nighttime sleep disturbance and a new item was created to assess decreased daytime arousal.

Once measures are administered and scales rated, performances are classified as consistent with confusion or not consistent with confusion based on scoring criteria developed by the authors of the CAP. Persons with four or more symptoms consistent with confusion are in PTCS and persons with three symptoms consistent with confusion are in PTCS if one of these symptoms is disorientation. All others are not in PTCS. Confused patients with three or four symptoms of confusion are in mild confusion, those with five symptoms are in moderate confusion, and those with six or seven symptoms are in severe confusion. CAP forms and other information can be downloaded at http://www.tbims.org/combi/ cap/index.html and additional information on the CAP can be obtained by contacting Mark Sherer at Mark.Sherer@memorialhermann.org.

(iii) Recommendations for Use in Clinical Care. On the inpatient neurorehabilitation unit, we recommend that all patients with TBI who are not in vegetative or MCSs at admission are assessed with the CAP on the day after admission. We find that some patients are so fatigued by the transfer process that findings from assessments on the day of admission may be misleading. The CAP should be implemented with initially vegetative or minimally conscious patients once they recover to a responsive state. Once begun, CAP assessments should continue on a two or three times a week basis until confusion resolves. Confusion is considered to have resolved when the patient obtains non-confused scores on two consecutive CAP assessments that are at least 24 h apart. Some may prefer to require three consecutive non-confused CAPs as we have seen some patients perform in the confused range on subsequent evaluations even after obtaining two consecutive non-confused scores. Of course, some patients may regress due to factors such as seizures, posttraumatic hydrocephalus, medication effects, sleep disturbance, etc. Patients who receive non-confused scores on the initial two CAPs are considered non-confused at admission and no other CAP examination need be performed. Absent medical complications or medication effects, patients are expected to show a recovering course for symptoms of confusion. We have found that roughly 75 % percent of patients show a decreased number of symptoms of confusion from the first to the third CAP assessment covering a period of 4-5 days. Failure to improve

problems that are interfering with recovery. CAP results can be reported in the medical records and patient staffings by reporting confusion status (confused, not confused), number of symptoms of confusion present, GOAT score, ABS score, cognitive impairment score, etc. Graphic presentation of results over time is helpful to the rehabilitation team, physicians, and family/significant others. Note that some patients who have just emerged from MCS at the initiation of CAP assessment may show increasing numbers of symptoms of confusion as they become more responsive and exhibit more behavior overall. However, most patients will show a

should prompt an assessment for possible treatable

recovering course with decreasing numbers of symptoms and even those who begin assessment early after emergence from MCS will eventually show a recovering course. Posttraumatic confusion is a transitional phase of recovery and patients will not remain confused indefinitely. A few patients may remain so amnestic that they never recover orientation, but other symptoms of confusion such as sleep disturbance, psychotic symptoms, decreased daytime arousal, agitation, and fluctuation will continue to resolve so that even the disoriented patient with severe or profound cognitive impairment will usually eventually emerge from confusion based on CAP diagnostic criteria. As with patients with dementia, patients with TBI who have persistent, severe cognitive impairment are at increased risk of becoming confused when stressed by medical conditions, medication effects, pain, sleep disturbance, or other factors.

(iv) Key Research. The CAP has been validated by comparing CAP classifications of patients as confused vs. non-confused to clinical diagnosis of delirium, showing that CAP findings are predictive of functional status outcomes both early after injury and at 1 year follow-up, demonstrating the associations of CAP scores with an important rehabilitation issue-cooperation with treatment, and illustrating patterns of recovery of symptoms of confusion. These studies provide strong support for the CAP as a research and clinical instrument. However, to this point, all studies published on the CAP have been conducted by the research group that originally developed the CAP. Sometimes assessment instruments do not perform comparably when used by groups other than the original group that developed the instrument. Clinicians and researchers will have more confidence in the CAP if findings shown by Sherer and colleagues are cross-validated by research conducted by other groups.

Sherer and colleagues [34] described the development of the CAP and presented early findings. The researchers selected CAP items and developed scoring rules based on the ability of these items to distinguish TBI patients who were clinically diagnosed as being in delirium using DSM IV criteria from those who were not in delirium. CAP development was based on findings from 62 persons with TBI who were admitted to a neurorehabilitation unit. In the same paper, the authors provided some initial validation data for the CAP in a new series of 112 patients with TBI who were studied during inpatient brain injury rehabilitation. Combining the CAP development sample with initial validation sample yields a group of 174 persons with TBI. For this combined group, CAP diagnosis of confusion was 93 % sensitive and 86 % specific for a clinical diagnosis of delirium indicating very good agreement. Sherer and colleagues have also provided data on the phenomenology of PTCS based on their seven key symptoms of confusion. As shown in Fig. 2, fluctuation in presentation was the most common symptom occurring in all confused patients with cognitive impairment occurring in about 95 % and disorientation occurring in over 90 % of confused patients. Psychotic-type symptoms were the least frequent symptom occurring in about 45 % of confused patients. There was overlap in symptoms between confused and non-confused patients for all symptoms other than disorientation which did not occur in non-confused patients. Decreased dayarousal, restlessness (agitation), time and psychotic-type symptoms were all rare in nonconfused patients with each occurring in no more than 10 % of cases. Fluctuation and cognitive impairment were relatively common in nonconfused patients who were in early recovery from TBI occurring in about 45 % and 35 % of patients, respectively. Multivariable linear regression analysis based on 80 cases showed that even after adjustment for other predictors including initial injury severity (days to follow commands), confusion status at rehabilitation admission predicted functional status at rehabilitation discharge with patients who were in PTCS at admission having poorer functional status.

Sherer and colleagues [43] extended earlier findings by showing that CAP data were not only predictive of functional status at rehabilitation discharge, but also of productivity outcome at 1 year post-injury. Reporting on a series of 168 persons with TBI admitted for inpatient rehabilitation, Sherer and colleagues showed that number of symptoms of confusion (possible range 0–7) shown on a single CAP evaluation at about 21 days post-injury was predictive of functional status at discharge from inpatient rehabilitation and of productivity status at 1 year post-injury. After adjustment for other predictors including injury severity (initial Glasgow Coma Scale score and days to follow commands), patients with only two symptoms of confusion at 21 days postinjury were about three times as likely to have favorable functional status at discharge as compared to those with five symptoms of confusion. Further, patients with two symptoms at 21 days post-injury were twice as likely to be productive (competitively employed or in school) at late follow-up 1 year post-injury as compared to those with five symptoms. An exploratory analysis of the significance of each individual symptom of confusion showed that absence of each symptom of confusion was associated with more favorable overall outcome than presence of each symptom. In these analyses, simple odds ratios were calculated and there was no adjustment for other predictors. A surprising finding was that patients who did not show psychotic symptoms at 21 days post-injury were over 14 times more likely to be productive at 1 year follow-up than patients who did show psychotic-type symptoms. This finding was obtained even though no patients in this series showed onset of a new psychotic disorder meaning that psychotic symptoms in all patients resolved when confusion resolved.

Sherer and colleagues [37] provided additional data on patterns of symptom recovery in confused patients. In a series of 107 confused patients assessed during inpatient neurorehabilitation, patients were generally shown to have rapid recovery of symptoms. From the first CAP assessment to the second CAP assessment (about 2 days), over 50 % of patients showed a decrease in the number of symptoms of confusion and from the second CAP to the third CAP (again about 2 days) over 50 % of patients with initial moderate or severe confusion again showed a decrease in the number of symptoms. Only about 25 % of patients with initial mild confusion showed a decrease in number of symptoms due to a floor effect. Data showed that nighttime sleep


Fig. 2 Phenomenology of confusion in patients with traumatic brain injury. Disorient indicates Disorientation, Cog imp—Cognitive impairment, Fluct—Fluctuation, Sleep dist—Nighttime sleep disturbance, Arouse—Decreased daytime arousal, Psychotic—Psychotic-type

symptoms. Adapted with permission from Sherer M, Yablon SA, Nakase-Richardson R, Nick T. Effect of severity of posttraumatic confusion and its constituent symptoms on outcome after traumatic brain injury. Arch Phys Med Rehabil 2008;89:42–47

disturbance, decreased daytime arousal, and psychotic-type symptoms are the earliest symptoms of confusion to resolve while fluctuation and cognitive impairment are the most persistent symptoms of confusion.

Silva and colleagues [44] examined the relationship between posttraumatic confusion as measured by the CAP with cooperation with treatment for persons with TBI undergoing inpatient rehabilitation. A series of 74 patients with TBI undergoing inpatient rehabilitation were assessed three times weekly with the CAP, while therapists treating the patients rated the degree of cooperation with therapy. CAP scores and therapist ratings were obtained independently. A regression model including only the number of CAP symptoms accounted for 25 % of the variability in cooperation ratings, while the full model including age at injury, years of education completed, time since injury, and injury severity (Glasgow Coma Scale score) accounted for 33 % of the variability in cooperation. Greater confusion was associated with poorer cooperation. To examine the associations of specific symptoms of confusion with cooperation ratings, Spearman's coefficient was calculated. Decreased daytime arousal showed the strongest association with a Spearman's coefficient of -0.42. Correlations for restlessness (agitation), psychotic symptoms, and cognitive impairment were -0.39, -0.39, and -0.24, respectively. In each case, presence of the symptom was associated with poorer cooperation.

Finally, Sherer and colleagues [45] followed up on earlier findings regarding the significance of psychotic symptoms in confused patients. In a series of 107 patients with TBI in inpatient rehabilitation who completed a total of 640 CAP assessments, Sherer and colleagues examined factors that were associated with the occurrence of psychotic-type symptoms. The strongest association was found for nighttime sleep disturbance. Patients with nighttime sleep disturbance were over four times as likely to show psychotic-type symptoms as those without sleep disturbance. Patients were more likely to show psychotic-type symptoms early after rehabilitation admission rather than later after rehabilitation admission though time since injury did not predict psychotic symptoms. Patients with a greater degree of cognitive impairment were more likely to show psychotic symptoms than those with more intact cognitive functioning. Sherer and colleagues also replicated their earlier finding showing that presence of psychotic-type symptoms was associated with a decreased likelihood of a productive outcome at 1 year post-injury. However, in this analysis with adjustment for age, years of education, and injury severity (time to follow commands), the effect was not as great with those with no psychotic symptoms being about four times as likely to be productive as compared to 14 times more likely in the earlier unadjusted analysis.

# Mississippi Aphasia Screening Test [46]

(i) Background and Purpose. Aphasia is a primary disturbance of the ability to use language. Aphasia can affect all aspects of language including expressive language, fluency of speech, repetition, naming, and language comprehension in both auditory and written modalities. As with other neurocognitive impairments, structured testing is essential in evaluating language functions as even experienced clinicians may under-estimate the degree of impairment if they rely on conversation or other non-structured interactions. For aphasics with preserved speech prosody and superficial social greetings, even very substantial language comprehension deficits may be missed. Language impairments are strongly associated with focal left hemisphere brain lesions, but can also be seen in patients with diffuse lesions such as those that often occur in persons with TBI. Absent a focal lesion in the language cortex, language impairment in persons with TBI tends to resolve as early confusion resolves and persistent aphasia after nonpenetrating TBI is rare. Nonetheless, assessment of patients in early recovery after TBI should include assessment of language. It is critical to

determine whether a patient can consistently and accurately respond yes and no to simple questions such as, "Are you in pain?" Yes/no responding is often the medium for patients to express preferences such as food choices in early recovery. Similarly, it is crucial to have an assessment of the patient's ability to comprehend spoken and written language. Some patients may adequately comprehend short passages of language, but breakdown in their comprehension of longer passages. While this phenomenon may indicate impairment of attentional functions or memory rather than a primary language disorder, this information is still important to clinicians providing care to the patient and family members interacting with the patient.

Comprehensive aphasia batteries may require up to 2 h to administer. Such lengthy assessments may not be well tolerated by confused patients or other patients in early recovery from TBI. Since language impairment is unlikely to be the key deficit in persons with TBI due to blunt head trauma, detailed assessment with a comprehensive battery may not be warranted. A brief battery that assesses a broad range of language functions and can be repeated to document improvements in functioning is adequate for many patients. The Mississippi Aphasia Screening Test (MAST) is a brief, easily repeated battery that assesses key aspects of language function and has been used with persons with TBI.

(ii) Administration and Scoring. The MAST consists of nine subtests that assess (1) naming, (2) automatic speech, (3) repetition, (4) yes/no accuracy, (5) object recognition, (6) verbal command following, (7) written command following, (8) verbal fluency, and (9) writing to dictation. Each subtest generates a score and subtest scores range from 0 to 10 except for yes/no accuracy which ranges from 0 to 20. Subtest scores contribute to index scores for receptive and expressive language that each range from 0 to 50 and an overall score that can range from 0 to 100. As with many aphasia tests, the MAST is a test of impairment so that persons with no language impairment are expected to obtain perfect or near perfect scores. The entire test can be administered in 5–10 min.

For the naming subtest, the patient is asked to name five common objects (e.g., pen, hand, watch). For the automatic speech subtest, the patient is asked to count to ten, say the days of the week, and complete three sentences (e.g., Three strikes and you're \_\_\_\_). On the repetition subtest, the patient repeats words and phrases (e.g., carrot, under the old wooden bridge). Verbal fluency is assessed by showing the patient a standard stimulus picture and asking the patient to describe what he/she sees. The number of items verbalized by the patient determines the score. If the patient can provide 11 or more verbalizations, he/ she will receive the highest possible score of 10. For the naming, automatic speech, repetition, and writing to dictation subtests, each correct response is worth two points. The naming, automatic speech, repetition, verbal fluency, and writing to dictation scores are summed to generate the expressive language index score.

For the yes/no accuracy subtest, patients respond yes or no to ten questions ranging from "Is your name [patient's correct]?" to "Does summer come after spring?" For the object recognition subtest, the subject is asked to point to a specific object from a field of five common objects. For verbal command following, the patient is asked to follow five verbal instructions and for written command following, the patient is asked to follow the same five instructions that are provided typed on sheets of paper. Each correct response for yes/no accuracy, object recognition, verbal command following, and written command following is worth two points. Yes/no accuracy, object recognition, verbal command following, and written command following scores are summed to calculate the receptive language index score and the expressive and receptive language index scores are totaled to yield the total score.

As noted above, with the possible exception of the verbal fluency subtest, neurotypical adults are expected to achieve essentially perfect scores. Any score less than perfect should be investigated. It is important to consider factors other than language impairment that can affect performance on the MAST. Patients in early recovery may have decreased daytime arousal and fluctuating attention. These factors can cause item failures even in those with intact language functions. Some patients may give poor effort because they believe the items are too simple and they are offended by being asked to complete such simple tasks. Other patients may refuse to complete the MAST altogether. Such non-compliance might reflect a general rejection of assessments, a rejection of assessments of cognitive abilities, or transitory fatigue or poor mood. Sometimes, refusals can be caused by some degree of self-awareness of deficits that motivates the patient to reject assessments so that he/she will not be confronted with deficits due to TBI.

It should be noted that non-patients reported on by Nakase-Thompson and colleagues [46] produced an average score substantially below the maximum score on the verbal fluency subtest, suggesting that scoring rules for the verbal fluency subtest may be too stringent. MAST users should be cautious in interpreting scores from this subtest.

(iii) Recommendations for Use in Clinical Care. While the MAST is primarily intended for patients who are at more substantial risk for having aphasia (i.e., persons with stroke), given the brevity of the test and the importance of detecting possible language impairments, it is reasonable to administer the MAST to all responsive patients with TBI. Patients should be assessed two or three times weekly until any possible language impairments resolve. Once a patient obtains a perfect or near perfect score on a given subtest on two consecutive administrations, this subtest can be omitted in subsequent administrations to save time. Patients who show substantial impairment on multiple subtests and who do not meet criteria for PTCS should be evaluated with a more comprehensive aphasia battery such as the Western Aphasia Battery. Patients who are in the confusional state often show signs of aphasia, perhaps even paraphasias, that resolve once the confusion resolves. Confused patients will often not tolerate assessment with a comprehensive aphasia battery. Serial assessment with the MAST will generally be sufficient to document language impairment and track any improvement until the confused state resolves.

For patients in inpatient rehabilitation, findings from MAST assessments should be discussed widely with the rehabilitation team as deficits associated with aphasia can have substantial impact on rehabilitation therapies. Patients with comprehension deficits may be perceived as uncooperative. Patients with impairment of expressive language, particularly those with nonfluent speech, are likely to be quite frustrated and this can cause withdrawal from therapy tasks. If patients do not have consistent, accurate yes/no responding, pain assessments may be invalid and patients may have difficulty obtaining assistance with primary needs (e.g., toileting). It is common that different rehabilitation professionals (e.g., speech language pathologist, physician, nurse, etc.) who have assessed the patient will have very different views of the degree of language impairment. This discrepancy is contributed to by fluctuation in the degree of language impairment, different approaches to the assessment, and differing degrees of sophistication among the clinicians. The psychologist should work to achieve consensus regarding the degree and type of aphasia as well as approaches to maximize the patient's ability to communicate.

Similarly, it is important for family/close others to be educated regarding any language impairments experienced by the patient. Family/close others will benefit from specific instructions on how to facilitate communication with persons with language impairment.

(iv) *Key Research*. Most research on the MAST has been conducted with patients with stroke. As compared to other brief aphasia screens, the MAST has broader coverage of language functions though there has been some concern that performance on some subtests (e.g., verbal fluency) could be affected by visual deficits such as neglect [47]. The MAST has been used broadly with patients with stroke and has been translated into Spanish [48], Czech [49], Italian, German, and French [50].

Initial data comparing neurotypical community volunteers to persons with right or left hemisphere stroke were provided by Nakase-Thompson and colleagues [46]. As expected, persons with left hemisphere stroke scored more poorly than neurotypicals on all subtests, index scores, and the total score. Persons with left hemisphere stroke scored more poorly than those with right hemisphere stroke on all subtests other than object recognition and verbal fluency and scored more poorly on both index scores and the total score. It is noted that raw score differences for object recognition and verbal fluency were in the expected direction. While some symptoms of acute confusion may mimic language impairment, the MAST has been used to assess aphasia even in patients with stroke who were in delirium [51].

While primarily used with patients with stroke, the MAST has been recommended for use in patients with TBI [52]. In a particularly helpful paper, Nakase-Richardson and colleagues [53] provide detailed information on the relative difficulty of the MAST yes/no items for patients in early recovery from TBI who are confused (in delirium) and not confused. As show in Table 1, questions about one's name are rarely answered incorrectly by confused patients and never answered incorrectly by non-confused patients. Similarly, yes/no accuracy for state of residence is good for confused patients and excellent for nonconfused patients. However, other simple, but more semantically complex questions were surprisingly difficult for both confused and nonconfused patients. Only 50 % of confused patients correctly answered, "Do you put your shoe on before your sock?" and, even more unexpectedly, only 74 % of non-confused patient correctly answered "Does summer come after spring?" It seems likely that inaccurate answers from either group are more due to attentional or arousal issues rather than to language processing impairment. Knowledge of these relative items difficulties facilitates a more sophisticated assessment of yes/no accuracy in patients in early recovery from TBI.

#### Cognitive Assessment

(i) Cognitive impairment early after TBI is universal for persons with moderate or severe TBI [54]. In contrast, as a group, those with mild TBI as indicated by posttraumatic amnesia less

	Item diff	iculty	
	(number of correct/number of overall responses)		
	All subjects	Confused	Non- confused
	N=144	N=105	N=39
1. Is your name Johnson? (incorrect)	.84	.78	1.0
2. Is your name (correct name)?	.82	.75	1.0
3. Do you live in Rhode Island? (incorrect)	.80	.74	.95
4. Do you live in Mississippi? (correct)	.82	.76	.97
5. Do you wear a glove on your foot?	.72	.67	.95
6. Am I touching my eye?	.76	.69	.95
7. Does Monday come before Tuesday?	.67	.63	.80
8. Does summer come after spring?	.62	.58	.74
9. Is a chicken bigger than a spider?	.72	.65	.90
10. Do you put your shoe on before your sock?	.60	.50	.85

Table 1 MAST yes and no accuracy for 144 persons with  $\ensuremath{\mathsf{TBI}}^1$ 

<sup>1</sup>Modified with permission from Nakase-Richardson R, Yablon SA, Sherer M, Nick TG, Evans CE. Emergence from minimally conscious state: Insights from evaluation of posttraumatic confusion. Neurology 2009;73:1120–1126

than 24 h or normal CT scan or time to follow commands of less than 1 h score equivalently to those with no TBI by 1 month post-injury [55]. Fortunately, cognitive impairment after TBI has a recovering course. By 1 year post-injury, significant improvement is noted for most patients with moderate and severe TBI though a subgroup of patients with severe TBI shows no progress (this might be partially due to a floor effect for some tests) [56]. Tasks requiring rapid cognitive or motor responding and those requiring memory may be particularly likely to show residual impairment at later assessments, while tasks requiring verbal knowledge and reasoning are likely to show a greater degree of recovery [56]. Contrary to the general pattern of improvement after TBI, a subset of patients may show decline over time [57]. Age at time of injury has been identified as a risk factor for cognitive decline after TBI with those with older age at injury being at greater risk for decline.

Degree of cognitive impairment is substantially determined by initial injury severity as indicated by Glasgow Coma Scale score, the length of time post-injury before the patient regains the ability to follow commands, and/or the duration of posttraumatic amnesia [54, 56]. As expected, persons with more severe injuries show greater cognitive impairment. Accordingly, the degree of cognitive impairment after TBI can be seen as a proxy variable for injury severity and improvement in cognitive functioning as an index of the degree of overall recovery after TBI. However, factors such as age at time of injury [58] and years of education completed [59] may also affect cognitive functioning after TBI. Greater age at injury is associated with greater cognitive impairment, while greater number of years of education is associated with more intact cognitive functioning.

Early assessment of cognitive abilities after TBI serves multiple purposes [1]. First, such assessments can guide early interventions and management. Patients with profound memory impairment cannot be expected to recall safety instructions or carryover training from one rehabilitation session to another, so detection of such impairment has implications for the amount of supervision needed on the nursing unit and the approach to therapy provided in the gym. Documentation of improvements in cognitive functioning over time provides encouragement that the patient has additional potential to improve. This information is reassuring to family members/close others and may prompt payers for rehabilitation services to extend the patient's access to inpatient and/or outpatient rehabilitation services. In contrast, stable cognitive impairment or a decline in cognitive functioning in a patient in early recovery may indicate the need for additional workup to determine if medication effects, undetected seizures, posttraumatic hydrocephalus, or some other factor is interfering with the expected course of recovery.

Finally, repeated cognitive assessment can be used to help determine whether the patient is showing a favorable response to medication interventions such as methylphenidate or amantadine. For postdischarge planning considerations such as return to driving or work, comprehensive assessments such as those described in chapter "Comprehensive Assessment" are more appropriate.

(ii) Assessment Batteries for Early Cognitive Testing. For early cognitive assessment, a fine grained comparison of the patient's performance to normative expectations may not be required. The psychologist may just wish to obtain a general idea of areas of greatest concern or to compare the patient's performance to another assessment completed a few days earlier. For these brief, bedside assessments, some psychologists develop their own procedures and rely on internal norms developed over years of professional practice. As an example, a psychologist could repeatedly assess a patient by presenting a wordlist learning task, some mental arithmetic questions, and some social reasoning questions among other needed tests. A wordlist can be increased in length as the patient improves and the arithmetic and reasoning questions can be made more complex. Such a battery would allow the psychologist to report, in a general way, whether or not the patient is improving and to provide feedback to therapists, physicians, and family members/close others. Examples of such feedback might be, "Last week the patient only recalled one of five words after a 3 min delay, but this week he was able to recall four of seven words after a delay." or "A couple of weeks ago the patient had great difficulty with simple arithmetic like 3+4 incorrectly answering three of five problems, but now she is able to do multiplication problems like 6×13 correctly answering four of five problems."

There are several options if one wishes to administer a battery of standardized tests. Pastorek and colleagues [60] demonstrated the feasibility of a brief battery for testing patients who were still in posttraumatic amnesia. This battery included the Complex Ideation Material subtest from the Boston Diagnostic Aphasia Examination [61], a shortened version of the Token Test [62], and Auditory and Visual Number Search Tests developed by Levin and colleagues [63]. This battery was administered to a sample of 105 patients with complicated mild, moderate, or severe TBI at about 1 month post-injury. The average Galveston Orientation and Amnesia Test score for the sample was 64.1 indicating that many patients were still in posttraumatic amnesia. Completion rates for the four tests ranged from 83 to 89 %, if ill patients (e.g., fever, nausea) are excluded, indicating that this battery is feasible for use in patients in early recovery from TBI.

Kalmar and colleagues [64] examined the feasibility of a brief neuropsychological battery in a sample of 354 patients with complicated mild, moderate, or severe TBI who were tested 2-6 weeks post-injury as part of the TBI Model Systems (TBIMS) program. Tests included in this battery were the Galveston Orientation and Amnesia Test, California Verbal Learning Test-II, Trail-making Test, Symbol Digit Modalities Test, Grooved Pegboard Test, FAS verbal fluency, Wechsler Test of Adult Reading, and Wisconsin Card Sorting Test-64 card version. Of the 354 patients, 218 (62 %) were able to complete all tests. An additional 94 (26 %) were able to at least attempt tasks so that it could be determined that they were substantially impaired. For persons, who attempted but could not complete tasks, the most impaired possible score was assigned. In this manner, it was possible to obtain scores that reflected the degree of impairment on 312 (88 %) of 354 patients. The remaining 42 (12 %) were not able to attempt some tests due to medical illness, time constraint, or some other factor. Feasibility of this battery is demonstrated by the ability of examiners to obtain meaningful scores on 88 % of persons assessed early after moderate and severe TBI.

Finally, the NINDS CDE initiative [9, 65] (http://www.commondataelements.ninds.nih. gov/tbi.aspx#tab=Data\_Standards) recommends a brief core cognitive battery for hospitalized patients. This battery consists of the Wechsler Adult Intelligence Scale-IV (WAIS-IV) Processing Speed Index, the Trail Making Test, and either the Rey Auditory Verbal Learning Test or the California Verbal Learning Test II. The WAIS-IV Speed Index is derived from the Symbol Search and Coding subtests. This battery is essentially a subset of the battery proposed by Kalmar and colleagues [64] as it includes a list learning task, the Trail Making Test, and measures of processing speed. In a preliminary evaluation of the feasibility of this battery conducted as part of the Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) study [66], 368 persons with TBI were assessed at 6 months post-injury. While completion rates for each test were not reported, given the similarity to the TBIMS battery and the greater time postinjury, one would expect that virtually all participants were able to complete the battery.

(iii) Recommendations for Clinical Use. A number of factors such as practice setting, professional time available, psychologist preference, physician preference, time to anticipated discharge, goal of the assessment, and patient clinical presentation will affect the type and frequency of brief bedside evaluations for inpatients with TBI. In our practice, the most common goal of such brief assessments is to document cognitive recovery or to detect failure to recover. For this purpose, brief assessments done 2-3 times a week will provide ample data to provide feedback to the treating physician, rehabilitation team, patient, and family. If there are more specific goals for the assessment such as determining capacity to give consent, need for supervision, ability to carryover safety instructions, etc., additional tests should likely be added to the brief battery and the patient should be observed in therapy or other functional activities to aid with the assessment.

The usefulness of brief cognitive evaluations depends as much or more on the skill of the clinician in giving feedback as on the tests actually administered. Feedback to the person with TBI can be seen as a therapeutic activity to improve self-awareness while maintaining a sense of hope and facilitating cooperation with treatment. Feedback to family members/significant others can play a key role in facilitating preparations for discharge and in enhancing the degree of effective support they provide to the person with injury. Feedback to the physician and clinical team provides guidance for treatment and an objective way to chart progress.

(iv) *Key Research*. Pastorek and colleagues [60] assessed 105 patients with complicated mild, moderate, and severe TBI at 1 month post-injury using the Complex Ideation Material test, modified Token Test, Auditory Number Search Test, and the Visual Number Search Test. In unadjusted univariable regression models, results of these early assessments were predictive of patient functional status at 6 months post-injury as measured with the Disability Rating Scale. In multivariable models using age, years of education, best Glasgow Coma Scale score on the day of injury, and pupil responses immediately after injury as covariates, only the Complex Ideational Material test made a significant improvement to prediction of functional status above that made by the covariates. Interestingly, simply knowing whether or not a participant was able to complete a cognitive test was more predictive of functional status than the actual test score. For each of the four tests, a dichotomized (yes/no) completion variable made a unique contribution to prediction of functional status even after adjustment for all four covariates. These results indicate that early testability is predictive of more favorable outcome at a later time post-injury.

Hanks and colleagues [67] administered the battery recommended by Kalmar and colleagues [64] (Galveston Orientation and Amnesia Test, California Verbal Learning Test-II, Trail Making Test, Symbol Digits Modality Test, Grooved Pegboard Test, FAS verbal fluency test, Animal Naming Test, and the Wechsler Test of Adult Reading) to 174 persons with moderate and severe TBI. Note that persons who did not complete all tests were excluded from this analysis; however, 23 % of the retained sample was still in posttraumatic amnesia at the time of cognitive testing. In their regression analyses, Hanks and colleagues entered three covariates in a first block and the cognitive test scores in a second block. Covariates included injury severity as indicated by time to follow commands and functional status at rehabilitation admission as indicated by Disability Rating Scale score and FIM score. Outcomes were functional status at 1 year postinjury as indicated by the Disability Rating Scale and the FIM as well as ability to live independently at 1 year post-injury as indicated by Supervision Rating Scale. In each case, the block of neuropsychological variables made a unique contribution to prediction of the outcome. Given intercorrelations among cognitive variables, it is difficult to determine which tests are most predictive, but some evidence indicated that the Trail Making Test Part B and the Wechsler Test of Adult Reading were particularly useful.

Taken together, results of the Pastorek and colleagues and Hanks and colleagues studies provide good evidence of the usefulness of relatively brief, early cognitive assessments for predicting outcome after TBI even when many participants were tested while still in posttraumatic amnesia. Of course, as noted above, prediction of later outcome is just one possible use of early cognitive assessments.

### **Emotional Status**

(i) Psychiatric disturbances are very common for persons recovering from TBI with an overall incidence rate of about 61 %. For most of these individuals, pre-TBI psychiatric disturbance is a contributing factor, but a significant number of persons have a new onset of psychiatric symptoms after TBI [68]. By far the most common psychiatric disturbances after TBI are mood disorder (generally major depression or depressive disorder not otherwise specified) and various anxiety disorders (most commonly anxiety disorder not otherwise specified) which are seen in 30-40 % of patients [68, 69]. The incidence of depression and anxiety after TBI is generally reported for the first year post-injury, but these problems can emerge early after injury. Therapists and physicians caring for patients with TBI frequently report that they are concerned about depression and anxiety that cause distress for the patient and are thought to interfere with cooperation with therapy. Similarly, family members/ close others may report that persons with TBI are depressed and/or anxious. In our experience, these early reports of depression and anxiety are more likely to come from care providers and family than from the patients themselves. Indeed, some patients seem indifferent to their circumstances early after injury due to confusion and/or impaired self-awareness. Others may be reluctant to report emotional distress due to their pre-injury personality or to avoid being perceived as "weak" as they struggle with the recovery process. As part of their empathy for the patient, staff and family may project their own emotional responses to the injury onto the patient.

Emotional distress must be distinguished from affective lability in early recovery from TBI. Patients with TBI may have crying spells that suggest depression to caregivers and family in the absence of a report of other clinical symptoms of depression by the patient. Indeed, some patients may be so confused that one would question whether they can meaningfully report on their own emotional state. It is challenging to understand just what it means for a disoriented patient with severe memory impairment to be depressed or anxious when they have no later recall of periods when others perceived them to be anxious or depressed. Affective lability is common in confused patients. Even experienced clinicians may have difficulty distinguishing true mood disorder from affective lability [70].

Some rehabilitation staff may have difficulty experiencing empathy for emotional responses to catastrophic injury so that they are intolerant of complains of anxiety or depression from patients. We have encountered therapists and nurses who become irritated with patients who express sadness or anxiety in the aftermath of severe injuries that likely would have long-term consequences. Other staff may feel unprepared to deal with patients' emotional responses to their injuries. They may turn to the psychologist for help or, perhaps, expect the psychologist to "fix" the patient so that he/she will be better able to participate in therapy or require less frequent care on the nursing unit.

Assessment of emotional functioning in persons with TBI requires a thorough interview of the patient, if possible, along with a careful history from a reliable informant. It is frequently helpful to observe the patient in therapies or on the nursing unit as the patient may behave quite differently during challenging therapy or self-care activities than during a calm interview with the psychologist. One can lose a good deal of professional credibility by reporting that a patient's mood is improving only to have the physical therapist follow with a report that the patient cries incessantly throughout all therapy sessions. Co-treatment of patients with therapists provides an opportunity to observe the patient during therapy activities and, potentially, to provide an intervention in that context. In addition, co-treatment sessions provide an opportunity to educate rehabilitation therapists regarding emotional responses, affective lability, the grieving process, and other psychological reactions to injury.

Brief rating scales that can be administered quickly and repeated easily are helpful to the ongoing assessment of these patients. Two scales that have been used in the TBIMS program are the Patient History Questionnaire-9 (PHQ-9) [71] and the Generalized Anxiety Disorder-7 (GAD-7) [72]. These scales incorporate diagnostic criteria from the DSM-IV and, thus, facilitate the process of developing a clinical diagnosis.

(ii) Scales for Assessing Depression and Anxiety. While there are a number of scales that could be used to assess depression in early recovery from TBI, the PHQ-9 is recommended because of its brevity, inclusion of DSM-IV diagnostic criteria, use with a wide range of clinical populations, previous validation for use in patients with TBI, and inclusion as a supplemental measure in the NINDS CDE recommendations. In responding to the PHQ-9, the patient rates the frequency of nine symptoms over the previous 2 weeks and then rates the extent to which symptoms have interfered with work, personal independence, or relationships with others. Symptoms rated are anhedonia, sad affect, sleep disturbance, fatigue, appetite, self-esteem, concentration, psychomotor disturbance, and suicidal thoughts. The rating scale for each item ranges from 0 to 3 with higher scores indicating more frequent symptoms. A total score is obtained by adding ratings for the nine symptoms. Scores of 1–4 indicate minimal depression, 5–9 indicates mild depression, 10–14 indicates moderate depression, 15–19 indicates moderately severe depression, and 20–27 indicates severe depression.

As with depression there are a number of scales that could be used to rate anxiety. The GAD-7 and the PHQ-9 were developed together as part of the Patient Health Questionnaire [73]. The Patient Health Questionnaire is used to screen for mental disorders in primary care settings. As for the PHQ-9, the GAD-7 is brief, has been used with a wide range of clinical populations, and has been used with persons with TBI, notably by the TBIMS program. If the GAD-7 and PHQ-9 are given together, use of the same rating scale and timeframe facilitates valid responding by the patient. Patients in early recovery from TBI may have difficulty recalling and staying consistent with rating scale anchors. Symptoms rated on the GAD-7 include pervasive anxiety, incessant worrying, numerous worries, difficulty relaxing, restlessness, irritability, and fearfulness. A total score is obtained by adding ratings for the seven items. Scores of 0-4 indicate no or minimal anxiety, 5-9 mild anxiety, 10-14 moderate anxiety, and 15-21 severe anxiety.

(iii) Recommendations for Clinical Use. As with cognitive assessments, a wide range of factors should be considered in screening and monitoring emotional distress. It is reasonable to screen all non-confused patients for anxiety and depression though one might consider withholding screening until there is some self-report of depression or anxiety or some behavioral evidence such as tearfulness, withdrawal, expression of sad mood, expression of suicidal ideation, etc. While screening all patients may reveal anxiety or depression in patients for whom there was no other reason to suspect these concerns, there is also the possibility that screening results will produce false positives due to patients misinterpreting items, losing set on the rating scale, or some other cause. Once a patient is screened as being depressed, one will generally need to continue to follow this issue throughout the hospitalization and make an appropriate referral at discharge.

Once identified as anxious or depressed or at risk for anxiety or depression through interview or observation; screening with the PHQ-9, GAD-7, or some other scale; family report including pre-injury history; or rehabilitation therapist, nurse, physician, or other report; patients should be seen regularly. Depending on various clinical considerations, it is reasonable to see such patients two times weekly or more to provide intervention and to rescreen to monitor possible improvement or decline. These follow-up encounters can frequently be as brief as 15 min though they may take longer. As noted above, it is extremely helpful to see patients conjointly with rehabilitation staff who are concerned about the patients' emotional distress. While patient information that will not affect clinical management by others should be kept confidential, once emotional distress is identified as a concern that is affecting overall clinical care, it is important to provide ongoing feedback to the rest of the clinical team. One goal of this feedback is to facilitate a consistent response by the team to the patient's expressions of emotional distress. It may be quite counterproductive for the physical therapist or other rehabilitation care provider to spend treatment sessions "counseling" the patient in a manner that may directly contradict interventions provided by the psychologist.

The patient's family/close others may be key in addressing patient emotional distress. It is important to have a clear assessment of patient capacity so that permission is sought from patients with capacity before discussing these issues with family/close others. For patients who lack capacity, the surrogate decision maker can give permission for the psychologist to discuss patient emotional status with key family members. If affective lability is an issue, it can be quite reassuring for family members to understand that tears do not necessarily mean painful depression. For patients with true emotional distress, family may benefit from education regarding the likelihood of improvement over time and the desirability of providing consistent support that is not unrealistically positive and encourages participation in ongoing therapies.

(iv) Key Research. The PHQ-9 has been used in a number of studies of depression in persons with TBI [74, 75]. In a study of 135 persons with mild, moderate, and severe TBI, Fann and colleagues [76] demonstrated that the PHQ-9 has excellent sensitivity and specificity for a clinical diagnosis of depression made using the gold standard for assessment of the depression, the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (SCID). Sensitivity was 0.93 and specificity was 0.89. Further, contrary to concerns expressed by some, Cook and colleagues [77] found that somatic (e.g., sleep disturbance, change in appetite) and cognitive (e.g., trouble concentrating) items of the PHQ-9 were valid indicators of depression in persons with TBI even though these symptoms overlap with symptoms directly caused by TBI in some patients.

The GAD-7 has been validated as a measure of generalized anxiety in primary care and psychiatric settings [78–80]. The GAD-7 has also been used in studies of persons with a variety of neurologic disorders [81–83]. However, to this point, there has only been limited use of the GAD-7 in persons with TBI. Harch and colleagues [84] found the GAD-7, and the PHQ-9, to be sensitive to the beneficial effects of an intervention for post-concussive symptoms.

Acknowledgement Preparation of this chapter was partially supported by U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020, H133A120085.

### References

- Sherer, M., & Novack, T. (2003). Neuropsychological assessment for monitoring recovery after traumatic brain injury and making treatment recommendations in adults. In G. P. Prigatano, M. Cullum, & N. Pliskin (Eds.), *Demonstrating the utility and cost effectiveness in clinical neuropsychology* (pp. 39–60). New York: Psychology Press.
- Giacino, J. T., Kalmar, K., & Whyte, J. (2004). The JFK Coma Recovery Scale-Revised: Measurement characteristics and diagnostic utility. *Archives of Physical Medicine and Rehabilitation*, 85, 2020–2029.

- Giacino, J. T., Kezmarsky, M. A., DeLuca, J., & Cicerone, K. D. (1991). Monitoring rate of recovery to predict outcome in minimally responsive patients. *Archives of Physical Medicine and Rehabilitation*, 72, 897–901.
- Kalmar, K., & Giacino, J. T. (2005). The JFK Coma Recovery Scale-Revised. *Neuropsychological Rehabilitation*, 15, 454–460.
- Kalmar, K., & Giacino, J. T. (2000). The JFK Coma Recovery Scale: An ordinal or interval measure? (abstract). Archives of Physical Medicine and Rehabilitation, 81, 1619.
- Seel, R. T., Sherer, M., Whyte, J., Katz, D. I., Giacino, J. T., Rosenbaum, A. M., et al. (2010). Assessment scales for disorders of consciousness: Evidence-based recommendations for clinical practice and research. *Archives of Physical Medicine and Rehabilitation*, *91*, 1795–1813.
- Schnakers, C., Vanhaudenhuyse, A., Giacino, J., Ventura, M., Boly, M., Majerus, S., et al. (2009). Diagnostic accuracy of the vegetative and minimally conscious state: Clinical consensus versus standardized neurobehavioral assessment. *BMC Neurology*, 9, 35.
- Løvstad, M., Frøslie, K. F., Giacino, J. T., Skandsen, T., Anke, A., & Schanke, A. K. (2010). Reliability and diagnostic characteristics of the JFK Coma Recovery Scale-Revised: Exploring the influence of rater's level of experience. *The Journal of Head Trauma Rehabilitation*, 25, 349–356.
- Wilde, E. A., Whiteneck, G. G., Bogner, J., Bushnik, T., Cifu, D. X., Dikmen, S., et al. (2010). Recommendations for the use of common outcome measures in traumatic brain injury research. *Archives of Physical Medicine* and Rehabilitation, 91, 1650–1660.
- Giacino, J. T., Whyte, J., Bagiella, E., Kalmar, K., Childs, N., Khademi, A., et al. (2012). Placebocontrolled trial of amantadine for severe traumatic brain injury. *New England Journal of Medicine*, 366, 819–826.
- Schiff, N. D., Giacino, J. T., Kalmar, K., Victor, J. D., Baker, K., Gerber, M., et al. (2007). Behavioural improvements with thalamic stimulation after severe traumatic brain injury. *Nature*, 448, 600–603.
- Gosseries, O., Schnakers, C., Ledoux, D., Vanhaudenhuyse, A., Bruno, M. A., Demertzi, A., et al. (2011). Automated EEG entropy measurements in coma, vegetative state/unresponsive wakefulness syndrome and minimally conscious state. *Functional Neurology*, 26, 25–30.
- Fernández-Espejo, D., Bekinschtein, T., Monti, M. M., Pickard, J. D., Junque, C., Coleman, M. R., et al. (2011). Diffusion weighted imaging distinguishes the vegetative state from the minimally conscious state. *NeuroImage*, 54, 103–112.
- Rodriguez Moreno, D., Schiff, N. D., Giacino, J., Kalmar, K., & Hirsch, J. (2010). A network approach to assessing cognition in disorders of consciousness. *Neurology*, 75, 1871–1878.
- Sacco, S., Altobelli, E., Pistarini, C., Cerone, D., Cazzulani, B., & Carolei, A. (2011). Validation of the

Italian version of the Coma Recovery Scale-Revised (CRS-R). *Brain Injury*, 25, 488–495.

- Schnakers, C., Majerus, S., Giacino, J., Vanhaudenhuyse, A., Bruno, M. A., Boly, M., et al. (2008). A French validation study of the Coma Recovery Scale-Revised (CRS-R). *Brain Injury*, 22, 786–792.
- Simões, J. F., Jesus, L. M., Voegeli, D., Sá-Couto, P., Fernandes, J., & Morgado, M. (2011). Assessment of comatose patients: A Portuguese instrument based on the Coma Recovery Scale-revised and using nursing standard terminology. *Journal of Advanced Nursing*, 67, 1129–1141.
- La Porta, F., Caselli, S., Ianes, A. B., Cameli, O., Lino, M., Piperno, R., et al. (2013). Can we scientifically and reliably measure the level of consciousness in vegetative and minimally conscious States? Rasch analysis of the coma recovery scale-revised. *Archives of Physical Medicine and Rehabilitation*, 94, 527–535.
- Formisano, R., D'Ippolito, M., Risetti, M., Riccio, A., Caravasso, C. F., Catani, S., et al. (2011). Vegetative state, minimally conscious state, akinetic mutism and Parkinsonism as a continuum of recovery from disorders of consciousness: An exploratory and preliminary study. *Functional Neurology*, 26, 15–24.
- Katz, D. I., Polyak, M., Coughlan, D., Nichols, M., & Roche, A. (2009). Natural history of recovery from brain injury after prolonged disorders of consciousness: Outcome of patients admitted to inpatient rehabilitation with 1-4 year follow-up. *Progress in Brain Research*, 177, 73–88.
- 21. Noé, E., Olaya, J., Navarro, M. D., Noguera, P., Colomer, C., García-Panach, J., et al. (2012). Behavioral recovery in disorders of consciousness: A prospective study with the Spanish version of the Coma Recovery Scale-Revised. *Archives of Physical Medicine and Rehabilitation*, 93, 428–433.
- Estraneo, A., Moretta, P., Loreto, V., Lanzillo, B., Santoro, L., & Trojano, L. (2010). Late recovery after traumatic, anoxic, or hemorrhagic long-lasting vegetative state. *Neurology*, *75*, 239–245.
- 23. Ashwal, S., Cranford, R., Bernat, J. L., Celesia, G., Coulter, D., Eisenberg, H., et al. (1994). Medical aspects of the persistent vegetative state (1). The Multi-Society Task Force on PVS. *New England Journal of Medicine*, 330, 1499–1508.
- 24. Seel, R. T., Douglas, J., Dennison, A. C., Heaner, S., Farris, K., & Rogers, C. (2013). Specialized early treatment for persons with disorders of consciousness: Program components and outcomes. *Archives of Physical Medicine and Rehabilitation*, 94, 1908–1923.
- Vanhaudenhuyse, A., Demertzi, A., Schabus, M., Noirhomme, Q., Bredart, S., Boly, M., et al. (2011). Two distinct neuronal networks mediate the awareness of environment and of self. *Journal of Cognitive Neuroscience*, 23, 570–578.
- Bruno, M. A., Majerus, S., Boly, M., Vanhaudenhuyse, A., Schnakers, C., Gosseries, O., et al. (2012). Functional neuroanatomy underlying the clinical sub-

categorization of minimally conscious state patients. *Journal of Neurology*, 259, 1087–1098.

- Bruno, M. A., Fernandez-Espejo, D., Lehembre, R., Tshibanda, L., Vanhaudenhuyse, A., Gosseries, O., et al. (2011). Multimodal neuroimaging in patients with disorders of consciousness showing "functional hemispherectomy". *Progress in Brain Research*, 193, 323–333.
- Crone, J. S., Ladurner, G., Höller, Y., Golaszewski, S., Trinka, E., & Kronbichler, M. (2011). Deactivation of the default mode network as a marker of impaired consciousness: An fMRI study. *PLoS One*, *6*, e26373.
- 29. Newcombe, V. F., Williams, G. B., Scoffings, D., Cross, J., Carpenter, T. A., Pickard, J. D., et al. (2010). Aetiological differences in neuroanatomy of the vegetative state: Insights from diffusion tensor imaging and functional implications. *Journal of Neurology, Neurosurgery, and Psychiatry,* 81, 552–561.
- Bekinschtein, T. A., Coleman, M. R., Niklison, J., Pickard, J. D., & Manes, F. F. (2008). Can electromyography objectively detect voluntary movement in disorders of consciousness? *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 79, 826–828.
- 31. Lehembre, R., Marie-Aurélie, B., Vanhaudenhuyse, A., Chatelle, C., Cologan, V., Leclercq, Y., et al. (2012). Resting-state EEG study of comatose patients: A connectivity and frequency analysis to find differences between vegetative and minimally conscious states. *Functional Neurology*, 27, 41–47.
- 32. Lapitskaya, N., Gosseries, O., De Pasqua, V., Pedersen, A. R., Nielsen, J. F., de Noordhout, A. M., et al. (2013). Abnormal corticospinal excitability in patients with disorders of consciousness. *Brain Stimulation*, 6, 590–597.
- 33. Casali, A. G., Gosseries, O., Rosanova, M., Boly, M., Sarasso, S., Casali, K. R., et al. (2013). A theoretically based index of consciousness independent of sensory processing and behavior. *Science Translational Medicine*, 5, 198ra05.
- Sherer, M., Nakase-Thompson, R., Yablon, S. A., & Gontkovsky, S. T. (2005). Multidimensional assessment of acute confusion after TBI. Archives of Physical Medicine and Rehabilitation, 86, 896–904.
- 35. Stuss, D. T., Binns, M. A., Carruth, F. G., Levine, B., Brandys, C. E., Moulton, R. J., et al. (1999). The acute period of recovery from traumatic brain injury: Posttraumatic amnesia or posttraumatic confusional state? *Journal of Neurosurgery*, 90, 635–643.
- 36. Gupta, N., de Jonghe, J., Schieveld, J., Leonard, M., & Meagher, D. (2008). Delirium phenomenology: What can we learn from the symptoms of delirium. *Journal of Psychosomatic Research*, 65, 215–222.
- Sherer, M., Yablon, S. A., & Nakase-Richardson, R. (2009). Patterns of recovery of post-traumatic confusional state in neurorehabilitation admissions after TBI. Archives of Physical Medicine and Rehabilitation, 90, 1749–1754.
- Francis, J., Martin, D., & Kapoor, W. N. (1990). A prospective study of delirium in hospitalized elderly. *JAMA*, 263, 1097–1101.

- Hart, R. P., Levenson, J. L., Sessler, C. N., Best, A. M., Schwartz, S. M., & Rutherford, L. E. (1996). Validation of a cognitive test for delirium in medical ICU patients. *Psychosomatics*, *37*, 533–546.
- 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, 675–684.
- Corrigan, J. D. (1989). Development of a scale for assessment of agitation following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 11, 261–277.
- 42. Trzepacz, P. T., Mittal, D., Torres, R., Kanary, K., Norton, J., & Jimerson, N. (2001). Validation of the Delirium Rating Scale-revised-98: Comparison with the delirium rating scale and the cognitive test for delirium. *Journal of Neuropsychiatry and Clinical Neurosciences*, 13, 229–242.
- 43. Sherer, M., Yablon, S. A., Nakase-Richardson, R., & Nick, T. (2008). Effect of severity of posttraumatic confusion and its constituent symptoms on outcome after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89, 42–47.
- 44. Silva, M. A., Nakase-Richardson, R., Sherer, M., Barnett, S. D., Evans, C. C., & Yablon, S. A. (2012). Posttraumatic confusion (PTC) predicts patient cooperation during TBI rehabilitation. *American Journal of Physical Medicine and Rehabilitation*, *91*, 890–893.
- 45. Sherer, M., Yablon, S. A., & Nick, T. G. (2014). Psychotic symptoms as manifestations of the Posttraumatic Confusional State: Prevalence, risk factors, and association with outcome. *The Journal of Head Trauma Rehabilitation*, 29(2), E11–E18.
- Nakase-Thompson, R., Manning, E., Sherer, M., Yablon, S. A., Gontkovsky, S. T., & Vickery, C. (2005). Brief assessment of severe language impairments: Initial validation of the Mississippi Aphasia Screening Test. *Brain Injury*, *19*, 685–691.
- Salter, K., Jutai, J., Foley, N., Hellings, C., & Teasdell, R. (2006). Identification of aphasia post stroke: A review of screening assessment tools. *Brain Injury*, 20, 559–568.
- Romero, M., Sanchez, A., Marin, C., Navarro, M. D., Ferri, J., & Noe, E. (2012). Clinical usefulness of the Spanish version of the Mississisppi Aphasia Screening Test (MASTsp): Validation in stroke patients. *Neurología*, 27, 216–224.
- Kostalova, M., Bednarik, J., Mitasova, A., Dusek, L., Michalcakova, R., Kerkovsky, M., et al. (2012). Towards a predictive model for post-stroke delirium. *Brain Injury*, 26, 962–971.
- 50. Tschirren, M., Laganaro, M., Michel, P., Martory, M.-D., Di Pietro, M., Abutalebi, J., et al. (2011). Language and syntactic impairment following stroke in late bilingual aphasics. *Brain and Language*, 119, 238–242.
- 51. Mitasova, A., Kostalova, M., Bednarik, J., Michalcakova, R., Kasparek, T., Balabanova, P., et al.

(2012). Poststroke delirium incidence and outcomes: Validation of the Confusion Assessment Method for the Intensive Care Unit (CAM-ICU). *Critical Care Medicine*, 40, 484–490.

- 52. Giacino, J., Fins, J. J., Machado, A., & Schiff, N. D. (2012). Central thalamic deep brain stimulation to promote recovery from chronic posttraumatic minimally conscious state: Challenges and opportunities. *Neuromodulation*, 15, 339–349.
- Nakase-Richardson, R., Yablon, S. A., Sherer, M., Nick, T. G., & Evans, C. C. (2009). Emergence from minimally conscious state: Insights from evaluation of posttraumatic confusion. *Neurology*, 73, 1120–1126.
- Dikmen, S., McLean, A., Temkin, N. R., & Wyler, A. R. (1986). Neuropsychological outcome at one-month postinjury. Archives of Physical Medicine and Rehabilitation, 67, 507–513.
- Dikmen, S., Machamer, J., & Temkin, N. (2001). Mild head injury: Facts and artifacts. *Journal of Clinical and Experimental Neuropsychology*, 23, 729–738.
- Dikmen, S. S., Machamer, J. E., Winn, R., & Temkin, N. R. (1995). Neuropsychological outcome at 1-year post head injury. *Neuropsychology*, *9*, 80–90.
- 57. Millis, S. R., Rosenthal, M., Novack, T. A., Sherer, M., Nick, T. G., Kreutzer, J. S., et al. (2001). Long-term neuropsychological outcome after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 16, 343–355.
- Senathi-Raja, D., Ponsford, J., & Schonberger, M. (2010). Impact of age on long-term cognitive function after traumatic brain injury. *Neuropsychology*, 24, 336–344.
- Sumowski, J. F., Chiaravalloti, N., Krch, D., Paxton, J., & Deluca, J. (2013). Education attenuates the negative impact of traumatic brain injury on cognitive status. Archives of Physical Medicine and Rehabilitation, 94(12), 2562–2564.
- Pastorek, N. J., Hannay, H. J., & Contant, C. S. (2004). Prediction of global outcome with acute neuropsychological testing following closed-head injury. *Journal of the International Neuropsychological Society*, 10, 807–817.
- Goodglass, H., & Kaplan, E. (1983). Assessment of aphasia and other related disorders (2nd ed.). Philadelphia: Lea and Febiger.
- De Renzi, E., & Vignolo, L. A. (1962). The Token Test: A sensitive test to detect receptive disturbances in aphasics. *Brain*, 85, 665–678.
- Levin, H. S., Hannay, H. J., Martin, A., & Kreutzer, J. (1988). Acute battery tests for the Army Penetrating Head Injury Project; Unpublished manuscript.
- 64. Kalmar, K., Novack, T. A., Nakase-Richardson, R., Sherer, M., Frol, A. B., Gordon, W. A., et al. (2008). Feasibility of a brief neuropsychologic test battery during acute inpatient rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89, 942–949.

- 65. Hicks, R., Giacino, J., Harrison-Felix, C. L., Manley, G. T., Valadka, A., & Wilde, E. A. (2013). Progress in developing common data elements for traumatic brain injury research: Version 2—The end of the beginning. *Journal of Neurotrauma*, 30(22), 1852–1861.
- 66. Yue, J. K., Vassar, M. J., Lingsma, H., Cooper, S. R., Yuh, E. L., Mukherjee, P., et al. (2013). Transforming research and clinical knowledge in traumatic brain injury (TRACK-TBI) pilot: Multicenter implementation of the common data elements for traumatic brain injury. *Journal of Neurotrauma*, 30(22), 1831–1844.
- 67. Hanks, R. A., Millis, S. R., Ricker, J. H., Giacino, J. T., Nakase-Richardson, R., Frol, A. B., et al. (2008). The predictive validity of a brief inpatient neuropsychologic battery for persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89, 950–957.
- 68. Gould, K. R., Ponsford, J. L., Johnston, L., & Schonberger, M. (2011). The nature, frequency and course of psychiatric disorders in the first year after traumatic brain injury: A prospective study. *Psychological Medicine*, 41, 2099–2109.
- 69. Bhalerao, S. U., Geurtjens, C., Thomas, G. R., Kitamura, C. R., Zhou, C., & Marlborough, M. (2013). Understanding the neuropsychiatric consequences associated with significant traumatic brain injury. *Brain Injury*, 27, 767–774.
- Arciniegas, D. B., & Topkoff, J. (2000). The neuropsychiatry of pathologic affect: An approach to evaluation and treatment. *Seminars in Clinical Neuropsychiatry*, 5, 290–306.
- Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16, 606–613.
- Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Lowe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. Archives of Internal Medicine, 166, 1092–1097.
- Spitzer, R. L., Kroenke, K., Williams, J. B. W., & The Patient Health Questionnaire Primary Care Study Group. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary care study. *JAMA*, 282, 1737–1744.
- 74. Hart, T., Hoffman, J. M., Pretz, C., Kennedy, R., Clark, A. N., & Brenner, L. A. (2012). A longitudinal study of major and minor depression following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 93, 1343–1349.
- Failla, M. D., Burkhardt, J. N., Miller, M., Scanlon, J. M., Conley, Y. P., Ferrell, R. E., et al. (2013). Variants of SLC6A4 in depression risk following severe TBI. *Brain Injury*, 27, 696–706.
- 76. Fann, J. R., Bombardier, C. H., Dikmen, S., Esselman, P., Warms, C. A., Pelzer, E., et al. (2005). Validity of the Patient Health Questionnaire-9 in assessing depression following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20, 501–511.

- 77. Cook, K. F., Bombardier, C. H., Bamer, A. M., Choi, S. W., Kroenke, K., & Fann, J. R. (2011). Do somatic and cognitive symptoms of traumatic brain injury confound depression screening? *Archives of Physical Medicine and Rehabilitation*, 92, 818–823.
- Lowe, B., Spitzer, R. L., Williams, J. B., Mussell, M., Schellberg, D., & Kroenke, K. (2008). Depression, anxiety, and somatization in primary care: Syndrome overlap and functional impairment. *General Hospital Psychiatry*, 30, 191–199.
- 79. Ruiz, M. A., Zamorano, E., Garcia-Campayo, J., Pardo, A., Freire, O., & Rejas, J. (2011). Validity of the GAD-7 scale as an outcome measure of disability in patients with generalized anxiety disorders in primary care. *Journal of Affective Disorders*, 128, 277–286.
- Kertz, S., Bigda-Peyton, J., & Bjorgvinsson, T. (2013). Validity of the generalized anxiety disorder-7 scale in an acute psychiatric sample. *Clinical Psychology & Psychotherapy*, 20, 456–464.

- Bo, M., Massaia, M., Merlo, C., Sona, A., Canade, A., & Fonte, G. (2009). White-coat effect among older patients with suspected cognitive impairment: Prevalence and clinical implications. *International Journal of Geriatric Psychiatry*, 24, 509–517.
- Robbins, M. S., Bronheim, R., Lipton, R. B., Grosberg, B. M., Vollbracht, S., Sheftell, F. D., et al. (2012). Depression and anxiety in episodic and chronic cluster headache: A pilot study. *Headache*, *52*, 600–611.
- 83. Rakesh, P. S., Ramesh, R., Rachel, P., Chanda, R., Satish, N., & Mohan, V. R. (2012). Quality of life among people with epilepsy: A cross-sectional study from rural southern India. *National Medical Journal of India*, 25, 261–264.
- 84. Harch, P. G., Andrews, S. R., Fogarty, E. F., Amen, D., Pezzullo, J. C., Lucarini, J., et al. (2012). A phase 1 study of low-pressure hyperbaric oxygen therapy for blast-induced post-concussion syndrome and posttraumatic stress disorder. *Journal of Neurotrauma*, 29, 168–185.

# **Comprehensive Assessment**

Thomas F. Bergquist, Maya Yutsis, and Jackie L. Micklewright

#### Abstract

This chapter reviews comprehensive assessment in persons with brain injury in both acute and post-acute settings. A model of assessment is described and the various components of the assessment in the context of this model are reviewed with goal of conducting an evaluation which comprehensively describes functioning and leads to effective rehabilitation planning. The importance of the neuropsychologist conducting assessments as part of a larger rehabilitation team is also reviewed. Finally, a case study is provided which demonstrates the use of this assessment model.

### Keywords

Assessment • ICF model • Acute • Post-acute

# Introduction

Research continues to demonstrate that even with advances in brain imaging and biomarkers, neuropsychological assessment remains the most robust means of identifying the presence and progression of acquired brain dysfunction [1]. Moreover, unlike other neurodiagnostic techniques, neuropsychological testing is unique in the ability to not simply diagnose, but to also quantify the impact of brain injury on cognitive, behavioral, and adaptive functioning [2]. A recent study found over 1,600 peer-reviewed studies have been published on the relationship of psychological functioning and TBI, making it one of the populations most often encountered and studied by neuropsychologists [3]. Similarly a recent

T.F. Bergquist, Ph.D., ABPP-Cn (⊠) Mayo Clinic College of Medicine, Rochester, MN, 55905, USA e-mail: Bergquist.thomas@mayo.edu

M. Yutsis, Ph.D. Polytrauma Transitional Rehabilitation Program, VA Palo Alto Health Care System, Palo Alto, CA, USA

J.L. Micklewright, Ph.D. Hennepin County Medical Center, Minneapolis, MN, USA

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_4, © Springer Science+Business Media, LLC 2014 survey of practicing clinicians ranked TBI as the first or second most common diagnosis referred for neuropsychological evaluation [4]. This chapter will focus on the role of neuropsychological assessment in both inpatient and outpatient settings by (1) describing its goals and utility, (2) outlining a model of assessment that can be used to better understand the relationship between brain injury and long-term functioning, and (3) highlighting unique considerations for the assessment and treatment of individuals with traumatic brain injury.

# Assessment in Acute Settings: (Hospital and Inpatient Rehabilitation Setting)

Individuals with traumatic brain injury present to the acute inpatient setting with a wide variety of clinical presentations and heterogeneous neurobehavioral sequelae [5]. Depending on the type and severity of the sustained injury, assessments of cognitive functioning can be complicated by confusion, posttraumatic amnesia (PTA), and significant emotional and behavioral changes. Acute inpatient assessments are conducted with an appreciation for injury severity and natural recovery trajectories with the goal of (1) describing changes in mental status over time and (2) documenting the nature and level of cognitive and functional impairment resulting from the injury.

# Assessing Consciousness, Orientation, and Posttraumatic Amnesia

PTA refers to disruptions in a patient's memory for the events and experiences that occur immediately following a head trauma [6]. Assessments of orientation and PTA are integral in the inpatient setting because they (1) assist in documenting recovery and determining long-term prognosis, and (2) assist neuropsychologists in identifying the optimal timeframe for a patient to undergo a formal neurocognitive evaluation (i.e., after the patient has emerged from PTA). 
 Table 1 Commonly used inpatient cognitive screening measures

Mental status & posttraumatic amnesia	Brief cognitive screening measures
Galveston Orientation & Amnesia Test (GOAT) [9]	Montreal Cognitive Assessment (MoCA) (www.mocatest.org)
Children's Orientation & Amnesia Test (COAT) [8]	Mini Mental Status Examination (MMSE) [10]
Orientation Log (O-Log) [11]	Kokmen Short Test of Mental Status (STMS) [12]
Agitated Behavior Scale [13]	
Confusion Assessment Protocol [14]	

Determining whether a patient is oriented or has emerged from PTA is most commonly achieved through the administration of standardized measures such as the Galveston Orientation and Amnesia Test (GOAT) and Children's Orientation and Amnesia Test (COAT) [7–9]. Most commonly, emergence from PTA based on GOAT scores is defined by the first day of two consecutive days that GOAT scores of 76 or greater are recorded within a period of 24–72 h. These measures allow for a serial assessment of patients' orientation to person, place, and time and knowledge/recall of the events leading up to and following their brain injury and hospital admission. Screening measures of mental status may also help evaluate the patients' understanding of his/her diagnosis, associated cognitive changes, and their implications on functioning. Table 1 provides a list of commonly used assessments of PTA and cognition in the acute stetting. The reader is referred to the previous chapter by Sherer and Giacino for a more thorough discussion of brief inpatient screening assessments.

Neuropsychologists should avoid conducting neurocognitive evaluations before a patient has cleared from PTA, as testing under these circumstances typically yields unreliable and/or invalid estimates of long-term cognitive capabilities. Furthermore, interpreting neuropsychological test results during this period of time may have serious implications for the patient's future plan of care (e.g., issues related to discharge planning, capacity, etc). If evaluations must be conducted the neuropsychologist should note that the results represent the patient's mental status during a point in time that is marked by dramatic fluctuations in attention, alertness, and cognition.

# Inpatient Neuropsychological Evaluations

Over the last few decades there has been a dramatic decline in the length of acute rehabilitation stays (LOS) from 20 to 12 days [15]. These changes have impacted the role of neuropsychologists in inpatient rehabilitation settings, shifting the focus away from comprehensive assessments toward identifying the severity of cognitive and neurobehavioral sequelae, preparing patients and families for discharge, and the next phase of the patient's rehabilitation. If a patient emerges from PTA during their inpatient rehabilitation stay, a more formal assessment of cognitive functioning is possible and appropriate. The period after emergence from PTA represents an early stage of recovery and assessments must be conducted with an appreciation for the fact that cognition will most likely continue to improve. Subsequent evaluations (inpatient or outpatient) may be necessary in order to generate the most meaningful recommendations for the patient, treatment team, and family.

Given the numerous patient-factors (e.g., neurofatigue, reduced tolerance for testing, aphasia, sensory/perceptual difficulties) and institutional limitations (i.e., requirements by insurance/ national carriers that patients participate in several hours of therapy daily to justify payment), even more comprehensive inpatient neuropsychometric assessments are typically relatively brief. Such assessments should attempt to estimate a patient's pre-injury level of intellect and functioning and provide a screening of their capabilities across a number of cognitive domains. While a comprehensive assessment of attention, language, visuoperception, learning and memory, and executive functioning may be ideal, these domains are typically evaluated using abbreviated (and if possible, repeatable) measures such as the Repeatable Battery for Assessment of

Neuropsychological Status (RBANS) [16]. An alternative approach may also involve the selective administration of tests/subtests from lengthier test batteries. Inpatient assessments, which are conducted for the purposes of discharge and treatment planning, are most useful when they include a thorough examination of learning, memory, and executive functioning, since these domains have been widely associated with long-term functional outcomes (i.e., return to work and productivity) following TBI [17, 18].

### Assessments of Mood, Adjustment, and Coping

In addition to cognitive sequelae, it is common for individuals in the acute phase of recovery from TBI to experience changes in emotion and behavior. Inpatient neuropsychologists are uniquely qualified to assess mood, coping, and adjustment and determine the potential implication of these factors on the recovery process. As improvement occurs and patients emerge from PTA, survivors often begin to develop a better appreciation of what has happened to them. Assessment of mood and adjustment during this phase of recovery may include a clinical interview with the patient and family, the use of standardized mood questionnaires (e.g., Beck Depression/Anxiety Inventories, Patient Health Questionnaire-9, Generalized Anxiety Disorder-7 item), and consultation with other members of the inpatient care team. Standardized mood measures also provide a means of tracking patients' distress levels/symptoms and providing patients and their families with feedback about changes in mood symptoms over time.

A clinical interview should be used to evaluate a patient's current mood symptoms, psychiatric and substance abuse history, awareness of the nature/severity of their injury and functional limitations, adjustment to the hospitalization and inpatient therapies, family/support network, and coping strategies. An understanding of past psychiatric and substance abuse difficulties is important in determining whether or not a patient is at greater risk for developing mood symptoms post injury, or relying on maladaptive coping strategies (e.g., substance use) in the post-acute period. Likewise, assessments of family and social support (e.g., friends, religion/ faith, community involvement, etc.) help in identifying positive resources the patient can rely on as they transition from the acute to postacute phase of recovery.

Patients in early recovery may have difficulty articulating their emotions and rating their own mood states. These difficulties can be further complicated by an incomplete awareness of the extent and implications of their injuries. These patients' mood can often be best assessed by direct observation. Observing the patient while they are involved in therapies or interacting with family members can be one of the most useful means of understanding the patient's current mood state. Moreover, this approach provides a more naturalistic setting that allows the neuropsychologist to communicate practical recommendations to other team members and the patient's family.

Since occupational, speech, and physical therapists typically spend significantly more time with patients than neuropsychologists (even in a well-staffed inpatient rehabilitation unit), these professional colleagues may have observed a richer and more extensive sampling of patient behavior. Similarly, given their interactions with patients for up to 12 h at a time, nurses can provide information about episodes of emotional dysregulation (i.e., tearfulness, anger) or signs of emotional distress (e.g., anxiety) during medical procedures and changes in behavior over time. Other team members are likely to have useful insights regarding an individual's general disposition, engagement, and frustration tolerance during challenging therapeutic exercises. To provide individualized recommendations for treatment, an inpatient neuropsychologist must regularly interact with other team members. By joining the rehabilitation team, speaking in a common language, and working in a truly collaborative manner with the patient and family, neuropsychologists will be well-suited to provide accurate and useful information that facilitates improved treatment outcomes [19].

# Assessment in Post-acute Settings (Residential- and Clinic-Based)

Recovery following traumatic brain injury extends long past a patient's discharge from an inpatient rehabilitation unit and often continues for months to years. Given reduced lengths of stay and the artificial nature of the hospital setting, the majority of rehabilitation now takes place in outpatient settings during the post-acute period. With the initiation of outpatient rehabilitation comes a greater need for determining the patient's level of functioning within the family, social network, and community at large. As a result, neurocognitive assessments in this phase of recovery gradually evolve from describing changes in functioning over time to assessing the impact of ongoing symptoms on an individual's daily activities and community participation. Outpatient rehabilitation practices are highly individualized and informed by the ongoing neurocognitive sequelae as well as needs of the persons being served.

Traditionally, neuropsychological assessment has focused on correlating brain dysfunction with behavioral changes. Clinically, this often means (1) diagnosing the presence of underlying brain pathology (e.g., learning disability, dementia), and/or (2) describing the level and pattern of impairment associated with a known cause of brain dysfunction (e.g., stroke or TBI). In the post-acute rehabilitation setting there is often little question regarding the cause or etiology of the brain dysfunction. Neuropsychological testing conducted in this setting is often done for a different purpose, which include quantifying or predicting the degree of limitations an individual will experience in everyday life and assisting him/her with learning to compensate for residual limitations. This can be quite challenging given that the pattern of neurocognitive impairment can vary widely as a function of type/severity of injury and associated physical and emotional sequelae. Nonetheless, neuropsychologists are well-suited to (1) assess the cognitive, neurobehavioral, and environmental factors important to recovery, (2) evaluate levels of functioning within

larger social contexts, and (3) outline treatment plans that facilitate return to community participation following TBI.

# Model for Conceptualizing Factors to Be Assessed in the Post-acute Setting

To assist with the conceptualization of physical and mental changes caused by known medical conditions (such as brain injury) and associated changes in functioning, the World Health developed Organization the International Classification of Functioning, Disabilities, and Health [20]. This model classifies the sequelae of brain injury into (1) body functions and structures, (2) activity, and (3) participation. Body functions and structures are measured by the presence of normal or abnormal (impaired) physical or mental functions. Activity limitations are defined by an individual's inability to complete an activity due to impairments or changes in body functions and structures (e.g., inability to recall appointments, to follow a recipe while cooking, recall a medication regimen, balance a checkbook, etc.). Activity limitations focus on limitations in specific individual activities, in contrast to participation restrictions which involve societal level role fulfillment. Participation restrictions represent a loss or change in social roles due to changes in body functioning and associated activity limitations (e.g., loss of a job or inability to attend college). Participation is typically assessed through patient or family report and measured by the degree to which an individual is (1) an active, productive member of society, and (2) well integrated into family and community life. In other words, participation restrictions reflect whether individuals are limited in their ability to run a household and maintain a network of friends and family, as well as their involvement in productive activities such as employment, education, and volunteer activities.

In the ICF model, there is a dynamic interplay among changes in body functions and structures (physical and cognitive), activity limitations, and the participation restrictions that impact the person's reintegration into the community. The most recent iteration of the ICF model [21, 22] has shifted its emphasis from solely a medical model to a model which includes a consideration of the environmental and personal factors that impact long-term outcomes. See Fig. 1 for a graphical illustration of the ICF model.

In our view the ICF model provides clarity regarding the role of neuropsychological testing in post-acute settings and a model for identifying the factors that should be considered and assessed as part of any comprehensive evaluation of individuals with TBI. We find this model particularly useful for several specific reasons.



Fig. 1 The international classification functioning (ICF) model

As described above, neuropsychological assessment measures impairments (or the lack thereof) in cognitive abilities. While assessment of the presence and degree of cognitive impairment is important in understanding common symptoms following TBI, in most instances it does not directly assess limitations in activity. For example, a patient who is impaired (below the fifth percentile) in the acquisition and retention of items from a word list might be expected to have difficulty correctly learning and implementing a new medication regimen following TBI. While it would be easy to assume that these cognitive impairments would prevent the patient from engaging effectively in this task, our traditional neuropsychological measures do not directly assess an individual's ability to follow a medication regimen. Nor do they assess other environmental or personal factors that may facilitate or interfere with this process.

According to the ICF model, environmental or situational moderators/factors that may obstruct or facilitate successful completion of an important activity of daily living (i.e., managing medications) need to be considered in order to make truly accurate predictions about functioning. Such factors may include personal history/experience with medication management, the use of compensatory strategies, or environmental supports (i.e., pillbox along with a pager or alarm system). Even when done comprehensively, testing focused solely on measurement of cognitive impairment without considering these other relevant factors, may not accurately predict whether an individual can perform important day-to-day activities.

The ICF model also provides an important tool for understanding the values and mechanism of action for interventions such as cognitive rehabilitation following TBI. There is an evergrowing literature that supports the value of cognitive rehabilitation in helping to diminish the level of activity limitations and participation restrictions after brain injury [23]. These interventions are effective largely because they facilitate the development of behaviors which compensate for changes in mental and cognitive status. In other words, persons undergoing cognitive rehabilitation may experience improvement in functioning without necessarily experiencing any measureable diminution in cognitive impairment. As such, an intervention is successful if it results in improved real world behaviors and increased independence, whether or not there have been changes in neuropsychological performances [2, 24].

# Components of the Comprehensive Outpatient Assessment in the Post-acute Phase of Recovery Following TBI

Using the ICF model as a template, we will outline an approach for comprehensive assessment at the post-acute stage of recovery and rehabilitation. In order to obtain such a wide breadth of information, a comprehensive outpatient neuropsychological assessment should include information obtained from a review of the patient's medical record and history (i.e., focused on assessing degree of severity and other aspects of the injury; see Table 2), an interview with the patient and family members/ caregivers, and standardized measures of cognitive and overall psychological functioning. We have provided a detailed outline of these important components of the neuropsychological assessment in Table 2.

# **Clinical Interview**

In the interview with a patient and family members, it is particularly important to gather information from the following areas: the patient's current psychological functioning, pre-injury psychiatric and substance abuse history, coping strategies, availability of social support and resources, premorbid level of functioning, and current use of compensatory strategies to facilitate independence.

1	1 1	e
Component	Source	Information to be obtained
Injury-related data	1. Record review	<ol> <li>Information gathered through admission records and EMT report regarding injury causing conditions: GCS, length of PTA, type and site of injury, anoxia, other physical injuries</li> </ol>
		2. Concurrent medical conditions impacting cognition and functioning
		3. Imaging: CT, MRI, EEG
Past medical psychiatric	1. Record review	1. Premorbid medical conditions, substance abuse, and treatment history
history	2. Patient & family interview	2. Psychiatric disorders, past hospitalizations, mental health treatment
Psychosocial	1. Record review	1. Current mood, adjustment difficulties, self-awareness
assessment	2. Patient & family interview	2. Presence of current psychiatric disorders/situational stressors
	<ol> <li>Self-report measures of emotional &amp; personality functioning</li> </ol>	3. Current and past coping strategies (active vs. passive, negative vs. positive)
	4. Behavioral observations	4. Underlying personality traits
Cognitive functioning	1. Neuropsychological testing	1. Areas of strength and weakness compared to (1) baseline, (2) normative data, (3) intra-individually
		2. Insight and level of awareness of post-injury changes in cognition and function
Functioning in community	1. Record review	1. Level of education, history of academic difficulties, need for special education services
	2. Patient & family interview	2. Type of previous employment and consistency of
	3. School & work history	employment
Use of compensatory strategies	1. Patient & family interview	1. Frequency and type of compensatory strategies used at home, school, college, and work prior and after the injury (calendar system, personal assistive devices, cue cards, environmental supports)
Social support & resources	1. Patient & family interview	<ol> <li>Size of social network, patient and family's understanding of injury and its impact on functioning, capacity to provide assistance</li> </ol>
		2. Financial resources
		3. Patient's willingness to accept and ask for help

Table 2 Components of a comprehensive outpatient assessment following TBI

# Psychiatric and Substance Abuse History

As delineated in the ICF model, past history of substance use and psychiatric history impact the recovery trajectory of a person with TBI. As such, it is important to assess in detail past history of substance use, its frequency, types of substances used, and the typical settings in which these substances were used (i.e., to cope with stress, alone, socially, etc.) both prior to and since the injury. Obtaining a thorough assessment of psychiatric and substance use history is crucial to any assessment, since it is meant to provide an accurate and complete evaluation of functioning and to lead to appropriate treatment recommendations.

Moreover, past psychiatric or substance abuse conditions may be contributing to cognitive impairments which are identified on testing and may in turn be a factor impacting a patient's current level of functioning. Premorbid substance and psychiatric histories can also identify those patients who are at increased risk for maladaptive coping or emotional difficulties following TBI. Assessing for these premorbid conditions is best done in the context of a thorough clinical interview of the patient, including information obtained from a reliable collateral source such as a family member. Brief alcohol disorders screening questionnaires such as the CAGE (Cut down, Annoyed, Guilty, Eye opener) [24], MAST (Michigan Alcoholism Screening Test) [25], or AUDIT (Alcohol Use Disorders Identification Test) [26] may also help with this process.

### Assessment of Awareness and Psychosocial Functioning

Historically, the assessment of awareness, personality, motivation, and other psychological factors was considered important only to the extent to which they interfered with the validity of the psychometric evaluation [27]. However, evaluations with such a narrow focus have been criticized as not providing sufficient information for conceptualizing the whole individual and have been replaced with more comprehensive evaluations of functioning. This broadening of the scope of neuropsychological evaluations is even reflected in changes between the third and fourth edition of Lezak's classic text on neuropsychological assessment, which now highlights emotional factors as integral components of a neuropsychological evaluation [28] (see Table 3). This change may be largely due in part to the movement of the field into rehabilitation settings and the consequent need to describe not only cognitive functioning but also how psychological factors may impact "real-world" situations.

Research has shown that psychosocial morbidity is often associated with increased long-term disability [29], unemployment [30, 31], and poorer rehabilitation treatment outcomes [32, 33] after TBI. Furthermore, impaired self-awareness is a common symptom of severe brain injury and is a strong predictor of long-term functional outcomes and employment [34, 35]. Crosson and colleagues argue that to be truly effective, clinical interventions in the post-acute phase of recovery need to incorporate an accurate assessment of self-awareness into specific treatment interventions [36].

Given the aforementioned correlations among awareness, mood and psychosocial disability, and long-term outcomes following TBI, it is important to address these psychosocial variables when conducting a comprehensive neuropsychological evaluation in the post-acute setting. One of the most valuable services provided by a neuropsychological assessment may be to correctly identify the presence of substance abuse or mood disorder, the nature and extent of their impact on functioning, and make appropriate referrals to qualified providers. Data regarding mood and psychosocial functioning may be best acquired through the patient and family interview, but may also be obtained through the use of standardized measures of mood, coping, awareness, and personality functioning. Specifically, measures such as the Beck Depression Inventory-II (BDI-II) [37], Beck Depression Inventory-Fast Screen (BDI-FS) [38], Beck Anxiety Inventory (BAI) [39], Patient Health Questionnaire (PHQ-9) [40], and Generalized Anxiety Disorder (GAD-7) [41] have been found to be useful adjunctive tools in neuropsychometric assessments. Such measures can easily be incorporated into the clinical interview or administered at the time of the neuropsychological assessment to assist the clinician with conceptualizing the patients' level of emotional functioning, associated needs, and assessing their capacity to benefit from rehabilitation interventions.

### Functioning in the Community: Premorbid and Current

Estimating pre-injury functioning is an important part of neuropsychological evaluation. In order to determine the optimal long-term outcome and the patient's ability to return to pre-injury level of functioning and to work, we first need to determine their pre-injury level of functioning. Lezak et al. [28] noted that accurately assessing the individual's educational and work history is crucial as it is one of the best predictors of postinjury level of functioning. Prior occupational history and the nature of pre-injury jobs are associated with post-injury employability 2-5 years post injury [42].

Vocational functioning following brain injury has key economic and clinical effects on

Table 3         Cognitive domains examined durin	ng neuropsychological evaluation	
Adapted from Lezak (1995)	Adapted from Lezak (2004)	Typical neuropsychological battery
1. Orientation and attention <sup>a</sup>	1. Mental activity <sup>b</sup>	WAIS-III/IV Working Memory Factor
	<ul> <li>Consciousness<sup>b</sup></li> </ul>	WAIS-III/IV Cognitive Speed Factor
	<ul> <li>Activity rate/speed of processing</li> </ul>	Continued Performance Test of Attention
	Attention	Trail Making Test (TMT) A and B
		Stroop Test
2. General cognitive abilities	2. General cognitive abilities	Wechsler Adult Intelligence Scale (WAIS-III/ IV)
	Intellectual functioning	
3. Perception <sup>a</sup>	3. Receptive functions <sup>b</sup>	WMS-III Orientation Subtest
	Sensory reception/perception	Benton Visual Form Discrimination
	Orientation	Judgment of Line Orientation
	<ul> <li>Awareness</li> </ul>	Rey-Osterrieth Complex Figure Test (Copy)
	Recognition/discrimination/patterning	WAIS-IV Visual Puzzles
4. Memory	4. Memory	WMS III/ IV Logical Memory I and II
	<ul> <li>Declarative/explicit</li> </ul>	WMS III/IV Visual Reproductions I and II
	Non-declarative/implicit memory	Rey Auditory Verbal Learning Test (AVLT)
5. Verbal and language functions	5. Expressive functions	Boston Naming Test
	Language	COWAT
	Constructional disorders	Category Fluency
		BDAE Complex Ideational Material
6. Construction <sup>a</sup>	6. Executive functions	Frontal Systems Behavioral Scale (FrsBe)
7. Concept formation and reasoning <sup>a</sup>	Cognitive flexibility	Wisconsin Card Sorting Test (WCST)
	Concept formation/abstract reasoning	DKEFS Tower Test
	Planning/organization	
	Goal-directed behavior <sup>b</sup>	
8. Executive functions	7. Personality/emotional variables <sup>b</sup>	Beck Depression Inventory (BDI-II)
9. Motor/sensory abilities <sup>a</sup>	Emotional lability/dullness/euphoria	Beck Anxiety Inventory (BAI)
	Disinhibition/impulsivity	Minnesota Multiphasic Personality Inventory (MMPI-2)
	<ul> <li>Reduced/increased social sensitivity</li> </ul>	Personality Assessment Inventory
	<ul> <li>Depression/anxiety</li> </ul>	
<sup>a</sup> Cognitive domains listed in the third edition <sup>b</sup> Cognitive domains added to the fourth editi	n of Neuropsychological Assessment textbook but remov ion of Neuropsychological Assessment textbook [28]	ved in the fourth edition of the textbook [28]

reintegration to life and is important to consider when examining predictors of participation restrictions in individuals with brain injury. While often overlooked, inquiring about past involvement in volunteer activities can also provide useful information. This can help to (1) identify skills an individual may have beyond those which they use on the job, and (2) determine the extent to which they value work in an unpaid setting. Many skills used in volunteer settings may be very similar to those used in paid employment. Volunteering after brain injury has also been shown to be associated with enhanced psychological well-being and may also provide an important step towards community-based employment [43].

Information on educational history, employment history, and volunteering should all be a standard part of the clinical interview. This would include specific inquiries about any challenges faced in academic settings (i.e., tutoring, need for special education services, repeating grades), accommodations on the job, and a history of any difficulty with maintaining steady employment. Knowledge of on-the-job difficulties prior to the TBI, such as history of frequent conflicts with coworkers or charges of sexual harassment, can be extremely helpful in developing plans for post-injury return to work. It should always be kept in mind that what appears to be problematic behavior directly related to the effect of brain injury may actually be a pattern of behavior which long predates the brain injury. Failure to obtain an accurate history of pre-injury functioning in the work place may lead to incomplete plans for return to work. Patients may be unwilling to disclose unpleasant aspects of their history, which only serves to reinforce the need for additional sources of information such as family members, former employers, or coworkers.

#### Instrumental Activities of Daily Living

As mentioned above, traditional neuropsychological tests do not directly assess daily activity limitations. However, daily activity limitations are the core difficulties that significantly impact a person's ability to return to or sustain pre-injury levels of functioning and independence [44, 45].

<b>T C</b>	<b>D</b>	• •		
	Rora	annet	0t 1	
1.1.	Deru	uuisi	erai	

Instrumental activities of daily living (IADL)-complex skills required to live independently	Activities of daily living (ADL)-basic self-care skills learned in early childhood
Telephone use	Feeding
Using public transportation	Toileting
Shopping	Grooming
Cooking/preparing meals	Bathing
Driving	Walking and transferring (e.g., from bed to chair)
Housework	Selecting proper clothing
Medications management	Dressing
Finance management	Maintaining continence

Table 4 Common ADLs and IADLs

Functional abilities are typically divided into two subgroups: Instrumental activities of daily living (IADLs) and basic activities of daily living (ADLs). Given that discussion of ADLs may not be common focus for many neuropsychologists, we have listed many common IADLs and ADLs in Table 4. Neuropsychologists working in outpatient rehabilitation settings typically focus on IADLs that encompass activities that allow individuals to function independently in everyday life including shopping, communication, driving, managing finances and medications, cooking, and transportation.

Outpatient neuropsychological evaluations should assess the past and current level of activity limitations through clinical interviews. Interviewing family members or other persons who have been able to directly observe the patient's function in day-to-day life can also be helpful with this assessment. When considered in the context of outpatient rehabilitation, which typically focuses on increasing a patient's activity and participation through the implementation of compensatory strategies rather than by remediating cognitive impairments, the utility of assessing IADLS becomes quite apparent.

#### **Use of Compensatory Strategies**

The development and application of compensatory strategies such as a use of a planner/calendar in therapy has been demonstrated to be an effective means of improving functioning and diminishing the impact of cognitive impairment on day to day life. While we know of no specific scale available to measure compensatory strategy use at present, direct inquiry as to the type and frequency of compensatory strategy use should be included as part of any comprehensive assessment. Assessing baseline compensatory strategy use is particularly relevant since research has shown that the exact frequency of compensatory strategy use may be associated with the level of independence in patients after acquired brain injury [46].

Our research has shown that successful completion of an internet-based cognitive rehabilitation program was more strongly correlated with baseline compensatory strategy use than cognitive impairment [47]. Moreover, we also found that improved functioning after treatment was associated with greater level of compensatory strategy use. Inquiring about attitudes toward compensatory strategies may further elucidate the discrepancy between a patient's current and predicted performance levels solely based on the measurement of cognitive impairment. It can also provide a means of determining the degree to which a patient will be open to, or capable of adopting, a treatment program suggested by their rehabilitation team members.

### Neuropsychometric Testing

### Symptom Validity and Measuring Objective Effort

In recent years there has been substantial growth in assessment of symptom validity as a part of routine neuropsychological evaluations. Inclusion of such measures in forensic evaluations is considered important to reach an accurate assessment of cognitive functioning. For persons working in clinical settings in which patients are referred for reasons of clinical need and not medical legal issues, true malingering is rare in our experience. However, poor performance on symptom validity testing still occurs in clinical settings. Discussion of effort testing and malingering in the TBI population is beyond the scope of this chapter and is discussed in more detail elsewhere in this book. However, we would briefly like to highlight some of the factors that should be considered when individuals presenting to a brain injury rehabilitation setting fail objective measures of effort. Possible factors which may account for suboptimal effort may include, but are not limited to, (1) the effects of comorbid medical/physical or sensory impairments, (2) the effect of pain, fatigue, or emotional distress on the patient's ability to engage in the evaluation, (3) communicating a cry for help, or (4) the effect of cognitive self-schemas and appraisals about the impact of the brain injury [48].

### **Behavioral Observations**

Beyond actual test performance, the emotional and physical reaction to undergoing testing can provide very useful information. Behavioral observations from the examiner and/or psychometrist are often quite valuable and may provide insight into a patient's frustration tolerance, reactions to success and failures, and the effect of fatigue on performance over the course of the evaluation. These behaviors may mirror many of the difficulties which individuals with brain injuries experience in challenging daily situations. If the focus of the assessment is on test performance alone, this potentially valuable information will be missed. By conducting an assessment that comprehensively measures psychological and cognitive functioning and qualitative aspects of performance during and after testing, neuropsychologists are better suited to integrate information from the clinical interview and assessment and develop a more sophisticated and comprehensive understanding of the individual with TBI.

### **Cognitive Functioning**

It is beyond the scope of this chapter to provide a detailed discussion of test selection when conducting an assessment, but a sampling of tests that address each component of the neuropsychological evaluation in a post-acute rehabilitation setting are included in Table 3. Many other texts describe the merits of specific tests and issues to be attended to more generally concerning test selection, administration, and interpretation [28]. While any neuropsychological assessment should comprehensively assess cognitive domains [28], some areas of cognitive functioning are stronger correlates of long-tern functional outcome than others. Executive functioning tests-including measures of cognitive flexibility and planning, speed of processing information, attention, and memory-have been shown to better predict psychosocial outcome both at 1-year and 10-years post injury, as compared to general measures of intellectual functioning and verbal fluency [49-52]. At the same time, measurement of executive functioning within limits imposed by the artificial and controlled nature of the testing environment is quite challenging. Consequently, family/ significant other ratings of specific behaviors associated with executive dysfunction, such as the Behavior Rating Inventory of Executive Functioning (BRIEF) or Frontal Systems Behavior Scale (FrSBe), are a helpful addition to any assessment.

# Successful Integration of Neuropsychology into the Outpatient Rehabilitation Team

In outpatient brain injury rehabilitation settings, team treatment and assessment are viewed as the gold standard of clinical service [53-55]. The challenge of a truly interdisciplinary approach to assessment and rehabilitation is working together in a manner in which each discipline compliments the role of the other as a result of joint commitment to the same patient population [56]. Practicing in such a setting, in which a neuropsychological assessment is an integral part of an overall team evaluation, can be extremely rewarding both professionally and clinically. It can also present a host of challenges due to working with disciplines whose approaches to assessment often differ from that typically conducted by neuropsychologists.

First and foremost, other practitioners within the rehabilitation discipline may not necessarily view a neuropsychological evaluation as offering any added value to the treatment process. This is particularly true if the neuropsychologist providing assessment services is not an actual member of the treatment team, but provides assessments in the role of an outside consultant. This lack of perceived value in the role of the neuropsychologist is often due to a lack of knowledge about the unique information which a neuropsychological evaluation can provide and/or having team members who experienced working with neuropsychologists who conduct evaluations which are narrowly focused on "impairment." Among typical rehabilitation team members, neuropsychologists have unique expertise in relating patterns of cognitive dysfunction with brain lesions and understanding the impact of factors such as mood, psychiatric history, personality style, and effort on cognitive functioning.

Within the context of a rehabilitation team, neuropsychologist's expertise can also be used to help strengthen the findings and assessments conducted by other team members. If done with professional respect and deference, promoting the value of a neuropsychological assessment can help enhance the reputation of the neuropsychologist on the team, increase the value of the findings of the assessments of other team members, and most importantly yield results that holistically describe the patient and their current situation.

Blair and Gorman [19] outlined the following common challenges that neuropsychologists face in rehabilitation settings: (1) the need to join the system (rather than simply consulting it), (2) deferring to other rehabilitation team members, and (3) helping other disciplines integrate what on the surface may appear to be disparate findings. For instance, examples of confusing and often contradictory behavior (e.g., having difficulty with initiation in one setting, versus being extremely disinhibited in another) may actually be examples of a common underlying problem (e.g., frontal systems dysfunction). By becoming actively involved with the team and practicing active listening, the neuropsychologist can uniquely integrate the opinions and findings of other team members, help with

behavioral management, and lead to an assessment which is much more than simply the sum of its collective parts.

## Providing Feedback to Patients, Families, Caregivers, and Other Rehabilitation Providers

Even a well-done comprehensive neuropsychological assessment is of no real value if the results are not conveyed to the persons being served. Only when these persons truly are assisted to understand, digest, and use this information, will the assessment have the intended impact upon the patient's situation. In our view, the work of the neuropsychologist conducting the evaluation is not entirely complete until feedback has been provided.

By giving feedback to the referral source, the neuropsychologist has the opportunity to provide more than information on changes in cognition and behaviors following brain injury. By incorporating the ICF model and including key categories of information which we have described here, the neuropsychologist can convey the important message that functioning may be understood only when the focus of the assessment goes beyond level of impairment. This may help a referral source understand why an individual with a long history of being an overachiever, and whose sense of self is tied to their professional productivity and ability to "do it all," seems disproportionately impacted by what seem to be very minimal changes in cognitive functioning on formal testing. Alternatively, providing feedback to a referral source may allow a neuropsychologist to explain why an individual with notable cognitive impairment but an established pattern of compensation strategies may be able to live in the community with minimal support. Providing this feedback and education is particularly important when the referral source is someone who is not familiar with rehabilitation and issues related to functioning. In other words, the neuropsychologist can highlight the idea that issues beyond level of impairment in mental skills determine level of functioning and, consequently, provide valuable service beyond the referral question.

Most neuropsychologists report that they commonly provide feedback directly to patients and that this information is viewed as helpful and positive by the vast majority [57]. Given the potential impact, direct feedback to the patient should be framed in a manner that is understandable and therapeutic. The challenge in giving feedback to persons with impaired cognitive function and minimal appreciation of their various limitations is in conveying this information in a meaningful and applicable-to-daily-life manner. The key point to remember is that the person receiving feedback may have cognitive difficulties that interfere with the ability to understand, remember, and process the information and how the findings would impact his/her daily functioning. As such, it is most important to involve key family members, caregivers, or other interested parties in the feedback session.

Correlating specific test results with examples of problems in everyday living, as well as using analogies and even metaphors for the problem being discussed, can be more palatable [58]. Frequent use of pauses, clarifications, and having the patient restate what they have learned can also be helpful. If the patient is scheduled to return for follow-up therapy, then further sessions may help patients in developing strategies to address problems. Finally, the feedback session should be followed-up with a letter summarizing the discussion.

While the assessment is focused on the patient, family and caregivers are also commonly experiencing significant emotional stress associated with adapting to the complex behavior and personality changes that occur after TBI. A majority of caregivers for persons with TBI report increased stress [59] and almost half of persons living with a loved one with TBI endorse symptoms that meet criteria for one or more psychiatric diagnoses [18]. Addressing the well-being of caregivers and family members can facilitate the rehabilitation and recovery of the family member with TBI [60]. Providing feedback to family members can validate their perceptions of the major issues which are impacting the survivor's functioning and provide additional insights which can lead to better methods of dealing with common challenges.

The feedback session also provides a venue for the discussion of issues that may predate the injury, but which the family had been unwilling or unable to confront previously. A family who is reluctant to discuss mental health issues or who has a permissive attitude about substance use can be provided with information that facilitates their understanding of how these factors impact the recovery and treatment of brain injury. By understanding how these issues impact recovery from brain injury, family members may be empowered to help their loved one seek out treatment for a problem which has existed for some time.

While feedback regarding assessment results to the patient and their family is obviously important, it is also important to provide feedback to rehabilitation team members who will ultimately provide treatment to address the various challenges described in the assessment. As we have described earlier, this information will be better received and utilized if the neuropsychologist providing the assessment is also functioning as part of the treatment team. Such an arrangement provides the obvious advantage of the opportunity to revisit concerns raised in the assessment as the team continues to work with the patient. Providing test feedback during one or more therapy sessions provided by key team members can be a very powerful means of increasing the chance that test results will be incorporated into treatment. For neuropsychologists who function in a consulting role, it is important that they find a forum in which the information is not simply relayed to treatment team members, but in which there is an active dialogue in which questions can be answered. This can best be done by participating in a regular team meetings and patient care conferences.

### Case Study

Jan is a 46-year-old female who was involved in a motor vehicle crash while driving to work, when her car slid off of the road one January morning and struck a tree. She was a restrained driver, the only person in the car, and no other vehicles were involved in the accident. She may have had some loss of consciousness, although there were no observations of that at the scene. Within minutes after the accident, she was found by a passing motorist who described her as being groggy but awake and talking in a somewhat nonsensical manner. Emergency responders were called and she was brought to the local hospital emergency department, where work-up including imaging studies of the brain were found to be negative.

She has no memory for the car crash or for events occurring an hour before or after, and incomplete memory for events for up to 24 h after the crash. She also suffered a fractured left femur for which she underwent orthopedic surgery. Her hospital stay lasted for several days, during which time her mental status quickly improved. Because of her femur fracture, she was for a time transferred to an extended care facility and ultimately returned home. She later went on to receive physical therapy once her weight bearing status was improved. Due to the rapid improvement in her mental status, no complaints of changes in her cognition, and the fact that she did relatively well on screening exams of her mental status within a few days of hospital admission, no further therapy was ordered to address cognitive issues.

At the time of her crash, Jan was employed as a full professor in humanities at a private religious-affiliated college. She had become a full professor just several years before and was quite accomplished and well published in her field. She is a single parent of three children and has primary responsibility for raising the children. Her children range from upper high school to upper grade school level. She was described by others as someone who is able to handle a variety of responsibilities and quite gifted at multitasking. In addition to her full time work and her responsibilities as a parent, she also was involved in a community singing group which performed throughout the region.

As her physical recovery improved, Jan attempted to return to work, but found this quite difficult. This included managing her teaching load, her writing responsibilities, and other professional organizations in which she participated. She felt overwhelmed by her work, was increasingly frustrated, and even became despondent to the point of concern for possible depression. She was ultimately referred to a specialized brain rehabilitation program approximately 6 months after her injury. She underwent workup, including neuropsychological evaluation, which found some mild slowing in her speed of cognition and marginally poor performance in selected aspects of executive functioning (e.g., conceptual reasoning ability), but otherwise generally average, or in most cases, above average performance. She initiated a program of cognitive rehabilitation. Prior to her injury, Jan had made limited use of compensation strategies, such as a planner, and prided herself on her ability to keep track of her own schedule and that of her children and other tasks quite well "in her head."

Further conversation with her indicated that she is someone, who when growing up, received limited reinforcement and encouragement for her many achievements from her parents in their attempts to not treat her differently than her siblings who were not as accomplished as she was. As a result, in order to obtain some sense of acknowledgment from her parents, she developed a strong focus on achievement which also was her principle means of developing any sense of self-worth and acknowledgment. She also admitted she enjoyed "doing it all," and pushing herself to the limit of her abilities. At such times, she felt as though she was accomplishing something and felt good about herself.

Initial attempts by her therapy team to help her develop compensation strategies and organizational strategies were met with a great deal of opposition and frustration, given that she could no longer "do it all in her head." She felt that having to write things down and rely on strategies to help her organize, retain information, and plan ahead for activities, was a reflection of the loss of ability that she had suffered and diminished her sense of self-esteem and self-worth. Further exacerbating her stress was the fact that many individuals who knew her were perplexed as to why she did not go back to work, and many even suggested that she could return to work if she really tried harder. In fact, several attempts to return to work early in her recovery had only caused her greater frustration and seemingly increased her difficulties with organizational ability, diminished fatigue, and increased irritability. In seemed to her that the harder she tried to function, the worse she ended up doing. To address these significant emotional and interpersonal issues, she was seen for psychotherapy in coordination with her cognitive rehabilitation, with the primary aim of maximizing the effectiveness of her treatment.

This case points out the need to go beyond traditional neuropsychological testing, focused as it is on cognitive impairment, and understand: the demands of the environment in which the individual was functioning previously and towards which they would like to return; historical, emotional, and psychosocial factors that may be impacting this individual at present, particularly aspects of personality style which impact interpretation of the changes associated with their history of traumatic brain injury; current use of compensation strategies and receptiveness to further developing such strategies.

#### Summary

The neuropsychological assessment provides a unique means of describing the many factors which impact functioning in the person with brain injury. In the acute phase of recovery, evaluation typically focuses on assessment of fluctuations in mental status, emotional adjustment, and neurobehavioral sequelae. In the post-acute phase of recovery, the focus of assessment changes to examining factors which together explain the challenges a patient faces in everyday life and making treatment recommendations. This type of assessment requires a change in focus from traditional neuropsychological assessment, with its emphasis on documenting level of cognitive impairment, to a model that appreciates and accounts for differences between changes in mental and body structures, activity limitations, and participation restrictions. Such a model also examines the various individual

and environmental factors that impact recovery, rehabilitation, and long-term outcomes. When a neuropsychologist becomes an integral part of the rehabilitation treatment team, it (1) ensures that the results of the assessment are provided to patients, their families, and referral sources, (2) provides results of the assessment as a means of understanding challenging cognitive and behavioral issues, and (3) increases the likelihood of treatment success, independence, and improved quality of life.

Acknowledgments Preparation of this chapter was partially supported by U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grant H133A120026.

# References

- Gomar, J. J., Bobes-Bascaran, M. T., Conejero-Goldberg, C., Davies, P., & Goldberg, T. E. (2011). Utility of combinations of biomarkers, cognitive markers, and risk factors to predict conversion from mild cognitive impairment to Alzheimer disease in patients in the Alzheimer's disease neuroimaging initiative. Archives of General Psychiatry, 68, 961–969.
- Marcotte, T. D., Scott, J. C., Kamat, R., & Heaton, R. (2010). Neuropsychology and the prediction of everyday functioning. In T. D. Marcotte & I. Grant (Eds.), *Neuropsychology of everyday functioning*. New York, NY: Guilford Press.
- Braun, M., Tupper, D., Kaufmann, P., McCrea, M., Postal, P., Westerveld, M., et al. (2011). Neuropsychological assessment: A valuable tool in the diagnosis and management of neurological, neurodevelopmental, medical, and psychiatric disorders. *Cognitive and Behavioral Neurology*, 24, 107–114.
- Sweet, J., Giuffre, M. D., Nelson, N., & Moberg, P. (2011). The TCN/AACN 2010 "salary survey": Professional practices, beliefs, and incomes of U.S. neuropsychologists. *The Clinical Neuropsychologist*, 25, 12–61.
- Meythaler, J. M., Peduzzi, J. D., Eleftheriou, E., & Novack, T. (2001). Current concepts: Diffuse axonal injury–associated traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 82, 1461–1471.
- Lucas, J. A., & Addeo, R. (2006). Traumatic brain injury and postconcussion syndrome. In P. J. Snyder, P. D. Nussbaum, & D. L. Robins (Eds.), *Clinical neuropsychology: A pocket handbook of assessment* (2nd ed.). Washington, DC: American Psychological Association.
- Bode, R., Heinemann, A., & Semik, P. (2000). Measurement properties of the Galveston Orientation and Amnesia test (GOAT) and improvement patterns during inpatient rehabilitation. *The Journal of Head Trauma Rehabilitation*, 15, 637–655.

- 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 for cute head injury and to recovery of memory. *Neurosurgery*, 27, 683–691.
- Levin, H. S., O'Donnell, V. M., & Grossman, R. D. (1979). The Galveston Orientation and Amnesia Test. A practical scale to assess cognition after head injury. *The Journal of Nervous and Mental Disease*, 167, 675–684.
- Crum, R., Anthony, J., Bassett, S., & Folstein, M. (1993). Population-based norms for the mini-mental state exam by age and educational level. *JAMA*, 269, 2386–2391.
- Jackson, W., Novack, T., & Dowler, R. (1998). Effective serial measurement of cognitive orientation in rehabilitation: The orientation log. *Archives of Physical Medicine and Rehabilitation*, 79, 718–720.
- Kokmen, E., Naessens, K., & Offord, K. (1987). A short test of mental status: Description and preliminary results. *Mayo Clinic Proceedings*, 62, 281–288.
- Bogner, J. (2010). The Agitated Behavior Scale. The Center for Outcome Measurement. *Brain Injury*. Retrieved from http://www.tbims.org/combi/abs
- Sherer, M., Nakase-Thompson, R., Yablon, S., & Gontkovsky, S. (2005). Multidimensional assessment of acute confusion after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 86, 896–904.
- Ottenbacher, K., Smith, P. M., Illig, S., Linn, R., Ostir, G., & Granger, C. (2004). Trends in length of stay, living setting, functional outcome and mortality following medical rehabilitation. *JAMA*, 292, 1687–1695.
- Randolph, C. (1998). *RBANS manual: Repeatable battery for the assessment of neuropsychological status*. San Antonio, TX: The Psychological Corporation.
- Boake, C., Millis, S. R., High, W. M., Delmonico, R. L., Kreutzer, J. S., Roosenthal, M., et al. (2001). Using early neuropsychologic testing to predict longterm productivity outcome from traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 82, 761–768.
- Kreutzer, J. S., Gervasi, A. H., & Camplair, P. S. (1994). Patient correlates of caregivers' distress and family functioning after traumatic brain injury. *Brain Injury*, 8, 211–230.
- Blair, K. L., & Gorman, P. W. (2003). Survival tips for the neuropsychologist in an inpatient rehabilitation setting. *Rehabilitation Psychology*, 48, 310–313.
- 20. World Health Organization. (2001). International classification of functioning, disability and health (ICF). Geneva, Switzerland: Author.
- Hurst, R. (2003). The international disability rights movement and the ICF. *Disability and Rehabilitation*, 25, 572–576.
- Threats, T. T., & Worrall, L. (2004). Classifying communication disability using the ICF. *International Journal of Speech-Language Pathology*, 6, 53–62.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., et al. (2011). Evidence-based cognitive rehabilitation: Updated

review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation*, *92*, 519–530.

- 24. Ewing, J. A. (1984). Detecting alcoholism: The CAGE Questionnaire. *JAMA*, 252, 1905–1907.
- Ashman, T., Schartz, M., Cantor, J., Hibbard, M., & Gordon, W. (2004). Screening for substance abuse in individuals with traumatic brain injury. *Brain Injury*, 18, 191–202.
- Ponsford, J., Whelen-Goodinson, R., & Bahar-Fuchs, A. (2007). Alcohol and drug use following traumatic brain injury: A prospective study. *Brain Injury*, 21, 1385–1392.
- Bergquist, T. F., & Malec, J. F. (2002). Neuropsychological assessment for treatment planning and research. In P. J. Eslinger (Ed.), *Neuropsychological interventions: Clinical research and practice*. New York, NY: Guilford Press.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York, NY: Oxford University Press.
- Dikmen, S. S., Machamer, M. A., Powerll, J. M., & Temkin, N. R. (2003). Outcome 3 to 5 years after moderate to severe traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84, 1449–1457.
- Ben-Yishay, Y., Silver, S. M., Piasetsky, E., & Rattock, J. (1987). Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *The Journal of Head Trauma Rehabilitation*, 2, 35–48.
- Sander, A. M., Krentzer, J. S., Rosenthal, M., Delmonico, R., & Young, M. E. (1996). A multicenter longitudinal investigation of return to work and community integration following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 11(5), 70–84.
- Koehler, M. (1989). Relationship between selfconcept and successful rehabilitation. *Rehabilitation Nursing*, 14, 9–12.
- Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: A literature review and recommendations. *Brain Injury*, 9, 81–92.
- McGlynn, S. M., & Schachter, D. L. (1989). Unawareness of deficits in neuropsychological disorders. *Journal of Clinical and Experimental Neuropsychology*, 11(2), 143–205.
- Sherer, M., Oden, K., Berfloff, P., Levin, E., & High, W. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *NeuroRehabilitation*, 10, 25–37.
- 36. Crosson, B., Barco, P. P., Velozo, C. A., Bolesta, M. M., Cooper, P. V., Werts, D., et al. (1989). Awareness and compensation in postacute head injury rehabilitation. *The Journal of Head Trauma Rehabilitation*, *3*, 46–54.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck Depression Inventory—II manual (2nd ed.). San Antonio, TX: The Psychological Corporation.
- Beck, A. T., Steer, R. A., & Brown, G. K. (2000). *BDI: Fast screen for medical patients*. San Antonio, TX: The Psychological Corporation.

- Beck, A. T., & Steer, R. A. (1993). Beck Anxiety Inventory manual. San Antonio, TX: The Psychological Corporation Harcourt Brace & Company.
- Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16, 606–616.
- Spitzer, R. L., Kroenke, K., Williams, J. B., et al. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine*, 166(10), 1092–1097.
- Fleming, J., Tooth, L., Hassel, M., et al. (1999). Prediction of community integration and vocational outcome 2±5 years after traumatic brain injury rehabilitation in Australia. *Brain Injury*, 13, 417–431.
- Ouellet, M. C., Morin, C., & Lavoie, A. (2009). Volunteer work and psychological health following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 24, 262–271.
- Seidenberg, M., Taylor, M. A., & Haltiner, A. (1994). Personality and self report of cognitive functioning. *Archives of Clinical Neuropsychology*, 9(4), 353–361.
- 45. Sunderland, A., Harris, J. E., & Baddeley, A. D. (1983). Do laboratory test predict everyday memory? A neuropsychological study. *Journal of Memory and Language*, 22, 341–357.
- Wilson, B. A. (1991). Long-term prognosis of patients with severe memory disorders. *Neuropsychological Rehabilitation*, 1, 117–134.
- 47. Bergquist, T. F., Gehl, C., Mandrekar, J., Lepore, S., Hannah, S., Osten, A., et al. (2009). The effect of internet-based cognitive rehabilitation in persons with memory impairments after severe traumatic brain injury. *Brain Injury*, 23, 790–799.
- Locke, D. E. C., Smigielski, J. S., Powell, M. R., & Stevens, S. R. (2008). Effort issues in post-acute outpatient acquired brain injury rehabilitation seekers. *NeuroRehabilitation*, 23, 273–281.
- 49. Ponsford, J., Draper, K., & Schonberger, M. (2008). Functional outcome 10 years after traumatic brain injury: Its relationship with demographic, injury severity, and cognitive and emotional status. *Journal* of International Neuropsychological Society, 14, 233–242.
- Royall, D. R., Palmer, R., Chiodo, L. K., & Polk, J. M. (2005). Executive control mediates memory's association with change in instrumental activities of daily living: The Freedom House Study. *Journal of American Geriatrics Society*, 53, 11–17.
- Royall, D. R., Lauterbach, E. C., Kaufer, D. I., Malloy, P., Coburn, K. L., & Black, K. J. (2007). The cognitive correlates of functional status: A review from the Committee on Research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neurosciences*, 19, 249–265.
- 52. Vilkki, J., Ahola, K., Holst, P., Ohman, J., Servo, A., & Heiskanen, O. (1994). Prediction of psychosocial recovery after head injury with cognitive tests and neurobehavioral ratings. *Journal of Clinical and Experimental Neuropsychology*, *16*(3), 325–338.

- Fordyce, W. (1981). ACRM presidential address on interdisciplinary peers. Archives of Physical Medicine and Rehabilitation, 62, 51–53.
- Prigatano, G. P. (1999). Principles of neuropsychological rehabilitation. New York: Oxford University Press.
- 55. 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, 179–181.
- Diller, L. (1990). Fostering the interdisciplinary team: Fostering research in a society in transition. *Archives* of *Physical Medicine and Rehabilitation*, 71, 275–278.

- Smith, S., Wiggins, C., & Gorske, T. (2007). A survey of psychological assessment. Assessment, 14, 310–319.
- Bergquist, T., & Jacket, M. (1993). Awareness and goal setting with the traumatically brain injured. *Brain Injury*, 7, 275–282.
- 59. Hanks, R. A., Rapport, L. J., & Vangel, S. (2007). Caregiving appraisal after traumatic brain injury: The effects of functional status, coping style, social support and family functioning. *NeuroRehabilitation*, 22, 43–52.
- Rotundi, A. J., Sinkule, J., & Spring, M. (2005). An interactive web-based intervention for persons with TBI and their families. *The Journal of Head Trauma Rehabilitation*, 20, 173–185.

# **Outcome Assessment**

### Nicholas J. Pastorek and Tracy L. Veramonti

#### Abstract

Outcome assessment in rehabilitation has gained increasing attention over the last decade, in part due to the influence that outcome assessment can exert on patients, clinicians, researchers, and third party payors. Following traumatic brain injury (TBI), relevant targets for outcome assessment include an almost innumerable list of signs, symptoms, and/ or functional limitations and restrictions. Furthermore, outcome after TBI is a dynamic construct dependent on a host of factors such as severity of injury, time since injury, age, social supports, and availability of rehabilitative care. Capturing the totality of outcome following TBI thus often requires the selection of a battery of outcome instruments, although there is currently little guidance outside of recommendations for research to help guide clinicians in the selection of appropriate outcome measures. This chapter outlines the process of selecting appropriate TBI outcome measures and provides clinically relevant information about commonly used measures. A case example highlights the potential uses of various measures.

### Keywords

Outcomes assessment • Outcome measures • Functional outcomes • Participation outcomes • Community integration measures • Rehabilitation outcome measures

N.J. Pastorek, Ph.D., ABPP (⊠) Michael E. DeBakey VA Medical Center, 2002 Holcombe Blvd (RCL 117), Houston, TX 77030, USA e-mail: Nicholas.Pastorek@va.gov

T.L. Veramonti, Ph.D. Mentis Houston, LLP, 9551 Fannin, Houston, TX 77045, USA Department of Physical Medicine & Rehabilitation, Baylor College of Medicine, Houston, TX, USA

Department of Psychology, University of Houston, Houston, TX, USA e-mail: tveramonti@mentisneuro.com

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_5, © Springer Science+Business Media, LLC 2014

### Introduction

Outcome assessment in rehabilitation has gained increasing attention over the last decade. A special issue of the Archives of Physical Medicine and Rehabilitation in October 2011 details the immense amount of resources that have been invested by the federal government in an attempt to establish a set of standards for outcome measurement in rehabilitation medicine. The authors of the introductory article describe how deliberate item development based on a clear vision of the purpose of outcome measurement in rehabilitation, combined with state-of-the-art data analytic strategies, is beginning to produce measures that will meet the needs of researchers and clinicians working in rehabilitation medicine [1]. The continuing emphasis on the development and application of rational outcome measurement in rehabilitation, as demonstrated in these series of articles, is understandable given the serious influence that outcome assessment can exert on patients, clinicians, researchers, and third party payors.

The noteworthy investment of resources dedicated to the development of standardized outcome assessments is necessary given the complexity inherent in conceptualizing and quantifying rehabilitation outcomes, especially outcome following traumatic brain injury (TBI). Following TBI, relevant targets for outcome assessment include an extensive list of signs, symptoms, and/or functional limitations and restrictions. Furthermore, outcome after TBI is a dynamic construct dependent on a host of factors such as severity of injury, time since injury, age, social supports, and availability of rehabilitative care. The context of the outcome assessment (e.g., treatment planning, intervention trial, etc.) also dictates the type of information which will be most pertinent. Lastly, there are potentially several stakeholders of any outcome assessment, each with varying needs and requirements. Clinicians may require outcome data to determine if their intervention has had the intended consequence and to identify further rehabilitation needs of clients served.

In addition, measuring outcomes after treatment is not only a requirement of accrediting bodies (e.g., Commission on Accreditation of Rehabilitation Facilities [CARF]), but affords opportunities to identify the characteristics of those clients who may benefit from a particular treatment, at a particular time, and for evaluating maintenance of gains made in treatment over time. Individuals with a history of TBI, their care providers, and their advocates may use outcome data to better understand the strengths and weakness of the individual (so as to inform treatment needs and approaches) and for the purposes of life care planning, if necessary. Third party payors and researchers may use outcome assessment as a means for objectively quantifying the benefit of individualized or programmatic interventions. Lawmakers, in turn, may use outcome information provided by advocates and researchers to shape public policy affecting those with a history of TBI. The complexities of outcome assessment delineated above are reflected in the diversity of assessment tools that are available and the ongoing development of new tools.

# Construct of Outcome Following TBI

The construct of outcome following TBI is as multifaceted as the seemingly endless number of unique clinical presentations in a population of TBI survivors. Capturing the totality of outcome following TBI thus often requires the selection of a battery of outcome instruments, although there is currently little guidance outside of recommendations for research to help guide clinicians in the selection of appropriate outcome measures [2]. The result has been the use of varying instruments across studies, making it difficult to compare results across studies and/or lack of inclusion of specific items sensitive to a rehabilitation population [1]. The rehabilitation community has, however, largely accepted the broad classification of health as outlined in the World Health Organization's International Classification of Functioning, Disability and Health (ICF) [3].

Within this framework, health-related problems, such as those expected following TBI, are organized under several important themes: impairments in body structures and functions, activity limitations, and participation restrictions. While a full review of the ICF framework is beyond the scope of the chapter, it is worth noting that this organizational scheme is well suited to aid in the conceptualization of outcome following a complex disease or injury such as TBI. For instance, the creators of the ICF intend that the influence of impairments in body structures and functions, activity limitations, and participation restrictions be viewed as multi-directional. This viewpoint is highly consistent with the experience of TBI specialists who have learned to recognize the complex interplay between injury, personal, and environmental factors.

Further complicating selection of an appropriate outcome battery is that severity of impairment, limitations, and restrictions associated with TBI can change drastically over the course of recovery. Of note, recent conceptualizations consider TBI as a "chronic" condition as opposed to a single event, emphasizing the need to capture relative outcomes, including assessment of quality of life, throughout the lifespan (particularly for those individuals with moderate to severe injuries) [4]. While widely used measures such as the Functional Independence Measure (FIM) [5] and the Disability Rating Scale (DRS) [6] may be useful in tracking patients through hospital discharge and initial outpatient therapies, these measures demonstrate significant ceiling effects in persons with less severe injuries and those more severely injured persons who have experienced particularly good recovery [7]. These ceiling effects reflect very real changes in the construct of "recovery" (i.e., how recovery is defined and measured) during the acute, sub-acute, and postacute recovery periods following TBI. For example, items measuring independence in grooming or feeding may capture important change early in recovery, while items focusing more on community reintegration issues, such as return to work or participation in leisure activities, become more pertinent as TBI survivors transition into late recovery. In order to address these limitations,

adjustments have been made to existing measures (e.g., the addition of the Functional Assessment Measures (FAM) to the FIM) and other measures have been developed specifically to assess community reintegration goals (e.g., Community Integration Questionnaire (CIQ) and the Mayo-Portland Adaptability Inventory-4).

Even when appropriate outcome assessment tools are identified, the application of these measures requires careful deliberation, as interpretation of data is not always straightforward. In cases where impaired awareness is a problem, outcome measures that rely largely on clinician and/or significant other report rather than selfreport may be more informative about the patient's overall level of functioning [8–12]. Conversely, absolute reliance on information provided by clinicians or significant others is problematic because this approach is highly dependent on the interviewer's knowledge of the patient's functional abilities, may change as rapport is built between the patient and interviewer, and can be untowardly influenced by extraneous patient characteristics or environmental variables [13, 14]. Developers of outcome measurement tools have attempted to address this problem by designing forms appropriate for use by patients and others, and by exploring the utility of combining and/or comparing ratings from the patient and others [15].

Even when highly reliable data regarding outcome following TBI are available, impairments, limitations, and restrictions identified through outcome assessment may not be solely attributable to the history of TBI. While moderate and severe TBI is frequently associated with neurological signs (e.g., hemiparesis, spasticity, visual field defect) that can be attributed directly to traumatically induced neuropathological changes, the cognitive and emotional symptoms often present after mild through severe TBI are non-specific in nature and share many features with psychiatric disorders. The issue of overlapping symptoms is especially problematic given the high frequency of psychiatric disorders in individuals with a history of TBI. For example, 53.1 % of a sample of 559 civilians hospitalized for complicated mild through severe TBI met criteria for major depressive disorder during the first year post injury [16]. The high prevalence of comorbid posttraumatic stress disorder in service members and civilians with a history of mild TBI is also compelling, with 33–39 % comorbidity in service members and 12–27 % comorbidity in civilians [17]. Carefully parsing the etiology of symptoms after TBI is important to the extent that therapeutic recommendations vary depending on the etiology.

#### Selecting Outcome Measures

Like most injuries and disorders affecting the central nervous system, TBI is associated with a range of impairments in body structures and functions, activity limitations, and participation restrictions. The manifestation of TBI-related problems in any one individual is influenced by a multitude of factors including: injury characteristics (e.g., initial TBI severity, related musculoskeletal injuries, time since injury); preinjury demographic factors (e.g., socioeconomic status, employment status, age, marital status, education level); personal factors (e.g., psychiatric history, overall health status, coping skills); and environmental factors (e.g., support from family and friends, accessibility in the community, access to healthcare). Furthermore, impairments, activity limitations, participations restrictions, and psychological health can exert complex, bi-directional influences [3, 18]. These bi-directional influences can be associated with changes in functioning that are not always easily predictable, but may be nonetheless extremely meaningful to the TBI survivor. For example, hemiparesis in one or more extremities is a common impairment in body function following severe TBI, which can result in problems carrying out activities of daily living and participation in community activities. While hemiparesis may be expected to improve over time, physical inactivity due to an increase in depression with subsequent social isolation months or even years post-injury can lead to an unexpected worsening of the hemiparesis. Due to the highly interactive influences of impairments,

activity limitations, and participation restrictions, targeted interventions may have unintended (or intended) consequences beyond their ostensible purpose. For this reason, the use of multiple outcome measures rather than a single outcome measure is often recommended to capture the totality of the effects of rehabilitation interventions [19]. Fortunately, a wide array of outcome measures are available to document the equally varied types of impairments and limitations that are common following TBI (for example, see *The Center for Outcome Measurement in Brain Injury*. http://www.tbims.org/combi/).

The previously discussed considerations in outcome assessment are perhaps among the most clinically relevant issues, but by no means represent an exhaustive list. Please see Table 1 for a comprehensive list of outcome assessment selection criteria adapted from Hall [20]. Hall identified many critical technical considerations in the selection of outcome measures. Inherent in these considerations for test selection is the tension that necessarily arises when attempting to satisfy each consideration while also meeting time and resource demands. Each researcher or clinician must carefully determine the goal for outcome assessment in their unique context (e.g., programmatic evaluation of a post-acute brain injury rehabilitation facility, assessment of an intervention for a specific cognitive problem) and find a balance between all the considerations listed in Table 1.

The remainder of the chapter will serve as a review of outcome measures commonly used following TBI. We will focus on measures used in the assessment of early and late outcomes from hospital discharge to late follow-up (1 year or greater post-injury). Clinically relevant information about each measure will be included in tables. Practical considerations regarding the use of measures in clinical practice will be discussed. Finally, a case example will be used to highlight the potential uses of various measures.

Case Example: Mr. Smith is a 48 year-old, righthanded, married gentleman who sustained a traumatic brain injury with polytrauma secondary to a motor vehicle accident. At the time of his injury, Mr. Smith was employed full-time as a foreman in
Definition
Extent to which a scale measures what it claims to measure
Degree to which results are replicable
Ability to detect change
Time required for administration
Amount of training and knowledge required by the rater
Frequency of use by other professionals
Time of recovery in which the measure is reasonably applicable and sensitive to change
Ability to administer the measure by phone or internet
Degree to which the scale was thoughtfully developed for the population
Quality of manualized information pertaining to administration procedures, limitations, etc.
Degree to which the instrument is relevant across the lifespan
Degree to which the instrument captures the multidimensional nature of outcome after TBI
Ease with which an instrument can be acquired

 Table 1
 Technical considerations for outcome measure selection

a construction company. Mr. Smith completed college and denied any history of academic difficulties. He reported a history of alcohol dependency, for which he received inpatient and outpatient treatment about 8 years prior to this injury. He was then sober for almost 7 years, but returned to regular, heavy alcohol consumption about 14 months prior to injury. There was also report of remote history of recreational drug use. Regarding his motor vehicle accident, Mr. Smith had positive loss of consciousness at the scene and was transported via EMS to a local level one trauma center, where his Glasgow Coma Scale (GCS) score upon arrival to the emergency room was 7. The initial cranial CT revealed a left frontotemporal subarachnoid hemorrhage, right frontal contusion, and subdural hematoma, without midline shift. Medical workup additionally revealed a positive blood alcohol level. Mr. Smith did not require neurosurgical intervention and there was no report of early seizure activity. He began following commands 7 days post-injury.

Mr. Smith was transferred to an acute rehabilitation hospital about 3 weeks post-injury. Duration of posttraumatic amnesia, based on serial assessment with the Galveston Orientation and Amnesia Test (GOAT), was 1 month. Results of a baseline neuropsychological evaluation at approximately 5 weeks post-injury were notable for impairments in memory, executive functioning, and cognitive and motor processing speed. Behaviorally, Mr. Smith's wife noted that he displayed reduced frustration tolerance since his injury. He also presented with poor awareness of his injury-related deficits and their implications.

Mr. Smith was transferred from the acute rehabilitation setting to a post-acute residential treatment facility at about  $2^{1/2}$  months post-injury, where his treatment program focused on facilitating his ongoing physical recovery as well as improving his awareness and implementing cognitive compensatory strategies. At 4 months postinjury, Mr. Smith transitioned to an outpatient day treatment program, where he worked with a vocational counselor to facilitate his vocational re-integration. Although the combined severity of his persistent cognitive and physical impairments did not allow for his return to his pre-injury position, his vocational counselor worked with Mr. Smith to identify alternative vocational options. Mr. Smith and his wife stayed with family during his post-acute recovery period so that he could attend the outpatient day treatment program, but eventually transitioned back to their home in a rural community.

### **Global Measures of Outcome**

For an overview of practical considerations regarding the use of global measures in clinical practice please refer to Table 2. Global outcome measures such as the Glasgow Outcome Scale (GOS) [21] are broad-based measures of outcome after TBI. As such, these measures play a very limited role in informing individualized treatment planning and may actually be best suited to describe outcomes in groups of cases recovering from severe TBI [22]. There is evidence, however, that ratings on global outcome measures in the first several months post injury are predictive of long-term psychosocial outcomes [23, 24]. These measures are characteristically rapid to administer, typically requiring the rater to briefly synthesize several indicators of impairment, activity, and participation before assigning a categorical rating to the person with TBI. The relative insensitivity to change in early global outcome measures, such as the fivecategory GOS, led to the development of global measures with a greater number of categories (i.e., Extended GOS) [25] and others that were summed across individual items to create an ordinal score (i.e., DRS; [6]). While the items on the DRS still measure a combination of impairment, activity, and participation factors with no attempt to assess the person's perception of their own level of functioning, the breaking down of broadly described categorical outcomes into more plainly delineated items represented an important transition to more circumscribed measures of activity and participation.

Case Example—continued. Mr. Smith's treatment teams in the acute rehabilitation setting and at the post-acute residential facility may have found ratings on global outcome measures, such as GOS and DRS ratings made at the time of discharge from the acute trauma center, helpful as prognostic indictors for long-term outcome. While precise predictions regarding specific long-term activity and participation restrictions would be unreasonable, the treatment staff at these facilities could have used results of initial injury severity characteristics and global outcome indicators to tentatively prepare the person with TBI and their family for the most likely scenarios regarding supervision needs or likelihood of returning to competitive employment [26]. Prognostic information of this sort could be essential in helping the family to prepare for the long-term consequences of a significant TBI (e.g., loss of income, need for supervision, etc.).

# Measures of Disability and Activities of Daily Living

Measures of disability and activities of daily living provide specific information about a range of behaviors considered essential for self-care (See Table 3). Behaviors are typically rated in terms of the patient's level of independence, or by how much assistance and supervision are necessary for the patient to safely and successfully complete common tasks. These basic self-care behaviors typically have been the focus of interventions during the acute and sub-acute recovery periods following a significant brain injury, thus making measures of activities of daily living ideally suited for tracking individual recovery during initial inpatient care. Of note, however, is that as inpatient, hospital-based rehabilitation lengths of stay have decreased, some of these goals are now frequently being addressed in the post-acute phase of recovery [27].

Since the raters must observe and evaluate many different behaviors, measures of disability and activities of daily living typically require longer administration times relative to global outcome measures. The design of these measures (i.e., many items rated individually on Likert-type scales with similar underlying anchors), however, allows for the application of powerful test development techniques such as Item Response Theory. The FIM [5] is one of the most widely used measures of activities of daily living. One important limitation of the FIM is its relative insensitivity to change in the post-acute stages of recovery as the focus of TBI rehabilitation transcends basic ADLS and transitions to maximizing functioning in the community [7]. It should be noted, however, that FIM scores measured during acute recovery are

Instrument (reference)	Content & administration	Relevant clinical issues
Glasgow Outcome Scale (GOS) [21]; Glasgow Outcome Scale-Extended (GOS-E) [22]	The GOS is a measure of global outcome that classifies individuals into 1 of 5 rank-ordered categories: good recovery, moderate disability, severe disability, vegetative state, dead. The GOS requires only a few minutes to rate; ratings can be obtained by medical record review The GOS-E has 8 rank-ordered categories: upper good recovery, lower good recovery, upper moderate disability, lower moderate disability, upper severe disability, lower severe disability, vegetative state, dead. The GOS-E may take 5–15 min to rate; ratings are made via structured interview	<ul> <li>"Gold standard" in neurosurgical outcome studies</li> <li>Quick and easy to complete, categories correspond to those used by "laypersons," making it clinically useful in delivering prognostic information</li> <li>GOS less sensitive to recovery after TBI beyond 6 months post-injury [41]</li> <li>GOS-E addressed limitations of GOS, by adding more specific categories to improve sensitivity and a structured interview to improve reliability; found to be associated with neuropsychological test findings and measures of disability [42]</li> </ul>
Disability Rating Scale (DRS) [6]	The DRS is a measure of global outcome intended to assess general functional changes over the course of recovery after brain injury, from coma to community. The DRS consists of 8 items corresponding to the following areas of functioning: eye opening, verbalization, motor response, level of cognitive ability for feeding, toileting, and grooming, overall level of independence, and employability. Scores range from 0 (no disability) to 29 (extreme vegetative state). The DRS can be rated in-person or via phone interview with the individual or his/ her support network or from retrospective medical record review. Time to rate the DRS depends on knowledge of individual (range <1–15 min)	<ul> <li>Developed in rehabilitation setting; can be used both in inpatient and follow-up evaluation to track recovery over long term</li> <li>Found to be more sensitive than GOS to changes in recovery; however, still insensitive to changes for those at higher range of functioning (i.e., lower end of scale) as well as of subtle but sometimes significant changes made by an individual in a specified time window. Likelihood for ceiling effects increase as time post injury continues</li> <li>DRS scores have been used to predict return to competitive employment after TBI. DRS scores have also been shown to correlate with supervision needs</li> </ul>
Rancho Level of Cognitive Functioning Scale (LCFS) [43]	The LCFS was developed to measure cognitive functioning in individuals emerging from coma to facilitate treatment planning and to assess recovery and outcome. Individuals are categorized into 1 of 8 levels: I no response; II generalized; III localized; IV confused-agitated; V confused; inappropriate; non-agitated; VI confused-appropriate; VII automatic- appropriate; VIII purposeful-appropriate	<ul> <li>Limited sensitivity to subtle changes in recovery. Can be difficult to classify individuals into one category when they manifest characteristics of multiple categories</li> <li>LCFS scores have been shown to be predictive of returning to work and school [44]</li> </ul>
Supervision Rating Scale (SRS) [45]	The SRS is a 1-item instrument that measures amount of supervision a person is receiving from caregivers. Responses are rated on a 13-point ordinal scale and range from 1 (no supervision needs) to 13 (patient in physical restraints). Ratings are optimally based on direct observation but can be extrapolated from information in medical record in certain cases; completion time is brief	<ul> <li>Ratings are based on amount of supervision actually received, not what is judged or predicted to be needed. Ratings reflect level of supervision due to cumulative impact of different (i.e., cognitive, behavioral, physical) symptoms</li> <li>SRS ratings have been shown to be strongly associated with ratings on DRS and GOS. SRS rating also shown to have consistent relationship with independence in self-care and instrumental activities of daily living [45]</li> </ul>

Instrument (reference)	Content & administration	Relevant clinical issues
Functional Independence Measure (FIM) [5]	The FIM is an 18-item ordinal scale consisting of 13 physical independence items (i.e., self-care, sphincter control, transfers, mobility) and 5 cognitive items (i.e., communication and social cognition). Scores on each item range from 1 (total assistance required) to 7 (complete independence). The Uniform Data System for Medical Rehabilitation (UDS) provides training materials and includes standards required for inter-rater reliability. The FIM can be completed in 20–30 min via clinician conference, observations, or telephone interview	<ul> <li>Widely used in acute inpatient rehabilitation setting to assess changes in level of functioning between admission and discharge</li> <li>Correlated with GOS and DRS</li> <li>Insensitive to more subtle changes expected after acute inpatient rehabilitation discharge; ceiling effects at 1 year post-injury [7]</li> <li>Few items emphasizing cognitive, behavioral, and communication, and community functioning—therefore, less relevant for TBI population</li> </ul>
Functional Assessment Measure (FAM) [28]	The 12-item FAM was developed as an adjunct to the FIM, to enhance applicability to those with brain injury, and does not stand alone. Items are also rated on a 7-point ordinal scale. The FAM addresses cognitive, behavioral, communication, and community functioning considerations. The combined 30-item FIM + FAM requires approximately 30 min to complete	<ul> <li>Ceiling effects less problematic when FAM added to the FIM [7]</li> <li>May be a more valid indicator of disability during follow-up assessments and for post-acute rehabilitation settings, given emphasis on community functioning [46]</li> <li>FAM does not contribute beyond FIM in predicting length of rehabilitation admission or costs. FAM also shown to have limited utility beyond FIM in predicting return to work [7, 47]</li> </ul>
Mayo-Portland Adaptability Inventory-4 (MPAI-4) [29]	The MPAI-4 contains 29 items with 3 subscales (Ability Index, Adjustment Index, Participation Index) assessing physical, cognitive, emotional, behavioral, and social sequelae as well as obstacles to community integration that may be encountered after brain injury. The MPAI-4 was designed to assist in: (1) clinical evaluation/ rehabilitation planning during the post-acute period of recovery; (2) evaluating the effectiveness of post-acute rehabilitation programs; and (3) better understanding long-term outcome after acquired brain injury. May be completed by the individual with injury, his/her significant other, and/or treating clinical staff. A manual for the MPAI is available online	<ul> <li>Ratings on MPAI at admission shown to correlate with outpatient rehabilitation outcomes [48]</li> <li>MPAI ratings and time since injury shown to be predictive of job placement after participation in vocational rehabilitation [49]</li> <li>Staff MPAI ratings predictive of vocational and independent living outcome 1 year after completion of outpatient rehabilitation [50]</li> <li>MPAI ratings found to be sensitive to change in studies of rehabilitation effectiveness [48, 50, 51]</li> </ul>

 Table 3 Measures of disability and activities of daily living

predictive of important psychosocial outcomes such as employment [23]. The relatively low ceiling of the FIM was addressed by the development of the FAM [28], an extension of the FIM which includes a greater focus on functioning in the community. While this attempt to raise the ceiling of the FIM by creating additional items relevant to community reintegration was conceptually sound, the FIM+FAM still suffers from significant limitations in monitoring long-term outcome from TBI [7].

The Mayo-Portland Adaptability Inventory-4 (MPAI-4) [29] offers an alternative to earlier measures of activity and was developed specifically to

monitor outcome following TBI. Rather than rating the level of independence in completing various self-care behaviors, the first 21 items of the MPAI-4 require the rater to determine the extent to which various cognitive, emotional, and social problems interfere with activities. The MPAI-4 also includes 8-items comprising a Participation Index [30], on which the rater is asked to determine how much assistance is required for successful completion of various instrumental activities essential for community participation. The Participation Index ostensibly makes the MPAI-4 an ideal measure for tracking a patient's recovery after discharge into the community. Another advantage of the MPAI-4 is the effort to allow for completion of the measure by the clinician, significant other, and the patient [15]. Allowing significant others to rate the survivor of brain injury seems ideal in cases where involvement of the treatment team may be limited after the injury, and the ability to compare self-ratings to clinician and significant other ratings provides the potential of exploring the extent of problems with awareness on self-reporting.

Case Example—continued: As with global outcome indicators, rehabilitation staff at the acute care hospital and residential treatment facility certainly found the FIM and FAM useful in making predictions regarding Mr. Smith's long-term psychosocial outcomes, such as employability. The design of these measures of activities of daily living also makes them ideal for selecting specific intervention targets (i.e., treatment planning) and tracking progress in therapy. In the case of Mr. Smith, the results of the FIM and FAM indicated need for additional physical and cognitive rehabilitation. Furthermore, the rehabilitation staff collected MPAI-4 ratings from the Mr. Smith, his rehabilitation team members, and his wife. Results from the three raters were used to assess and redress issues with awareness.

# Measures of Community Participation

Community participation, as defined by independent living, social and leisure activity, and productivity, is the ultimate goal of persons with a history of TBI and the most important indicator of long-term success for rehabilitation programs [31]. Widely used measures, such as the Craig Handicap Assessment and Reporting Technique (CHART) [32] and the CIQ [33], focus on observable indicators of outcome (e.g., frequency of behaviors) (See Table 4). While measurement of observable behavior should arguably result in a more psychometrically robust measure, it has been suggested that a limitation of these and other (e.g. MPAI-4) participation measures is the lack of method to address individual differences in priorities [31]. More recently developed measures of participation, such as the Participation Objective, Participation Subjective (POPS) [34], allow the person with TBI to share their subjective impressions regarding the importance of each activity (Participation Subjective), in addition to indicating the frequency with which they engage in that activity (Participation Objective). As intended by the design of this measure, the subjective scales are more highly related to measures of well-being than objective scales. The Participation Assessment with Recombined Tools—Objective (PART-0) [35] and Subjective (PART-S) [36] also separate the objective and subjective constructs of participation.

The Mayo-Portland Participation Index (M2PI) [30] is a somewhat unique measure of participation in that it is a subscale of a lengthier measure designed specifically to evaluate the recovery of persons in the post-acute period following acquired brain injury. Other subscales of the Mayo-Portland Adaptability Inventory-4 [29] measure abilities (i.e., impairment in cognitive and physical domains) and adjustment to injury. Of note, not all 8-items of the M2PI are believed to clearly represent concepts of participation as delineated by the ICF. The response options on the M2PI are difficult to categorize as either objective or subjective making it somewhat challenging to compare this measure to other measures of participation.

Case Example—continued. Measures of participation became most important as Mr. Smith began post-acute rehabilitation. At the time of admission to the residential program, community

 Table 4
 Measures of community participation

Instrument (reference)	Content & administration	Relevant clinical issues
Craig Handicap Assessment and Reporting Technique (CHART) [32] Craig Handicap Assessment and Reporting Technique—Short Form (CHART-SF) [52]	The CHART is a 32-item measure (CHART-SF includes 19 items) assessing the degree to which one's disability impacts their societal participation or community integration. Participation in typical social roles is measured in 6 domains: Physical Independence, Mobility, Occupation, Social Integration, Cognitive Independence, and Economic Self-Sufficiency. The CHART was designed to be administered via interview but can be self-rated or completed by proxy. Requires 7 (CHART-SF) to 15 (CHART) minutes to complete	<ul> <li>Initially developed for use with persons with spinal cord injury. Shown to have high test-retest reliability with TBI</li> <li>The CHART is a quantitative assessment of participation measured by asking number of hours spent in various activities, frequency of community engagement, counts of interactions with friends, coworkers, etc.</li> <li>Based on WHO model of impairment, disability, and handicap</li> <li>Can be re-administered at differing intervals post-injury to capture change</li> <li>Although it can be self-administered, an interview is preferred to provide prompts and/or address any definitional questions that may arise</li> <li>Scores can be influenced by pre-injury individual characteristics as well as family support, substance abuse, and awareness of vocational outcomes. There is no means of capturing individual differences in the priorities persons with TBI place on various community reintegration activities [31]</li> </ul>
Community Integration Questionnaire (CIQ) [33]	The CIQ contains 15 items relevant to integration across 3 domains: (1) home and family life (e.g., meal preparation, child care); (2) social activity (e.g., shopping, leisure activity); and (3) educational, vocational or other productive activity. Scores range from 0 to 29, with higher scores indicating greater integration. The CIQ may be completed by the person with TBI or a proxy in about 15 min; data collection can be through self-administered questionnaires or an interview	<ul> <li>The CIQ has been shown to validly differentiate between persons with TBI and non-disabled controls. Also shown to distinguish between individuals living independently and those requiring support or living in an institutional setting [53]</li> <li>Subscale scores have demonstrated sensitivity up to 1 year post-injury. There do not appear to be substantial ceiling or floor effects [54]</li> <li>The CIQ has been utilized to capture change in community integration in individuals participating in post-acute rehabilitation and to assess maintenance of those gains over time [55, 56]</li> <li>The CIQ is an objective measure and thus there is a lack of method to address individual differences in priorities [31]</li> </ul>

#### Table 4 (continued)

Instrument (reference)	Content & administration	Relevant clinical issues
Participation Objective, Participation Subjective (POPS) [34]	The POPS is a self-report measure comprised of 26 items that assesses an individual's level of engagement (i.e., frequency or duration of participation) in household and community activities (objective assessment) as well as their satisfaction with their level of engagement, weighted by their rating of an activity's importance (subjective assessment). The 26 items, or elements of participation, are summed within 5 categories: Domestic Life, Major Life Activities, Transportation; Interpersonal Interactions and Relationships; and Community, Recreational and Civic Life. The POPS requires about 10–20 min to administer	<ul> <li>Different from other community integration measures by considering both societal expectations for activity participation, as well as individual preferences for activities</li> <li>Unique in: (1) focus solely on activities; (2) metric is duration or frequency of activity; (3) all measured activities are intrinsically social, part of household/ occupational functioning or recreational [35]</li> <li>Subscale creation designed to conform with the International Classification of Functioning and Health categorization of activities and participation</li> </ul>
MPAI-4 Participation Index (M2PI) [30]	The M2PI includes the last 8 items of the MPAI-4 (see Table 3) and includes content covering the degree of limitations in the areas of initiation, social contact, leisure/recreation, self-care, independent living/ homemaking, transportation, employment, and financial management	<ul> <li>M2PI index shown to correlate highly with the entire MPAI-4 [30]</li> <li>M2PI correlated with the PART-O [35]</li> </ul>
Participation Assessment with Recombined Tools-Objective (PART-O) [35]	The PART-O is a composite participation measure encompassing 24 items from the CHART, CIQ, and POPS. The focus of item content is on ways people can be productive members of society (e.g., work, school), are socially integrated (e.g., interacting with family, friends, etc.), and are involved in the community (e.g., going shopping, eating out)	<ul> <li>Development motivated by a need to replace a measure of participation in the Traumatic Brain Injury Model Systems National Database</li> <li>The content of items in the PART-O includes aspects of participation identified in the ICF</li> <li>Highly correlated with the FIM (motor and cognitive), SRS, GOS-E, and DRS, with greater functional independence being associated with greater participation in persons with moderate to severe TBI [35]</li> <li>Further psychometric validation necessary (e.g., cross-validation on a new sample, test-retest reliability, subject-proxy agreement)</li> </ul>

integration goals were prioritized with input from Mr. Smith and his family, in the context of understanding potential environmental factors impacting longer-term outcome and community integration. This information was used in the development of his treatment plan and for informing participation in therapeutic outings and community events. Unfortunately, when his functioning exceeded the need for residential services and he was ready to transition to outpatient treatment, Mr. Smith's rural hometown did not offer specialized TBI outpatient services, so he and his wife stayed with family in a larger city where these services were located. With regard to measuring participation, there was concern that Mr. Smith might experience some challenges in his attempts to participate in a community that was not his own and a goal of rehabilitation necessarily involved examining the generalizability or maintenance of participating in activities in a major city to his more rural hometown (where resources and opportunities for participation may be less). Nevertheless, participation is an important goal for persons with TBI in the post-acute phase of recovery. Measures such as the CHART, CIQ, and POPS assisted his treatment team in identifying barriers to participation, in understanding the degree of impact of existing barriers, and tracking his progress through serial administrations of these measures. Aside from the use of these measures for treatment planning and tracking progress, the day treatment program planned to re-administer the participation measures at a defined follow up point (e.g., 1 year post discharge) as a way to evaluate the effectiveness of their services.

# **Environmental Factors**

Whiteneck and Dijkers [37] provide an excellent historical review of the waxing and waning appreciation for the impact of environmental factors on activity and participation. The authors conclude that the influential ICF model now clearly recognizes the influence of environment on health outcomes and offers reasonable guidance in the conceptualization of environmental factors as they relate to health. This renewed focus on environmental factors is clinically meaningful since careful measurement and manipulation of environmental facilitators and barriers can augment more traditional interventions that focus largely on the person with TBI rather than on the broader context within which their recovery is taking place (see Table 5 for practical clinical considerations of common measures). The Craig Inventory of Environmental Factors (CHIEF) is a 25-item inventory assessing the frequency and perceived magnitude of physical and structural barriers, attitudinal and support barriers, barriers to service and assistance, policy

barriers, and barriers at work and school [38]. The measure has been shown to differentiate between persons with and without disability, as well as between various clinical groups, in fairly predictable ways. CHIEF scores have been shown to correlate with overall life satisfaction. A 12-item short form (CHIEF-SF) [39] is also available. Both measures share the same response options, which allow the person with TBI to indicate how often each barrier has been a problem over the last 12 months (i.e., a five-point Likert scale ranging from never to daily) and how much of an impact the barriers have had on their lives (i.e., little problem versus big problem). A briefer measure of environmental barriers is available in the Service Obstacles Scale (SOS) [40], a six-item scale that assesses caregivers' satisfaction with treatment resources, finances as an obstacle to receiving resources, and transportation as an obstacle to receiving resources. Scores on the SOS have been shown to be related to number of unmet needs and quality of life ratings.

Case Example—continued. While any treatment plan would be incomplete without a clear understanding of environmental barriers and facilitators that a person with TBI will face as he reenters the community, the need for a careful assessment of environmental factors that could potentially affect Mr. Smith was especially important. It was reasonable to expect that his rural community would offer relatively few resources for persons with disabilities and that accessibility challenges may be significant. Understanding the environmental barriers and facilitators presented within his home community helped the treatment team to appreciate the true impact of any disability on his daily functioning. This information was absolutely essential in creating an individualized treatment plan with an inherent long-term goal of maximizing participation.

#### Future of Outcome Measurement

While careful assessment of activity limitations and participation restrictions with measures described earlier in this chapter is important for treatment planning and documentation of treat-

Instrument (reference)	Content & administration	Relevant clinical issues
Craig Hospital Inventory of Environmental Factors (CHIEF) [38] Craig Hospital Inventory of Environmental Factors— Short Form (CHIEF-SF) [39]	The CHIEF assesses the degree to which factors in ones' environment affect the person with disability and includes 25 items measuring 5 domains: (1) Attitudes and Support; (2) Services and Assistance; (3) Physical and Structural; (4) Policies; (5) Work and School. Both the frequency (i.e., problem encountered daily, monthly, etc.) and magnitude of impact of identified environmental barriers (i.e., "little" or "big" problem) are quantified. The CHIEF requires between 10 and 15 min to rate; the instrument can be self-administered or administered by interview. Use of proxy data is discouraged. The CHIEF-SF includes 12 items from the original 5 subscales	<ul> <li>Using the CHIEF, persons with disabilities have been shown to encounter more frequent and problematic environmental barriers as compared to non-injured individuals; the impact of barriers has been shown to be associated with the type and severity of disability</li> <li>Greater impact from environmental barriers has been associated with lower levels of participation and life satisfaction after TBI [38]</li> </ul>
The Service Obstacles Scale (SOS) [40]	The SOS is a 6-item scale evaluating individuals with TBI and their caregivers' perceptions of the quality and accessibility of brain injury community services. It has three main components: (1) satisfaction with resources; (2) finances as an obstacle to receiving services; and (3) transportation as an obstacle to receiving services. The SOS can be completed in a few minutes, either by interview or self-administered format	<ul> <li>Lack of community resources and lack of money to pay for resources were the greatest obstacles voiced in a sample of individuals with TBI. Reports of greater obstacles and lesser satisfaction with community resources were also associated with lower ratings of quality of life [40]</li> </ul>

Table 5 Environmental factors

ment efficacy, these measures are not linked by a common metric. Without linking, it is difficult to accurately compare scores on different instruments, which is a significant impediment to tracking the progress of persons recovering from TBI as they transition from inpatient to outpatient care and to those attempting to compare treatment effects as measured by different instruments. In 2002, the National Institutes of Health (NIH) started the "Roadmap" initiative to address the failure of existing measures to capture the selfreported experience of persons with chronic disease [1]. This "Roadmap" emphasized the importance of developing measures of healthrelated quality of life (HRQOL) using patientreported outcomes of physical health, emotional functioning, level of social support, and participation in the community [1]. An outgrowth of the "Roadmap" was the development of the Patient Reported Outcomes Measures Information System (PROMIS) [57]. The goal of PROMIS is to provide a number of well-calibrated item banks that assess a continuum of functioning and can be administered to persons with a variety of health conditions, thus allowing comparison across studies and databases. The method used to calibrate item banks in PROMIS also enables development of computer-adaptive testing (CAT), where the choice of which items to administer depends on response to prior items. This allows for individualization of items administered and a decreased assessment burden, while still maintaining the ability to calculate scores and compare across persons and samples. The PROMIS methodology has been adopted for assessment of HRQOL in persons with neurological disorders. The resulting Neurology Quality of Life (Neuro-QOL) developed items banks for use in persons with stroke, multiple sclerosis, Parkinson's Disease, epilepsy, and amyotrophic lateral sclerosis [58]. In a subsequent effort, Tulsky and colleagues developed the TBI-QOL to assess HRQOL is persons with TBI [59]. Both the Neuro-QOL and the TBI-QOL contain items from PROMIS, but have additional items that are specific to the issues faced by persons with the respective disorders. While the extremely thoughtful approach to the development of these quality of life measures is impressive, they are not intended to capture objective information regarding impairment, activity, and participation in persons with TBI. Efforts are well underway, however, to apply the rigorous methodology used in the development of these HRQOL measures to the creation of standardized cognitive, emotion, motor, and sensation instruments [60].

#### References

- Tulsky, D., Carlozzi, N. E., & Cella, D. (2011). Advances in outcomes measurement in rehabilitation medicine: Current initiatives from the National Institutes of Health and the National Institute on Disability and Rehabilitation Research. Archives of Physical Medicine and Rehabilitation, 92, S1–S6.
- Wilde, E., Whiteneck, G. G., Bogner, J., Bushnik, T., Cifu, D. X., Dikmen, S., et al. (2010). Recommendations for the use of common outcome measures in traumatic brain injury research. *Archives of Physical Medicine and Rehabilitation*, *91*, 1650–1660.
- 3. World Health Organization. (2001). *International classification of functioning, disability and health.* Geneva, Switzerland: World Health Organization.
- Masel, B. E., & DeWitt, D. S. (2010). Traumatic brain injury: A disease process, not an event. *Journal of Neurotrauma*, 27(8), 1529–1540.
- Hamilton, B. B., Granger, C., Sherwin, F. S., Zielezny, M., & Tashman, J. S. (1987). A uniform national data system for medical rehabilitation. In M. Fuher (Ed.), *Rehabilitation outcomes: Analysis and measurement* (pp. 137–147). Baltimore: Brookes.
- Rappaport, M., Hall, K., Hopkins, H. K., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: Coma to community. *Archives of Physical Medicine and Rehabilitation*, 63, 118–123.
- Hall, K. M., Mann, N., High, W., Wright, J., Kreutzer, J., & Wood, D. (1996). Functional measures after traumatic brain injury: Ceiling effects of FIM, FIM + FAM, DRS and CIQ. *Journal of Head Trauma Rehabilitation*, 11(5), 27–39.
- Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired selfawareness after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84, 168–176.
- Leathem, J. M., Murphy, L. J., & Flett, R. A. (1998). Self- and informant-ratings on the Patient Competency Rating Scale in patients with traumatic brain injury.

Journal of Clinical and Experimental Neuropsychology, 20, 694–705.

- Prigatano, G., & Altman, I. W. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 71, 1057–1064.
- Sherer, M., Boake, C., Levin, E., Silver, B. V., Ringholz, G., & High, W. M., Jr. (1998). Characteristics of impaired awareness after traumatic brain injury. *Journal of the International Neuropsychological Society*, 4, 380–387.
- Prigatano, G. (1996). Behavioral limitations TBI patients tend to underestimate: A replication and extension to patients with lateralized cerebral dysfunction. *The Clinical Neuropsychologist*, 10, 191–201.
- Sherer, M., Oden, K., Bergloff, P., Levin, E., & High, W. M., Jr. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *NeuroRehabilitation*, *10*, 25–37.
- Simmond, M., & Fleming, J. (2003). Reliability of the self-awareness of deficits interview for adults with traumatic brain injury. *Brain Injury*, 17, 325–337.
- Malec, J. (2004). Comparability of Mayo-Portland Adaptability Inventory ratings by staff, significant others, and people with acquired brain injury. *Brain Injury*, 18, 563–575.
- Bombardier, C., Fann, J. R., Temkin, N. R., Esselman, P. C., Barber, J., & Dikmen, S. S. (2010). Rates of major depressive disorder and clinical outcomes following traumatic brain injury. *Journal of the American Medical Association*, 303, 1938–1945.
- Carlson, K., Kehle, S. M., Meis, L. A., Greer, N., Macdonald, R., Rutks, I., et al. (2011). Prevalence, assessment, and treatment of mild traumatic brain injury and posttraumatic stress disorder: A systematic review of the evidence. *Journal of Head Trauma Rehabilitation*, 26, 103–115.
- Whyte, J. (2009). Directions in brain injury research: From concept to clinical implementation. *Neuropsychological Rehabilitation*, 19, 807–823.
- Bagiella, E. (2009). Clinical trials in rehabilitation: Single or multiple outcomes? *Archives of Physical Medicine and Rehabilitation*, 90, S17–S21.
- 20. Hall, K. (1999). Functional assessment in traumatic brain injury. In M. Rosenthal, E. Griffith, J. S. Kreutzer, & B. F. A. Pentland (Eds.), *In rehabilitation* of the adult and child with traumatic brain injury. Philadelphia: Davis Company.
- Jennett, B., & Bond, M. (1975). Assessment of outcome after severe brain damage: A practical scale. *Lancet*, 1, 480–484.
- Wilson, J. T., Pettigrew, L., & Teasdale, G. M. (1998). Structured interviews for the Glasgow Outcome Scale and the Extended Glasgow Outcome Scale: Guidelines for their use. *Journal of Neurotrauma*, *15*(8), 573–585.
- Grauwmeijer, E., Heijenbrok-Kal, M. H., Haitsma, I. K., & Ribbers, G. M. (2012). A prospective study on employment outcome 3 years after moderate to severe

TBI. Archives of Physical Medicine and Rehabilitation, 93, 993–999.

- McCauley, S., Hannay, H. J., & Swank, P. R. (2001). Use of the Disability Rating Scale recovery curve as a predictor of psychosocial outcome following closedhead injury. *Journal of the International Neuropsychological Society*, 7, 457–467.
- 25. Teasdale, G. M., Pettigrew, L. E., Wilson, J. T., Murray, G., & Jennett, B. (1998). Analyzing outcome of treatment of severe head injury: A review and update on advancing the use of the Glasgow Outcome Scale. *Journal of Neurotrauma*, 15, 587–597.
- 26. Kothari, S. (2007). Prognosis after severe TBI: A practical, evidence-based approach. In N. Zasler, D. Katz, & R. Zafonte (Eds.), *Brain injury medicine: Principles and practice*. New York: Demos Medical.
- Braunling-McMorrow, D., Dollinger, S. J., Gould, M., Neumann, T., & Heiligenthal, R. (2010). Outcomes of post-acute rehabilitation for persons with brain injury. *Brain Injury*, 247, 928–938.
- Hall, K. M., Hamilton, B., Gordon, W. A., & Zasler, N. D. (1993). Characteristics and comparisons of functional assessment indices: Disability Rating Scale, Functional Independence Measure and Functional Assessment Measure. *Journal of Head Trauma Rehabilitation*, 8, 60–74.
- Malec, J., Kragness, M., Evans, R. W., Finlay, K. L., Kent, A., & Lezak, M. D. (2003). Further psychometric evaluation and revision of the Mayo-Portland Adaptability Inventory in a national sample. *Journal* of Head Trauma Rehabilitation, 18(6), 479–492.
- Malec, J. (2004). The Mayo-Portland Participation Index (M2PI): A brief and psychometrically-sound measure of brain injury outcome. *Archives of Physical Medicine and Rehabilitation*, 85, 1989–1996.
- Sander, A., Clark, A., & Pappadis, M. R. (2010). What is Community Integration Anyway?: Defining meaning following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 25(2), 121–127.
- 32. Whiteneck, G., Charlifue, S. W., Gerhart, K. A., Overhosler, J. D., & Richardson, G. N. (1992). Quantifying handicap: A new measure of long-term rehabilitation outcomes. *Archives of Physical Medicine and Rehabilitation*, 73, 519–526.
- Willer, B., Rosenthal, M., Kreutzer, J. S., Gordon, W. A., & Rempel, R. (1993). Assessment of community integration following rehabilitation for traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8, 75–87.
- 34. Brown, M., Dijkers, M. P. J. M., Gordon, W. A., Ashman, T., Charatz, H., & Cheng, Z. (2004). Participation Objective, Participation Subjective: A measure of participation combining outsider and insider perspectives. *Journal of Head Trauma Rehabilitation*, 19, 459–481.
- 35. Whiteneck, G. G., Dijkers, M., Heinemann, A. W., Bogner, J. A., Bushnik, T., Cicerone, K. D., et al. (2011). Development of the participation assessment with recombined tools-objective for use after

traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 92, 542–551.

- 36. Dijkers, M. P., Cicerone, K., Heinemann, A. W., Brown, M., & Whiteneck, G. G. (2009). PART-S: A new measure of satisfaction with participation. [abstract]. *Archives of Physical Medicine and Rehabilitation*, 90, e39.
- 37. Whiteneck, G., & Dijkers, M. P. (2009). Difficult to measure constructs: Conceptual and methodological issues concerning participation and environmental factors. Archives of Physical Medicine and Rehabilitation, 90(Suppl 1), S22–S35.
- Whiteneck, G., Gerhart, K. A., & Cusick, C. P. (2004). Identifying environmental factors that influence the outcomes of people with traumatic brain injury. *Journal* of Head Trauma Rehabilitation, 19(3), 191–204.
- 39. Ephraim, P., MacKenzie, E. J., Wegener, S. T., Dillingham, T. R., & Pezzin, L. E. (2006). Environmental barriers experienced by amputees: The Craig Hospital Inventory of Environmental Factors–Short Form. Archives of Physical Medicine and Rehabilitation, 87(3), 328–333.
- Kolakowsky-Hayner, S., Kreutzer, J. S., & Miner, D. K. (2000). Validation of the Service Obstacles Scale for the traumatic brain injury population. *NeuroRehabilitation*, 14(3), 151–158.
- 41. Jennett, B., Snoek, J., Bond, M. R., & Brooks, N. (1981). Disability after severe head injury: Observations on the use of the Glasgow Outcome Scale. *Journal of Neurology, Neurosurgery and Psychiatry*, 44, 285–293.
- 42. Levin, H. S., Boake, C., Song, J., Mccauley, S., Contant, C., Diaz-Marchan, P., et al. (2001). Validity and sensitivity to change of the extended Glasgow Outcome Scale in mild to moderate traumatic brain injury. *Journal of Neurotrauma*, 18, 157–584.
- Hagen, C., Malkmus, D., & Durham, P. (1972). Levels of cognitive functioning. Downey, CA: Rancho Los Amigos Hospital.
- Rao, N., & Kilgore, K. M. (1992). Predicting return to work in traumatic brain injury using assessment scales. *Archives of Physical Medicine and Rehabilitation*, 73, 911–916.
- Boake, C. (1996). Supervision Rating Scale: A measure of functional outcome from brain injury. *Archives* of *Physical Medicine and Rehabilitation*, 77, 765–772.
- 46. Marosszeky, J., editor. (1992). UDS and Australia: An international perspective. In *Conference on Progress* in *Medical Rehabilitation: Issues in Measurement*. Buffalo, NY.
- 47. Gurka, J. A., Felmingham, K., Baguley, I. J., Schotte, D. E., Crooks, J., & Marosszeky, J. E. (1999). Utility of the Functional Assessment Measure after discharge from inpatient rehabilitation. *Journal of Head Trauma Rehabilitation*, 14(3), 247–256.
- Malec, J. F., Moessner, A., Kragness, M., & Lezak, M. D. (2000). Refining a measure of brain injury sequelae to predict postacute rehabilitation outcome: Rating

scale analysis of the Mayo-Portland Adaptability Inventory. *Journal of Head Trauma Rehabilitation*, 15, 670–682.

- 49. Malec, J. F., Buffington, A., Moessner, A. M., & Degiorgio, L. (2000). A medical/vocational case coordination system for persons with brain injury: An evaluation of employment outcomes. Archives of Physical Medicine and Rehabilitation, 81, 1007–1015.
- Malec, J. F. (2001). Impact of comprehensive day treatment on societal participation for persons with acquired brain injury. *Archives of Physical Medicine* and Rehabilitation, 82, 885–895.
- Constantinidou, F., Thomas, R., Scharp, V. L., Laske, K. M., Hammerly, M. D., & Guitonde, S. (2005). Effects of categorization training in patients with TBI during postacute rehabilitation: Preliminary findings. *Journal of Head Trauma Rehabilitation*, 20, 143–157.
- Whiteneck, G., Tate, D., & Charlifue, S. (1999). Predicting community reintegration after spinal cord injury from demographic and injury characteristics. *Archives of Physical Medicine and Rehabilitation*, 80, 1485–1491.
- Willer, B., Ottenbacher, K., & Coad, M. L. (1994). The Community Integration Questionnaire: A comparative examination. *American Journal of Physical Medicine and Rehabilitation*, 73, 103–111.
- 54. Sander, A., Fuchs, K. L., High, W. M., Hall, K., Kreutzer, J. S., & Rosenthal, M. (1999). The Community Integration Questionnaire revisited: An assessment of factor structure and validity. *Archives*

of Physical Medicine and Rehabilitation, 80(10), 1303–1308.

- 55. Seale, G., Caroselli, J. S., High, W. M., Becker, C. L., Neese, L. E., & Scheibel, R. (2002). Use of the community integration questionnaire (CIQ) to characterize changes in functioning for individuals with traumatic brain injury who participated in a post-acute rehabilitation programme. *Brain Injury*, 16, 955–967.
- 56. Sander, A., Roebuck, T. M., Struchen, M. S., Sherer, M. S., & High, W. M. (2001). Long-term maintenance of gains obtained in post-acute rehabilitation by persons with traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 16(4), 356–373.
- Amtmann, D., Cook, K. F., Johnson, K. L., & Cella, D. (2011). The PROMIS initiative: Involvement of rehabilitation stakeholders in development and examples of applications in rehabilitation research. *Archives of Physical Medicine and Rehabilitation*, 92, S12–S19.
- Cella, D., Nowinski, C., Peterman, A., Victorson, D., Miller, D., Lai, J. S., et al. (2011). The Neurology Quality of Life measurement initiative. *Archives of Physical Medicine and Rehabilitation*, 92, S28–S36.
- Carlozzi, N. E., Tulsky, D. S., & Kisala, P. A. (2011). Traumatic brain injury patient-reported outcome measure: Identification of health-related quality-oflife issues relevant to individuals with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 92, S52–S60.
- Nowinski, C. J., Victorson, D., Cavazos, J. E., Gershon, R., & Cella, D. (2010). Neuro-QOL and the NIH Toolbox: Implications for epilepsy. *Therapy*, *7*, 533–540.

# Neuroimaging in Traumatic Brain Injury

# Elisabeth A. Wilde, Jill V. Hunter, and Erin D. Bigler

# Abstract

Neuroimaging plays an important role in the initial diagnosis, clinical management, and prognosis of patients with traumatic brain injury (TBI). Specifically, imaging contributes to immediate decisions regarding hospital admission, and early surgical intervention (e.g., hematoma evacuation or ventricular drainage catheter and intracranial pressure monitor placement). In the subacute period, imaging may also be used to monitor lesion or postsurgical change or to detect lesions not immediately evident on initial imaging. In more chronic phases of recovery, imaging may also be used to characterize and monitor late-appearing tissue changes.

This chapter will review current clinical imaging guidelines, briefly review basic information about computed tomography (CT) and magnetic resonance imaging (MRI), discuss the appearance of common forms of TBI-related pathology on conventional imaging, discuss qualitative and quantitative approaches to imaging analysis in TBI, highlight more advanced forms of imaging as they relate to trauma, and review some of the caveats and future directions of imaging in TBI. Since CT and MRI are the most universally available and commonly performed neuroimaging procedures, this chapter will focus primarily on CT and structural MRI as they are currently used in clinical practice.

E.A. Wilde, Ph.D. (🖂)

Departments of Physical Medicine and Rehabilitation, Neurology, and Radiology, Baylor College of Medicine, Houston, TX, USA

Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA e-mail: ewilde@bcm.edu

J.V. Hunter, M.D. Departments of Radiology and Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA Department of Pediatric Radiology, Texas Children's Hospital, Houston, TX, USA

E.D. Bigler, Ph.D., ABPP-Cn Department of Psychology and Neuroscience Center, Brigham Young University, Provo, UT, USA

The Brain Institute and the Department of Psychiatry, University of Utah, Salt Lake City, UT, USA

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_6, © Springer Science+Business Media, LLC 2014

#### **Clinical Imaging Guidelines**

Several clinical practice guidelines have been proposed, generally based upon systematic review of existing current literature followed by peer review of the recommendations generated by an expert panel or working group. Among the most widely used guidelines include those established by the Brain Trauma Foundation [1], the American College of Emergency Physicians (ACEP) [2], and the American College of Radiologists [3] in the United States, and those established by the National Institute for Health and Clinical Excellence [4] and the Scottish Intercollegiate Guidelines Network (2009) [5] in the United Kingdom. Based upon these guidelines, current consensus is that emergent imaging (CT) should generally be performed in patients with a Glasgow Coma Scale (GCS) [6] score less than 13 and should be performed in all cases of severe TBI (GCS=3-8) given that 90 % of patients with severe TBI have been shown to demonstrate abnormalities on CT [1].

There is more variability in guidelines regarding the use of imaging in patients with mild or moderate injury, or in children. Two of the more wellestablished guidelines for mild TBI include the Canadian CT Head Rule (CCHR) [7–9] and the New Orleans Criteria (NOC) [10]. The Canadian Head CT Rule was based upon a prospective study of 3,121 patients with "mild" TBI as assessed by GCS score of 13-15 to determine "high" risk factors (five criteria) which indicate the likelihood that a patient will need neurosurgical intervention as well as "medium" risk factors (two criteria) for predicting the likelihood of findings on CT (see Table 1). The NOC guidelines were based on data from a series of over 1,400 patients and established the sensitivity of seven factors (see Table 2); validity of the scale was subsequently confirmed in a prospective study, where the NOC criteria correctly classified patients who required neurosurgical intervention, and those that had "important" versus "unimportant" brain injuries. Both the CCHR and NOC have been subsequently tested in a consecutive sample of mildly head-injured adults who

#### Table 1 Canadian CT head rule for mild TBI

High risk factors (high risk for necessary neurosurgical intervention)

- Score of less than 15 on the GCS at 2 h post-injury
- · Suspected open or depressed skull fracture
- More than two episodes of vomiting
- Physical evidence of basal skull fracture (e.g., haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from ears or nose, Battle's sign)
- Age >65 years

Medium risk factors (for predicting brain injury on CT)

- Amnesia for events that happened more than 30 min before the impact
- Dangerous mechanism of injury (e.g., pedestrian struck by motor vehicle, fall from higher than 3 ft or 5 stairs)

GCS Glasgow Coma Scale Score

#### Table 2 New Orleans criteria for mild TBI

CT is indicated if any of the following are present:

- Headache
- Vomiting
- Age over 60 years
- · Drug or alcohol intoxication
- · Deficits in short-term memory
- Posttraumatic seizure
- · Evidence of injury above the clavicles

presented to the emergency department (ED) with witnessed loss of consciousness, disorientation or amnesia, and GCS 13-15, and demonstrated equivalently high sensitivities for detecting any traumatic intracranial lesion on CT, clinically important brain injury, and neurosurgical intervention, though the CCHR was shown to be more specific [11], confirming earlier reports of the higher specificity of the CCHR for clinical outcomes [12]. A subsequent study comparing the CCHR and the NOC demonstrated that the CCHR demonstrated higher sensitivity than the NOC, with higher negative predictive power when primary outcome was the need for neurosurgical intervention [13]. A recent systematic review of clinical decision rules for adults with mild TBI including the CCHR, NOC, and other guidelines concluded that the CCHR has consistently shown high sensitivity for identifying injury requiring neurosurgical intervention with an acceptable specificity to allow considered use of cranial computed tomography; it has been widely validated and consistently demonstrated acceptable results [14].

More recently, evidence-based guidelines were proposed by a multidisciplinary panel representing a joint effort between the ACEP and the Centers for Disease Control and Prevention (CDC) which were intended for patients with blunt head trauma who present to the Emergency Department (ED) within 24 h of injury, who have a GCS score of 14 or 15 on initial evaluation in the ED, and are 16 years of age or older [2]. These recommendations include the indication for a noncontrast head CT in patients with loss of consciousness or posttraumatic amnesia if one or more of the following is present: (1) headache, (2) vomiting, (3) age greater than 60 years, (4) drug or alcohol intoxication, (5) deficits in shortterm memory, (6) physical evidence of trauma above the clavicle, (7) posttraumatic seizure, (8) GCS score less than 15, (9) focal neurologic deficit, or (10) coagulopathy (Level A recommendations). Additionally, recommendations include consideration of a noncontrast head CT in head trauma patients with no loss of consciousness or posttraumatic amnesia if there is a focal neurologic deficit, vomiting, severe headache, age 65 years or greater, physical signs of a basilar skull fracture, GCS score less than 15, coagulopathy, or a dangerous mechanism of injury (e.g., ejection from a motor vehicle, pedestrian struck by a vehicle, fall from a height of more than 3 ft or 5 stairs). Other federal agencies including the Veterans Administration and the Department of Defense have also established guidelines for the use of imaging in mild TBI in deployed and nondeployed military settings [15, 16].

Additionally, special consideration exists for use in infants and children. No single set of clinical criteria has been identified to detect radiographic lesions in all pediatric patients. Infants have been reported to develop intracranial hematomas despite "normal" initial CT scanning and examination, and age younger than 2 years has long been recognized as an independent risk factor for significant head injury [17]. Symptoms such as vomiting and seizures may be less specific and sensitive in infants and young children and a significant number may exhibit no initial signs or symptoms of brain injury [18, 19]. Therefore, some have argued that CT should be considered following any nontrivial injury, particularly when there are significant scalp findings such as hematoma [20]. In addition, others have suggested that investigation with CT is advisable in young children since fractures can be missed on magnetic resonance imaging (MRI). With regard to mild TBI, both the CCHR and the NOC have been applied in children [21] as have other clinical decision rules including the Canadian Assessment of Tomography for Childhood Head Injury (CATCH) rule [22, 23], the Children's Head injury Algorithm for the prediction of Important Clinical Events (CHALICE) rule [24], and the pediatric emergency care applied research network (PECARN) rule [25]. A recent review of these and other rules applied in pediatric populations concluded that the PECARN rule appears to be the best validated rule for both children and infants, with the largest study cohort, highest sensitivity, and acceptable specificity for clinically significant intracranial injuries, though further validation of the PECARN rule is necessary [26].

In the acute post-injury interval (>72-96 h), CT (noncontrast) is often the preferred method of imaging in trauma as it can be rapidly and relatively inexpensively performed, is sensitive in detecting fractures, mass effect, and hemorrhages that may require immediate medical attention or surgical intervention, is available 24 h in Trauma I and II centers, and can be performed in more severely injured patients who may be otherwise unable to undergo MRI (e.g., presence of MRIincompatible medical equipment or other contraindications for MRI, patient is unable to remain still for the longer duration of MRI). However, even in cases of acute mild TBI, CT is generally the preferred imaging technique to rule out pathology that may require observation or attention. In the subacute (<72–96 h post-injury) and chronic phases of injury, MRI may be the preferred method of imaging following TBI due to its increased sensitivity to more subtle forms of injury or to forms of injury more detectable in a chronic post-injury interval. As demonstrated in Fig. 1, CT provides gross anatomical information



**Fig. 1** Comparison of computed tomography (CT) and different standard magnetic resonance imaging (MRI) sequences in the same healthy (uninjured) individual. Images are in the axial (transverse) plane and at approximately the same level. Each MRI sequence uniquely

assesses brain parenchyma and cerebrospinal fluid. *T1* T1-weighted MRI, *T2* T2-weighted MRI, *FLAIR* fluid attenuated inversion recovery, *GRE* gradient recalled echo, *PD* proton density

related to the brain and skull; however, MRI sequences are generally superior in visualizing brain anatomy and, in some contexts, underlying neuropathology.

# Sensitivity of CT Versus MRI

From a practical standpoint where imaging is used to impact clinical decision-making in the acute phase of injury, a CT scan is typically considered "positive" in the acute phase of injury if it reveals a traumatic intracranial lesion that requires either intervention or observation including a subdural, epidural, or parenchymal hematoma, subarachnoid hemorrhage, cerebral contusion, or depressed skull fracture. Although MRI demonstrates a clear advantage in the detection of axonal injury, small areas of contusion, and subtle forms of injury, MRI and CT are currently considered equivalent for clinical diagnosis of surgically correctable lesions in the acute setting. However, as will be discussed further, MRI may have a distinct advantage in non-acute imaging studies.

# **Basics of Structural Imaging**

# **CT Basics**

CT utilizes X-ray beam technology and subsequent cross-sectional image reconstruction in the axial or transverse plane. As the X-rays are emitted, tissue absorbs some portion of the X-rays that pass through, and the unabsorbed portion is captured and measured. Sophisticated computer algorithms are then applied to the received X-ray to estimate how much was absorbed within a small volume of tissue. In the context of CT, attenuation refers to the ability to block or absorb x-rays as they pass through a tissue or substance (e.g., bone absorbs most of the X-ray, and air absorbs very little). The amount of attenuation in each kind of tissue is relatively constant and is known as that tissue's attenuation coefficient. In CT, attenuation coefficients are mapped to an arbitrary scale (termed the Hounsfield scale) between -1,000 (air) and +1,000 (bone) in Hounsfield units (HU). In CT, descriptions of notable aspects of the image (abnormalities that are detected) are often described in terms of *density* or attenuation relative to the expected appearance of certain tissues (e.g., "hypodense"/"hypoattenuation" or dark, "isodense"/"iosattenuation" or similar in radiodensity to another or adjacent tissue, "hyperdense"/"hyperattenuation" or bright).

#### **MRI Basics**

MR images represent the intensities of electromagnetic signals from hydrogen nuclei. The MR signal results from resonance interaction between hydrogen nuclei and externally applied magnetic fields which are spatially encoded to provide a mapping of the area imaged. The signal intensity depends on the magnetic environment and the density of the hydrogen nuclei (i.e., protons in water), which have distinct differences between white and gray matter and cerebrospinal fluid (CSF) as demonstrated by the different MRI sequences depicted in Fig. 1. The fundamental principle of clinical MRI is that, when placed in a strong magnetic field, the protons in the body (mostly from hydrogen in water) respond to electromagnetic waves by absorbing and then reradiating these waves in accordance with the magnetic environment of the tissue. Thus, the reradiated waves have a signal strength that characterizes the tissue type. Different tissues are characterized by their signal intensity, appearing brighter or darker on the images as reflected in the standard MRI sequences shown in Fig. 1. In contrast to CT, which reflects "density" in the description of abnormal signal, the word "intensity" is used in description for MRI (e.g., "hyperintense", "isointense", or "hypointense").

The use of innovative methods for varying magnetic field strength, the delays between the sending and receiving of electromagnetic waves, and the acquisition and display of the signal intensity allow the production of a range of images, some of which best demonstrate anatomy while others are more useful in detecting specific forms of pathology. For example, Tl-weighted images tend to show greater anatomical detail, but may not be necessarily sensitive to certain forms of pathology because they are less sensitive to differences in adjacent tissignal. In contrast, scans that sue are T2-weighted generally show normal structures as having an intermediate (gray) intensity, while fluid and many pathologic abnormalities appear with high intensity (white), often providing the kind of contrast between normal and abnormal tissue types which is indicative of certain pathology. Other sequences that provide averages of Tl- and T2-weighting are called balanced or proton density sequences. Common appearances of typical tissues on MRI are listed in Table 3.

Different forms of T2-weighted imaging may be particularly useful for detecting various forms of TBI-related pathology. For example, the fluid attenuated inversion recovery sequence (FLAIR) is particularly sensitive to several trauma-related abnormalities within the white matter reflected as hyperintense (bright) signal (e.g., gliosis). The gradient (recalled) echo (GRE) affords excellent image detail with short imaging times and is sensitive to the presence of blood and a blood breakdown product called hemosiderin, which when present is reflected as a hypointense or dark signal. Detection of hemosiderin in brain parenchyma can also suggest the presence of neurodegerative changes such as gliosis in surrounding tissue. Susceptibility-weighted imaging (SWI) is considered to be sensitive in detecting microhemorrhages, which appear as hypointense (dark) regions.

		T2-weighted	T2-weighted
Tissue	T1-Weighted	GRE	FLAIR
Gray matter	Gray	Light gray	White/light gray
White matter	White	Dark gray	Dark gray
CSF or water	Black	White	Black
Blood	Depends on timing (white-gray)	Black with blooming	Black without blooming
Fat	White	Black	Black
Air	Black	Black	Black
Bone or calcification	Black	Black	Black
Edema (established)	Gray	White	White
Demyelination or gliosis	Gray-black	White	White
Ferritin deposits	Dark gray	Black	Black
Calcium bound to protein	White	Dark gray	Dark gray
Proteinaceous fluid	White	Variable	Variable

 Table 3
 MRI appearance of commonly scanned tissues

*Note*: On fast spin echo (FSE) sequences (a faster variant of the SE sequence), fat appears bright in T2-weighted. Blooming=exaggeration of the lesion

# Common Forms of TBI-Related Pathology Detectable on Imaging

As part of the inter-agency Common Data Elements (CDE) initiative, Haacke et al. [27] define and review 22 forms of TBI-related pathology identifiable on conventional CT and MRI at both acute and chronic post-injury intervals (see also [28]). These include extra-axial (external to the brain parenchyma) blood collections (epidural hematoma (EDH), subdural hematoma, subarachnoid hemorrhage, and intraventricular hemorrhage) as well as intra-axial (within the brain) lesions (contusions, intra-parenchymal hematoma, diffuse/traumatic axonal injury) and other forms of secondary injury (hypoxia, ischemia, edema), and degenerative change (volume loss, encephalomalacia, gliosis). Other functional changes are also detectable with additional applications of MRI. Table 4 summarizes the most common forms of TBI-related pathologies visible on imaging, their causes, and their characteristic appearance on CT and MRI at both acute and chronic post-injury intervals. Fig. 2 illustrates some of these characteristic forms of TBIrelated pathology in cases of severe TBI on day-of-injury CT.

# **Qualitative Imaging Interpretation**

The normal human brain is generally symmetric, with one hemisphere being a mirror copy of the other. While minor asymmetry is commonplace and may be considered normal in certain contexts, prominent asymmetry generally denotes the presence of pathology. Fig. 3 shows an agematched healthy control at the base of the brain showing the inferior frontal and temporal lobes as well as cerebellum at the level of the IV ventricle compared to a patient who sustained a severe TBI. Note the general symmetry between the hemispheres, where one side can be compared to the other, and they generally have a similar appearance in terms of size and shape. The uniformity of the two sides is also reflected in the general symmetry of the cortical ribbon of gray matter and the central appearance of white matter. However, note that in the individual with TBI, a massive loss of parenchyma has occurred in the right temporal lobe (arrow), which is different than the damage seen on the left. An older surface contusion damaged the lateral aspect of the temporal lobe leaving encephalomalacia (distal white arrow), but white matter signal is abnormal (middle arrow in left temporal lobe), along with

Table 4 Appearance of Comm	on Forms of TBI-related Pathology on Im-	ıging		
Pathology	Cause and typical location	Appearance on CT	Appearance on conventional MRI sequences	Detectable on advanced MRI sequences
Blood-related lesions				
Note: Extra axial blood is usual	lly best visualized on a T2-weighted or gra	dient recalled echo (GRE) seque	nce	
Epidural hematoma (EDH)	Collection of blood (between the dura mater and skull) that does not generally extend past the sutures of the skull (though it can cross midline with associated tearing of the dura mater), usually caused by tearing of meningeal artery/vein, diploic vein, or dural venous sinus; typically occurs at the site of impact and is often associated with an overlying skull fracture; most common in the temporal or temporoparietal regions	Classically, a lentiform- shaped blood collection <i>Acute</i> : hyperdense in the majority of cases: sometimes mixed lucency (hyperdense and hypodense features) during active bleeding <i>Subacute</i> : homogeneously isodense to hyperdense as active bleeding stops <i>Chronic</i> : at least partially isodense to hypodense with breakdown and resorption appears on CT as a shallow blood collection that follows the convexity of the skull and can cross suture lines, but do not cross midline	<i>Acute:</i> isointense to minimally hyperintense mass (clot) on T1-weighted imaging (and markedly hypointense on T2-weighted imaging) separated by dura by a thin rim of extruded serum, which is hyperintense on T1- and T2-weighted sequences <i>Subacute:</i> hyperintense on T1-weighted imaging <i>Chronic:</i> will become isointense as blood is reabsorbed	N/A (CT and conventional MR sequences are typically used for detection of EDH)
Subdural hematoma (SDH)	Collection of blood (between the dura mater and the arachnoid membrane) resulting from breakage of bridging vessels between the brain surface and the dura; often crescent-shaped; often occurs at the primary site of traumatic impact or "contre-coup" site; SDH are often associated with deceleration injuries with rupturing of veins via a shearing mechanism; occur most often around frontal and parietal lobes, though they can also occur in the posterior cranial fossa	Acute: often hyperdense but can be isodense (e.g., severe anemia or blood loss, if CSF is mixed with hemorrhage); can also be heterogeneous in some contexts (e.g., active bleeding, coagulopathy, acute-on-chronic hemorrhage) <i>Subacute</i> (1–2 weeks): may be isodense <i>Chronic</i> : (greater that 2 weeks) may be isodense relative to CSF or hypodense if older than 3 weeks	<i>Acute/Subacute:</i> hyperintense, with more intermediate intensity associated with acute hemorrhage; can be hypointense on GRE <i>Chronic:</i> isointense to CSF on T1-weighted images and hypointense on T2-weighted and GRE images. FLAIR imaging may be the most sensitive to chronic SDH	N/A (CT and conventional MR sequences are typically used for detection of SDH)
				(continued)

Pathology	Cause and typical location	Appearance on CT	Appearance on conventional MRI sequences	Detectable on advanced MRI sequences
Subarachnoid hemorrhage (SAH)	Collection of blood in the subarachnoid space that is normally filled with CSF (e.g., cistern, brain convexity), which may result from direct laceration of small cortical vessels, redistribution of IVH flowing from the fourth ventricle or direct extension from cortical contusion/hematoma	<i>Acute:</i> areas of hyperdensity outlining the cerebral sulci, Sylvian fissures or basal cisterns <i>Chronic:</i> N/A; will become isodense as blood is reabsorbed	<i>Acute:</i> can appear isointense on T1 and T2-weighted images, but generally hyperintense on FLAIR <i>Chronic:</i> N/A; will become isointense as blood is reabsorbed	N/A (CT and FLAIR sequences are typically used for detection of SAH)
Intraparenchymal hemorrhage (IPH)	Collection of blood within the cerebral hemisphere; often in the frontal and temporal lobes; may leave hemosiderin deposits as macrophages digest hemoglobin from the blood	Acute: areas of hyperdensity	<i>Acute:</i> hypo- to isointense on T1-weighted imaging, and markedly hypointense on T2-weighted imaging <i>Subacute:</i> hyperintense on T1-weighted imaging and hypointense (dark) on T2-images <i>Chronic:</i> bright (hyperintense) on both T1- and T2-weighted imaging	Susceptibility weighted imaging (SWT) may be used to detect small microhemorrhages in the parenchyma
Intraventricular hemorrhage (IVH)	Blood in the ventricles, which often settles in the occipital horns of the lateral ventricles; IVH is often associated with rotation injury which tears subependymal veins on the ventral surface of the corpus callosum and along the formix	Acute: areas of hyperdensity within the normally dark ventricles	Acute: iso- to hyperintense to CSF on T1-weighted images, and especially hyperintense on FLAIR	N/A (CT and conventional MR sequences are typically used for detection of IVH)
Contusion	Bruising of the brain by contact with irregular inner surfaces of the skull, especially the sphenoid wings and petrous ridges; often occur at the site of impact or at a "contre-coup" location; most often found in the anterior and lateral temporal and frontal areas	<i>Acute:</i> initially hypodense (though can be isodense) patchy and ill-defined lesion, often progressing over time (usually 24–48 h) in size ("blooming") and number <i>Chronic:</i> resolution of hypodense signal	<i>Acute:</i> MRI is sensitive to acute hemorrhagic contusions; blood appears as hyperintense signal on FLAIR, isointense to hypointense on T1-weighted, hyperintense on T2-weighted images, and may be hypointense on gradient echo sequences <i>Chronic:</i> resolution of hypointense signal	Conventional MR sequences are typically used for detection of contusion, though these may be also detected via SWI

 Table 4 (continued)

femosiderin biffuse or traumatic axonal injur Taumatic axonal injury	Deposits from blood products of IPH that remain visible on MRI long after they are no longer visible on CT <i>yy</i> ( <i>TAI</i> ) <i>yy</i> ( <i>TAI</i> ) Shear-strain of the axon, usually	Acute: N/A Chronic: N/A TAI is often difficult to	Acute: N/A Chronic: dark on T2-weighted imaging; best detected by GRE/SWI sequences; will fade over time Acute: nonhemorrhagic lesions	N/A Conventional MR sequences (T2-weighted, FLAIR) are typically used for detection of contusion Small hemorrhagic TAI may
	associated with acceleration/ deceleration injury	identify on CT (most lesions are nonhemorrhagic), but may appear as hyperdense petechial hemorrhages at the gray-white matter junction, usually in cerebral hemispheres, corpus callosum, or brainstem	are best appreciated by FLAIR imaging (hyperintense), and hemorrhagic lesions appear on T2-weighted, GRE and SWI (hypointense); T1-weighted imaging may not detect TAI <i>Chronic</i> : T2-weighted imaging may become hypointense as the hemorrhagic lesion ages due to hemosiderin deposit. GRE imaging may be useful in the chronic interval	become more visible using SWI (appear as areas of hypointensity)
Edema; usually occurring in the Vote: Edema is usually best visue	acute and subacute interval alized on a T2-weighted or GRE sequence			
Vasogenic Edema Extracellular)	Extracellular water collection resulting from disruption of the blood-brain barrier	<i>Acute:</i> loss of sulci, compression of basal cisterns and flattening of the ventricular margins, but gray/white attenuation and differentiation remain intact <i>Chronic:</i> N/A	<i>Acute:</i> hyperintense on T2/ FLAIR, may appear as midline shift/displacement of the ventricle if sufficiently large and causing mass effect <i>Chronic:</i> N/A	Can also be detected by diffusion-weighted imaging
				(continued)

Table 4 (continued)				
Pathology	Cause and typical location	Appearance on CT	Appearance on conventional MRI sequences	Detectable on advanced MRI sequences
Cytotoxic edema (cellular)	Results from sustained intracellular water collection (fluid accumulating within cells); sometimes due to high levels of excitatory amino acids or failure of cell membrane pumps	<i>Acute</i> : appears as loss of gray-white matter differentiation <i>Chronic</i> : N/A	Acute: "slit-like ventricles" and effaced sulci and CSF spaces Chronic: N/A Note that it is generally not possible to differentiate between vasogenic and cytotoxic edema on conventional imaging, though diffusion-weighted imaging can differentiate	Diffusion-weighted imaging may be required to differentiate between vasogenic and cytotoxic edema (restricted in cytotoxic; unrestricted in vasogenic)
Ischemia, hypoxia				
Acute infarction/ischemia	Uncoupling between the metabolic demands of the neurons due to depolarization caused by trauma and hypoperfusion likely due to transiently disturbed neurovascular functioning, can occur due to mechanical compression of a blood vessel or direct vascular injury, dissection, vasospasm, occlusion, or pseudoaneurysm	<i>Acute:</i> Not usually visualized prior to 24 h. After that, areas of low density may be apparent (from days 1–10, with peak around days 2–4 post-injury) <i>Chronic:</i> appears as a pseudo normalization of density changes ("fogging" effect) by 10 days (e.g., areas of low density are no longer visible by 10 days	<i>Acute:</i> T2 hyperintense on T2-weighted imaging, can also appear as hemorrhage or mass effect. On diffusion-weighted imaging (DWI) may be restricted diffusion as early as 20 min post-infarction/ ischemic insult <i>Chronic:</i> as with CT, by 10 days, pseudo normalization occurs, and T2 hyperintensity may no longer be evident; atrophy will eventually be appreciated on any sequence as degenerative processes complete	Appears as areas of restricted diffusion on DWI
Hypoxia	Related to ventilatory or circulatory compromise, including cardiac arrest or chest injury; injury may be particularly visible in basal ganglia, hippocampus, and dentate gyrus	<i>Acute</i> : usually not visible before 24 h <i>Chronic</i> : hypodensity (when visible)	<i>Acute:</i> usually not visible before 24 h <i>Chronic:</i> may not be visible on T1-weighted imaging; hyperintense on T2-weighted imaging; will fade over time	DWI can be helpful; on DWI, may be restricted diffusion as early as 20 min post-hypoxic insult

120

Other degenerative changes (no.	t generally detected in acute or subacute i	nterval)		
Gliosis	Consequence of posttraumatic	Acute: N/A	Acute:	
	proliferation of astrocytes which may cause the formation of a glial scar	Chronic: faint areas of hypodensity (can be difficult to detect on CT)	Chronic: MRI is more sensitive than CT for detection of gliosis, which appears as areas of hyperintensity on T2-weighted imaging	
Encephalomalacia	Alteration of brain tissue resulting from ischemia or inflammation, typically due to vascular insufficiency or degenerative change; may be cystic	<i>Acute:</i> N/A <i>Chronic:</i> Hypodense (dark) areas and absence of effacement or poor sulcal definition	<i>Acute:</i> N/A <i>Chronic:</i> hypointense on T1-weighted imaging and hyperintense on T2-weighted images	
Volume loss	Degenerative changes in white or gray matter which become apparent in the subacute and chronic phases of injury	<i>Acute:</i> N/A <i>Chronic:</i> appears as prominence of ventricles, sulci, and subarachnoid CSF. Volume loss can be detected globally and regionally as early as 2 weeks, though may be progressive volume loss may occur	Acute: N/A Chronic: as with CT, but can be detected more easily and in specific cortical regions and subcortical structures such as the corpus callosum and hippocampus	Recent advances in image acquisition (usually involving 3D T1-weighted or other sequences) and post-processing have made it possible to perform quite detailed volumetric changes in cortical and subcortical areas
Functional changes				
Biochemical or metabolic alteration		Cannot be detected with CT in either the acute or chronic period	Cannot be detected with conventional structural sequences in either the acute or chronic period	Magnetic resonance spectroscopy has been used to examine metabolic or biochemical concentration alteration
Disruption of network connectivity	Structural or functional alteration of known "network" functioning within the brain	Cannot be detected with CT in either the acute or chronic period	Cannot be detected with conventional structural sequences in either the acute or chronic period	Functional connectivity MRI (fcMRI) and DTI have been used to demonstrate TBI-related changes in connectivity; these techniques are undergoing validation



**Fig. 2** (a) Traumatic pathology as visualized on CT in patients with severe TBI (left-hand column) as compared to CT in an uninjured individual at the same level (*right-hand column*). (The *top row* depicts axial CT through the plane of the centra semiovale demonstrating bilateral posterior fronto-parietal fractures (comminuted with fragment on the left as indicated with *arrow*) with

overlying subcutaneous hematomas (*arrow*) and underlying bilateral (lentiform) hemorrhagic collections (*arrow*), likely representing epidural hematomas, with hyperdensity outlining the interhemispheric fissure which may represent evidence for subdural blood (*arrow*). The *middle row* portrays an axial CT slice through the plane of the bodies of the lateral ventricles. There are bilateral subcutaneous



Fig. 2 (continued) hematomas (as indicated with *arrow*) with associated air likely related to laceration. There is a large mixed density extra-axial collection outlining most of the right cerebral hemisphere (likely subdural or SDH as indicated by arrow) causing midline shift and mass effect (see dotted line for projected placement of midline in an uninjured condition) bowed convex to the left. Loss of gray matter-white matter differentiation is also appreciated (arrow). The bottom row depicts axial unenhanced CT through the plane of the bodies of the lateral ventricles. There is loss of normal gray-white matter differentiation (arrow) with curvilinear mixed density collection outlining the right posterior fronto-parietal region (arrow) with mass effect displacing the effaced bodies of the lateral ventricles to the left of midline (termed "midline shift"; dotted line depicts the projected placement of midline in an uninjured state). The appearances may represent evidence for acute bleeding. There is subcutaneous soft tissue swelling overlying the right frontal bone. Consistent with radiologic convention, the right side of the head (marked R) is depicted on the left side of the figure; L left side of the head). Traumatic pathology as visualized on CT in patients with severe TBI (left-hand column) as compared to CT in an uninjured individual at the same level (right-hand column) (continued). Top row depicts

unenhanced CT through the plane of the bodies of the lateral ventricles. There is evidence for intraventricular blood outlining the body of the left lateral ventricle (arrow). In addition there is subdural blood outlining the interhemispheric fissure (arrow) and hyperdensity seen outlining the right-sided cingulate sulcus (arrow). Subcutaneous hematoma is noted within the left infratemporal fossa. (b) The bottom row depicts axial CT acquired without contrast at the level of the bodies of the lateral ventricles. There is evidence of bifrontal edema in association with hemorrhagic contusion/shear injury (depicted with arrow) seen in association with shallow subdural hematomas overlying the right fronto-temporal lobes. There is mild midline shift (dotted line depicts the projected placement of midline in an uninjured state) bowed convex to the left seen in association with effacement (partial collapse of the ventricle so it is difficult to distinctly visualize; arrow) of the right greater than left body of the lateral ventricle. Subdural blood is noted within the anterior greater than posterior aspect of the interhemispheric fissure. Subcutaneous hematomas are noted within the right infratemporal fossa and overlying the left parietal bone. Consistent with radiologic convention, the right side of the head (marked R) is depicted on the left side of the figure; L left side of the head)



**Fig.3** This patient (*right*) sustained a severe TBI in a fall where bilateral temporal lobe contusions occurred, with the one on the right associated with temporal bone skull fracture and neurosurgical treatment that included a partial temporal lobectomy. An age-matched control is included at the left at a similar level for comparison of the expected appearance of white and gray matter as well as the appearance of the frontal and temporal lobes and the cerebellum. Note the symmetry of the right and left hemispheres in the uninjured individual. Comparison of the severe TBI patient on the right with the control on the left enables identification of the loss of parenchyma on the right hemisphere due to surface contusion (*arrow*). The

temporal horn dilation (medial arrow). Although having the comparison subject facilitates detecting trauma-related abnormalities, the types of pathologies seen in the TBI patient on the left can be readily discerned by understanding the basic principles of similarity and symmetry in image interpretation.

One of the most commonly examined features in a chronic phase of injury is the dilation of the ventricular system, a condition referred to as ventriculomegaly [29]. Slight asymmetry of the lateral ventricles may be normal, but when significant asymmetry exists, the more asymmetric side of the ventricle typically reflects the hemisphere and region with greater volume loss. The appearance of the brain overall as well as specific regions of interest (ROI) should likewise be consistent with standards considered to be within normal limits. Thus, two principles apply to viewing any brain image, readily

left temporal lobe is also damaged, and simple comparison of the left with the right temporal lobes in this injured individual highlights the differences (lack of symmetry) between hemispheres. The most distal *arrow* on the left points to an old surface contusion that has resulted in focal atrophic changes (encephalomalacia) with the upward point *arrow* pointing toward degraded white matter and the most medial *arrows* pointing to the temporal horn of the lateral ventricles which are dilated in comparison to the control. Consistent with radiologic convention, the right side of the head (marked *R*) is depicted on the left side of the figure; *L* left side of the head)

observed in Fig. 1-brain typical development at any age appears symmetric and similar in appearance to a known healthy standard. To demonstrate this point, Fig. 4 shows a teenager who sustained a severe TBI with penetration injury to the frontal lobe. The scan is approximately 2 years post-injury and shows several asymmetries. This time frame post-injury is long past any gross inflammatory reaction that could displace or misshape the ventricle and classic midline indicators so the right-ward extension of the ventricle (images are not in radiological convention since three-dimensional (3D) imaging is being shown) is a result of what is referred to as hydrocephalus ex vacuo. In other words, as brain tissue degenerates, that damaged tissue loses volume and the ventricle passively expands to fill the void. The asymmetry may be best appreciated in the T2-weighted image depicting the anterior horn and the dorsal 3D views of



**Fig. 4** The *top row* depicts single slice MRIs of a teenager with focal frontal pathology on the right hemisphere (*arrow* in the T1-weighted image). Note how the anterior horn of the lateral ventricle is prominently asymmetric with the head of the right anterior horn most distinctly pointing to the degradation of white matter in the frontal lobe. The right frontal lobe white matter damage and ventricular asymmetry may be visualized in each sequence which highlights different aspects of white and gray matter degeneration. The upward pointing *arrow* on the far right of the DTI image shows the asymmetry of the ventricle and also points toward the reduced integrity of white matter at the level of the forceps minor, which results in the decreased brightness of the colors which represent fiber directionality, particularly the green color in the frontal white matter representing white matter fibers coursing in an anterior-posterior direction. The *bottom row* shows a three-dimensional view of the ventricular asymmetry (ventricles in aqua color) superimposed upon a 3D surface image of the brain (*lower left*). The middle image depicts the ventricle asymmetry but also includes regional damage, where red color depicts localized gray matter loss/cortical encephalomalacia, flesh-tone reflects white matter focal damage, and the yellow reflects areas of residual hemosiderin deposition from prior hemorrhage. The lateral view (*right*) with corpus callosum DTI tractography overlaid demonstrates substantial loss of the frontal projections of the corpus callosum (*arrow*) which is consistent with the presence of the focal pathology

the entire ventricular system. Note that the symmetric distension of the right anterior horn points to where all of the white matter pathological changes have occurred. This is particularly evident in the diffusion tensor imaging (DTI) scan in the upper right which shows that the white matter pathology extends throughout the frontal white matter into the forceps minor region of the corpus callosum (lower arrow).

# **Classification Schemes for CT**

There are a limited number of CT classification systems for use with acute TBI. These classification tools are based on the presence of certain trauma-related abnormalities (qualitatively determined) on the admission head CT that were determined to have the greatest prognostic

Classification/Grade	Definition
Diffuse injury I	No visible intracranial pathological changes seen on CT scan
Diffuse injury II	Cisterns are present with midline shift of 0–5 mm and/ or lesions densities present; no high or mixed density lesion >25 cm <sup>3</sup> ; may include bone fragments and foreign bodies
Diffuse injury III (swelling)	Cisterns compressed or absent with midline shift of 0–55 mm; no high or mixed density lesion >25 cm <sup>3</sup>
Diffuse injury IV (midline shift)	Midline shift >5 mm; no high or mixed density lesion >25 cm <sup>3</sup>
Evacuated mass lesion	Any lesion surgically evacuated
Non-evacuated mass lesion	High or mixed density lesion >25 cm <sup>3</sup> ; not surgically evacuated

Table 5 Marshall classification

<i>Note</i> : In the Marshall Classification, likelihood of mortal-
ity is calculated for Grades 1-4 of Diffuse Injury as fol-
lows: Grade 1 (normal CT)=9.6 %, Grade II (cisterns
present, midline shift <5 mm)=13.5 %, Grade III (cisterns
compressed or absent, midline shift <5 mm)=34 %;
Grade IV (midline shift >5 mm) = 56.2 $\%$

significance (using 6-month mortality as the outcome measure) among a larger set of CT features associated with TBI. The most widely used is the Marshall Classification system [30], which is based upon findings in the Trauma Coma Data Bank (TCDB) in severely injured patients. The classification consists of six categorical descriptions of patients with TBI (see Table 5). The Marshall system has been used for descriptive purposes, injury severity classification, as well as prediction of mortality and risk for increased intracranial pressure. Modified versions of the Marshall Classification system have also been proposed and utilized (see Table 6), which take into account the number and location of lesions. The Marshall classification has several recognized limitations, including difficulties in assigning a classification to patients with multiple injury types, difficulties associated with the lack of standardization of certain features on CT scan, its limited sensitivity in mild and moderate

 Table 6
 Rotterdam CT classification scheme

Predictor	Score
Basal cisterns	
Normal	0
Compressed	1
Absent	2
Midline shift	
No shift or shift ≤5 mm	0
Shift >5 mm	1
Epidural mass lesion	
Present	0
Absent	1
Intraventricular or subarachnoid hemorrhag	e
Absent	0
Present	1
Sum of scores for each predictor above	
	+1
Final score	

The number corresponding to the best description of each predictor is circled, and the scores are summed across all predictors. The final score is the sum of the rating for each predictor +1

The final score predicts 6-month mortality as follows: 1=0 % mortality; 2=7 % mortality; 3=16 % mortality; 4=26 % mortality; 5=53 % mortality; 6=61 % mortality

TBI, and limited ability to predict later functional recovery [31]. An additional scale called the Morris-Marshall scale, based on traumatic sub-arachnoid hemorrhage (tSAH), has been used as tSAH has been shown to be a strong predictor of outcome and mortiality [32–34].

The Rotterdam Classification Scheme [35] was developed more recently and also uses CT-based findings to predict outcome. It incorporates several of the features involved in the Marshall system as well as the addition of the presence of EDH, since the presence of EDH has been associated with better clinical prognosis than other intracranial abnormalities such as subdural or intraparenchymal hematoma. The Rotterdam CT classification system has been successfully used as a prognostic factor in patients undergoing decompressive craniectomy [36] and is included in the international guidelines for clinical management and prognosis of TBI [37], but still requires additional validation [31].

Recent analyses attempting to determine parameters which may be the most predictive of

unfavorable outcome suggest that the presence and magnitude of midline shift may be among the most important variables, surpassing even the Marshall CT score when used as a continuous variable [38]. There is also a significant degree of colinearity between midline shift and hematoma volume. Finally, the addition of a score of traumatic subarachnoid/intraventricular blood may increase the predictive accuracy of the scale. The addition of CT parameters has been found to add 6–10 % additional estimated explained variance in the presence of the important clinical variables of age, GCS score, and pupillary response, and CT results have been an important factor in prognostic models [39–41].

# **Quantitative Imaging Analysis**

Quantitative image analysis may also contribute substantially to past and future efforts to link imaging and outcome following TBI. The first quantitative CT measurements of pathology were linear measurements, including the measures of the extent of midline displacement, width of a hematoma at its apex, etc., and these are still utilized. SDH or EDH may be measured as the linear distance from the inner table of the skull to the widest indent into brain parenchyma. Alternatively, certain kinds of lesions (e.g., hemorrhage) can be measured by identifying the boundaries of the hemorrhage on all scan slices where it can be identified to determine a surface area (or estimation of volume by multiplying the total surface area summed across slices by slice thickness). Midline shift is typically defined by connecting the frontal notch with the straight line of the posterior segment of the interhemispheric fissure and the occipital notch and measurement of the deviation of the septum pallucidum (a membrane typically present between the two lateral ventricles best seen at the level of the anterior horns, particularly evident in the axial plane) is displaced from its presumed original position.

Another quantitative measurement is the calculation of the ventricle-to-brain ratio or VBR, which can be done with CT or MRI. CSF within the ventricle is pressurized and produced at a steady rate, which creates an internal pressure gradient within the ventricle. In the typical, healthy brain this is very important because the brain is a soft organ that would collapse inwardly if it were not for the internal pressure gradient created by the CSF. Pathology that results in neural tissue loss disturbs the CSF to brain parenchymal balance where parenchymal volume loss is replaced by the passive expansion of the ventricle. By calculating either a single slice or whole brain VBR, this measure quantifies the expansion of the ventricle, which indirectly reflects a loss of brain volume.

Despite the utility and ease of some of these measures, medical imaging techniques for quantitative CT and especially MRI analysis now permit additional methods that measure volume, surface area, cortical thickness, gyrification patterns, shape, and contour of any region of interest (ROI). While there are certainly other tissues present, imaging quantification is typically restricted to whole brain or regional gray matter, white matter, and CSF volume and/or morphology.

In MRI, the T1-weighted image is the most commonly used sequence for quantification, and differences in pixel intensity on a gray scale form the basis for "segmentation" of the brain image into tissue types (white and gray matter) and CSF. For example, Fig. 5 demonstrates the initial T1-weighted image (Fig. 5a) followed by automated segmentation where gray matter is now colorized as red, white matter as white, and CSF as black (see Fig. 5b). Next, either via manual-tracing or the use of highly automated software, 'classification' or identification of different structures and ROIs occurs according to the gray matter, white matter, and CSF boundaries (see Fig. 5c) of each structure or region. Once classified, the volume, thickness, area, shape, or a variety of other readily quantifiable measures may be derived. The most common quantitative MRI analysis metric is regional volume [42], which has relevance for predicting outcome, such as the relation between hippocampal volume in the chronic post-injury interval and memory outcome [43, 44].

Another quantitative neuroimaging technique referred to as voxel-based morphometry (VBM) [45] uses digital MRI data that are realigned and



**Fig. 5** This illustration depicts the process of volumetric analysis, using the native T1-weighted image (**a**), segmenting it into gray matter (colorized as *red*), white matter (shown in *white*) and CSF (shown as *black*) (**b**), and then classifying the segmented image into specific structures (**c**). For example, the thalamus is *dark green*, the hip-

pocampus is *yellow*, the amygdala is *light blue*, etc. By classifying the different regions of interest (ROI), additional information can be derived including cortical surface area and volume, cortical thickness measurement, and shape and contour analyses



**Fig. 6** Three-dimensional rendering of a patient with severe brain injury, with pink color depicting relatively intact brain tissue, dark blue depicting the ventricular system, light blue depicting abnormal FLAIR white matter intensities, red representing areas of encephalomalacia,

yellow depicting areas of gliosis, and violet depicting foci of hemosiderin deposition (**a**) see also (**b**) for axial twodimensional image). (**c**) An axial T1-weighted MRI image, and (**d**) is a T2-weighted image, (**e**) FLAIR image, and (**f**) is a susceptibility-weighted image

co-registered (e.g., rotating and rescaling data and transforming it into a uniform 3D space with a common coordinate system so that all brains fit within the same space), allowing for direct comparison across the group despite individual variation in head size, shape, and structure. Each pixel is then classified as being either white matter, gray matter, or CSF; then, determining the relative concentration of different pixel types within a specified voxel (3D pixel) allows the computation of "voxel-by-voxel" white, gray, and CSF comparisons. Volume changes in pixel density as determined by the VBM technique can objectively identify specific areas of difference in a patient or a group of patients compared to a reference sample.

Using other conventional MR sequences such as FLAIR or GRE, a quantitative approach can be applied to determine white matter hyperintensity or hemorrhagic lesion burden, respectively [46–48]. For example, presence of hemosiderin has been considered a reflection of shearing forces within the brain and a proxy measure for diffuse axonal injury (DAI; see [49, 50]). The total of hemosiderin-identified lesions can be summed to calculate an overall lesion burden due to parenchymal hemorrhage. Increasing detection of the presence of prior hemorrhage resulting from TBI has been shown to reflect greater tissue damage and worse outcome both in children and adults [51–53].

#### Advanced MRI

Advanced MR techniques are rapidly gaining favor in studies of TBI due to their potential ability to better detect more subtle forms of injury and improve diagnosis and prognosis (especially in mild TBI), monitor structural and functional changes (both advantageous and deleterious) that occur over time and may not be as evident on conventional imaging, elucidate different injury mechanisms in TBI (e.g., presence and time course of cytotoxic edema or progressive neural degeneration), identify patients that may be most able to benefit from certain treatments or evaluate the efficacy of interventions, and better understand how connectivity is disrupted in the brain following injury. Detailed discussion of advanced MR techniques is beyond the scope of this chapter, but a few of the more widely used advanced modalities will be briefly highlighted. Other excellent resources are available which focus on the use of emerging advanced imaging techniques and their application in TBI [54-56].

#### **Diffusion Tensor Imaging**

DTI represents another method to probe white matter integrity [57] based on principles of water diffusion that can be empirically measured. Using DTI tractography, impressive disruptions in white matter and tract continuity can be graphically plotted (see [58]). While DTI methods have tremendous potential to provide insight into the relation of white matter integrity to outcome following brain injury (see [59]), the methods are rapidly evolving and require further validation and study. Global white matter integrity as determined by DTI may ultimately turn out to be one of the better predictors of outcome (see [60]) and several studies have also demonstrated some specificity in brain-behavior relations.

#### Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy (MRS) is another sensitive, noninvasive method of examining brain metabolites in the acutely injured brain and tracking changes over time [61]. Briefly, MRS uses magnetic properties of certain nuclei within molecules to determine information about the structure, dynamics, reaction state, and chemical environment properties of those molecules. MRS utilizes a continuous band of radio wave frequencies to excite atoms in many chemical compounds. These compounds then absorb and emit radio energy at characteristic frequencies, or spectra, which can be used to identify them.

There has been great interest in the use of MRS to examine metabolite alteration following TBI, particularly in tissue where there is no visible injury on conventional imaging [62, 63]. MRS has also shown potential for providing early prognostic information regarding clinical outcome in patients with both accidental and non-accidental TBI [63–71]. However, it should be noted that the use of MRS in rehabilitation outcome is still an emerging area of clinical research [72].

# Resting State fMRI and Connectivity Analysis

Considerable progress has been made to utilize and integrate functional neuroimaging, especially functional magnetic resonance imaging (fMRI), with identified structural brain pathology, particularly as it relates to clinical outcome (see [73]). Resting state fMRI (rs-fMRI) provides a tool which enables examination of a harmonic of the blood oxygen-dependent level (BOLD) MRI signal under the assumption that connected regions would exhibit the same harmonic at rest [74]. This provides the framework to establish functional connectivity (fc) maps from rs-fMRI [75].

# **Multimodal Imaging**

Integrating different MRI techniques that bring together structure and function, as well as the integration of electrophysiological and magnetoencephalographic findings with MRI, show great promise in further defining underlying brain pathology and utilizing this information to enhance clinical outcome [76]. However, the standardization and clinical implementation of these advanced forms of MRI remains a complex issue and additional work is required before these modalities can be utilized on a widespread scale.

# Caveats and Limitations of Imaging in TBI

Because of the multiplicity of both structural and functional abnormalities that may accompany an injury, the presence of lesions alone may not necessarily be predictive of outcome or directly relate to aspects of cognition or behavior that are conventionally ascribed to that brain region (see [77–79]). One possible exception is brainstem pathology, where neuroimaging-identified brainstem abnormalities have been shown to carry a poor prognosis for outcome (see [80, 81]).

Future neuroimaging studies will probably be best utilized with consideration for the totality of pathology identified, rather than focusing on a particular region of interest or quantitative measure. The case shown in Fig. 4 is a case of a severe TBI and illustrates the complexities inherent in selecting a single type of lesion to predict outcome. Inspection of the different MR sequences demonstrates the sensitivity of each to particular lesion types, but relative insensitivity to other types. Another complicated aspect of quantitative neuroimaging involves accuracy in the measurement of lesions. For example, when detecting hemosiderin, GRE and SWI sequences over-represent the true size of the lesion. Alternately, gliosis may not be visible on T1-weighted imaging. Volume loss can be evident qualitatively when one scan is compared with an earlier scan or can be quantitatively estimated based upon comparison with the other (less affected) hemisphere, or by comparison to intracranial volume as a proxy for premorbid brain volume, but it is difficult to accurately measure an absent entity. Finally, even in seemingly more straightforward measurements such as those of blood collections, there may be variability and inconsistency as to how measurements are derived and how much of the tissue abnormality is assessed and ascribed to a particular form of pathology. For example, intraparenchymal hemorrhages can be quantified in terms of hemorrhagic volume, but detectable edema is often associated with the formation of a clot, both within the core of where the hemorrhage has displaced brain parenchyma as well as on its outer margin. Measurement could include the hemorrhage only, or, alternatively, a measurement of hemorrhage that also includes surrounding edema and changes reflective of abnormal brain parenchyma.

In the example below, color-coding each type of abnormality identified by each sequence illustrates that no one abnormality captures the totality of pathology. For example, as impressive as the frontal encephalomalacia is in the T1-weighted image, the amount of CSF signal is underestimated in the T1-weighted compared to the T2-weighted sequence, and neither sequence detects the location of most of the small traumatic hemorrhages as demonstrated in the GRE sequence. The 3D rendering of the brain has been



**Fig. 7** Sequential imaging over time in an adult male with severe TBI. (a) Day-of-injury (DOI) admission CT when first evaluated in the emergency room. Note the downward *arrow* points to a large epidural hematoma that is causing midline displacement and evident shift in the ventricular system. (b) Immediately after the epidural was

removed, major subdural (*top arrow*) and intraparenchymal hemorrhaging (*bottom arrow*) occurred on the opposite side. By 6-weeks post-injury (c), generalized volume loss is evident, with ventricular enlargement and focal encephalomalacia where the intraparenchymal hemorrhaging and neurosurgical debridement occurred

colorized to show that no one lesion type captures all pathology present in this TBI and that measurement of any *one* lesion type underestimates the overall lesion burden induced by this TBI.

Finally, newer imaging techniques have demonstrated that the structural and functional integrity of neural networks [82] may be affected by lesions in brain regions that are seemingly remote and, in cases of mild TBI, from the lack of any lesion visible on conventional imaging. White matter lesions may adversely affect frontal lobe functions, regardless of the actual location of one or more focal lesions because of disrupted connectivity to frontal regions critical to behavioral expression [83]. To date, many neuroimaging variables in TBI have shown only modest relationships to outcome, likely due to these complexities (see [78]). Strangman and colleagues [43] have demonstrated that prediction of memory rehabilitation outcome is improved if both global indicators of pathological changes in the brain are combined with specific quantitative changes in target ROIs like the hippocampus known to be critical to the cognitive function being assessed (see also [44]). The time at which lesions are measured may affect their presence and/or volume, since some types of lesions are more evident immediately after injury, others within a subacute period, and others develop only after days to

months. Certain forms of "lesions" may evolve (e.g., change in size or shape or appearance), dissipate, or stabilize with time and depending on the imaging modality that is used. Therefore, the type of scan performed, its sensitivity for detecting certain abnormalities, and post-injury interval become critical variables in assessing the complex relation between neuroimaging findings and outcome. An example of this is highlighted in Fig. 7, images from a patient who sustained a severe TBI as the result of an assault. On the initial day of injury (DOI) CT scan, a very large right temporoparietal epidural hematoma (see arrow in Fig. 7a) was evident, in addition to midline shift. There is loss of definition of the cortical sulci and graywhite matter differentiation, indicators of generalized cerebral edema as well as the influence of a mass lesion. There is also subdural and/or subarachnoid hemorrhage on the left, but it is less perceptible on the DOI CT, likely because the right-sided epidural hematoma and increased intracranial pressure displace the brain to the left, and the epidural and generalized edema compressed these other hemorrhagic lesions and kept them in check, until the patient underwent a surgical evacuation of the EDH (see Fig. 7b). Once the right EDH was evacuated, the intraparenchymal lesion pattern and types of pathology change as shown in Fig. 7b, and evolve over time (Fig. 7c).

# Standardization of Neuroimaging Acquisition Protocols

Recent attempts have been made to better standardize MRI acquisition protocols in the field of TBI. In the military clinical sector, standardization may promote cost-saving by ensuring that patients undergo a sufficiently detailed exam tailored to TBI to reduce the need for additional scans later. Additionally, standardization may facilitate direct comparison of imaging results over time in patients that require monitoring of intracranial pathology. Defense and Veterans Brain Injury Center (DVBIC) has recently established guidelines related to the recommended "standard of care" clinical imaging protocols and parameters for use in mild TBI (http://www.dvbic.org/audiences/ resources/3). In the research arena, standardization facilitates research by promoting comparability between data collected across different sites and contribution to large datasets which are not feasible or cost-prohibitive to collect at a single site. These larger datasets may also be helpful in better establishing normative data. Such an example is the federal interagency CDE initiative (Haake et al. 2010; Duhaime et al. 2010). The CDE has published recommended sequence parameters for TBI-related research as well as variables which can be collected from standard CT and MRI imaging. Additionally, the American College of Radiology (ACR) and the Institute of Medicine (IOM) have also initiated efforts to examine how greater consistency in imaging parameters across sites may be achieved, particularly with regard to advanced sequences such as DTI.

# **Future of Neuroimaging in TBI**

Justification for neuroimaging must fill a clinical need by providing information necessary for diagnosis, monitoring, treatment, enhanced understanding of the patient's condition, and/or prognosis. The typical neuroradiological report is often a summary note to the referring clinician, typically with a concluding statement about whether "abnormalities" are present, and their location, size, and nature. These general radiological impressions are used by clinicians to form a global understanding of the extent of brain damage; however, such clinical impressions are often imprecise, do not address specific neural systems of most interest to the rehabilitation clinician or neurologist, and may not capture the totality of structural or functional alterations resulting from injury.

Newer methods of image analysis are now increasingly automated, sensitive, and precise. Once validated and properly normed, these quantitative findings may eventually be integrated into clinical reports to determine the presence of abnormalities which are not visible or subtle on conventional CT and MRI sequences, assess how abnormalities may impact not only tissue which is lesioned but also larger neural networks, better understand and monitor tissue change over time, more completely anticipate cognitive and functional domains which are expected to create challenges for a patient, tailor rehabilitation strategies, predict response to treatment, and evaluate the efficacy of new and existing rehabilitation interventions [73, 84-86].

# Conclusion

The application of neuroimaging techniques has moved beyond simple classification schemes that define medically important abnormalities requiring neurosurgical intervention to comprehensive assessment metrics that detect various types of pathologies relevant to outcome, in particular how the network that underlies all of cognition and behavior functions. As neuroimaging techniques become better refined, more and more subtle detection of pathological changes relevant to neurobehavioral and neurocognitive outcome have been established. These advanced neuroimaging methods are beginning to provide information that guides and directs rehabilitation.

Acknowledgments We gratefully acknowledge the assistance of Tracy Abildskov for assistance in creation of the figures and Joann Petrie, Ph.D. for assistance in manuscript preparation. Support from the Michael E. DeBakey Veterans Affairs Medical Center Traumatic Brain Injury Center of Excellence is gratefully acknowledged.

# References

- 1. Brain Trauma Foundation IaAAoNS. (2007). Guidelines for the management of severe traumatic brain injury (3rd ed.). New York: Brain Trauma Foundation.
- Jagoda, A. S., Bazarian, J. J., Bruns, J. J., Jr., Cantrill, S. V., Gean, A. D., Howard, P. K., et al. (2008). Clinical policy: neuroimaging and decisionmaking in adult mild traumatic brain injury in the acute setting. *Annals of Emergency Medicine*, 52(6), 714–48; Epub 2008/11/26.
- National Guideline Council. (2009). ACR Appropriateness Criteria<sup>®</sup> head trauma. Rockville MD: Agency for Healthcare Research and Quality (AHRQ). Retrieved 11 Jan 2013 from http://www. guideline.gov/content.aspx?id=37919&search=acr+c t+head+trauma.
- Excellence NIfHaC. (2007). *Triage assessment, investigation and early management of head injury in infants, children and adults* (NICE Clinical Guideline 56). London: National Institute for Health and Clinical Excellence.
- National Guideline Council. (2009). Early management of patients with a head injury. A National Clinical guideline. Rockville, MD: Agency for Healthcare Research and Quality (AHRQ). Retrieved 11 Jan 2013 from http://www.guideline.gov/content.aspx?id=15207&search=ct+head+trauma.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81–4; Epub 1974/07/13.
- Stiell, I. G., Lesiuk, H., Wells, G. A., Coyle, D., McKnight, R. D., Brison, R., et al. (2001). Canadian CT head rule study for patients with minor head injury: methodology for phase II (validation and economic analysis). *Annals of Emergency Medicine*, 38(3), 317–22; Epub 2001/08/29.
- Stiell, I. G., Lesiuk, H., Wells, G. A., McKnight, R. D., Brison, R., Clement, C., et al. (2001). The Canadian CT Head Rule Study for patients with minor head injury: rationale, objectives, and methodology for phase I (derivation). *Annals of Emergency Medicine*, 38(2), 160–9; Epub 2001/07/27.
- Stiell, I. G., Wells, G. A., Vandemheen, K., Clement, C., Lesiuk, H., Laupacis, A., et al. (2001). The Canadian CT Head Rule for patients with minor head injury. *Lancet*, 357(9266), 1391–6; Epub 2001/05/18.
- Haydel, M. J., Preston, C. A., Mills, T. J., Luber, S., Blaudeau, E., & DeBlieux, P. M. (2000). Indications for computed tomography in patients with minor head injury. *The New England Journal of Medicine*, 343(2), 100–5; Epub 2000/07/13.
- 11. Papa, L., Stiell, I. G., Clement, C. M., Pawlowicz, A., Wolfram, A., Braga, C., et al. (2012). Performance of the Canadian CT Head Rule and the New Orleans Criteria for predicting any traumatic intracranial injury on computed tomography in a United States

Level I trauma center. *Academic Emergency Medicine*, *19*(1), 2–10; Epub 2012/01/19.

- Stiell, I. G., Clement, C. M., Rowe, B. H., Schull, M. J., Brison, R., Cass, D., et al. (2005). Comparison of the Canadian CT Head Rule and the New Orleans Criteria in patients with minor head injury. *JAMA, the Journal of the American Medical Association, 294*(12), 1511–8; Epub 2005/09/29.
- Bouida, W., Marghli, S., Souissi, S., Ksibi, H., Methammem, M., Haguiga, H., et al. (2013). Prediction value of the Canadian CT head rule and the New Orleans criteria for positive head CT scan and acute neurosurgical procedures in minor head trauma: a multicenter external validation study. *Annals of Emergency Medicine*, 61(5), 521–7; Epub 2012/08/28.
- Harnan, S. E., Pickering, A., Pandor, A., & Goodacre, S. W. (2011). Clinical decision rules for adults with minor head injury: a systematic review. *The Journal* of *Trauma*, 71(1), 245–51; Epub 2011/08/06.
- Marshall, K. R., Holland, S. L., Meyer, K. S., Martin, E. M., Wilmore, M., & Grimes, J. B. (2012). Mild traumatic brain injury screening, diagnosis, and treatment. *Military Medicine*, 177(8 Suppl), 67–75; Epub 2012/09/08.
- Management of Concussion/mTBI Working Group. (2009). VA/DoD clinical practice guideline for management of concussion/mild traumatic brain injury. *Journal of Rehabilitation Research and Development*, 46(6), CP1–68.
- Greenes, D. S., & Schutzman, S. A. (1998). Occult intracranial injury in infants. *Annals of Emergency Medicine*, 32(6), 680–6; Epub 1998/12/02.
- Schutzman, S. A., Barnes, P., Duhaime, A. C., Greenes, D., Homer, C., Jaffe, D., et al. (2001). Evaluation and management of children younger than two years old with apparently minor head trauma: proposed guidelines. *Pediatrics*, 107(5), 983–93; Epub 2001/05/23.
- Schutzman, S. A., & Greenes, D. S. (2001). Pediatric minor head trauma. *Annals of Emergency Medicine*, 37(1), 65–74; Epub 2001/01/06.
- Glauser, J. (2004). Head injury: which patients need imaging? Which test is best? *Cleveland Clinic Journal* of Medicine, 71(4), 353–7; Epub 2004/05/01.
- 21. Schachar, J. L., Zampolin, R. L., Miller, T. S., Farinhas, J. M., Freeman, K., & Taragin, B. H. (2011). External validation of the New Orleans Criteria (NOC), the Canadian CT Head Rule (CCHR) and the National Emergency X-Radiography Utilization Study II (NEXUS II) for CT scanning in pediatric patients with minor head injury in a non-trauma center. *Pediatric Radiology*, 41(8), 971–9; Epub 2011/04/06.
- Gerdung, C., Dowling, S., & Lang, E. (2012). Review of the CATCH Study: A Clinical decision rule for the use of computed tomography in children with minor head injury. *CJEM*, 14(4), 243–7; Epub 2012/07/21.
- Osmond, M. H., Klassen, T. P., Wells, G. A., Correll, R., Jarvis, A., Joubert, G., et al. (2010). CATCH:

A clinical decision rule for the use of computed tomography in children with minor head injury. *CMAJ: Canadian Medical Association Journal, 182*(4), 341–8; Epub 2010/02/10.

- 24. Dunning, J., Daly, J. P., Lomas, J. P., Lecky, F., Batchelor, J., & Mackway-Jones, K. (2006). Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. *Archives of Disease in Childhood*, 91(11), 885–91; Epub 2006/10/24.
- Kuppermann, N., Holmes, J. F., Dayan, P. S., Hoyle, J. D., Jr., Atabaki, S. M., Holubkov, R., et al. (2009). Identification of children at very low risk of clinicallyimportant brain injuries after head trauma: A prospective cohort study. *The Lancet*, *374*(9696), 1160–70.
- 26. Pickering, A., Harnan, S., Fitzgerald, P., Pandor, A., & Goodacre, S. (2011). Clinical decision rules for children with minor head injury: A systematic review. *Archives of Disease in Childhood*, 96(5), 414–21; Epub 2011/02/12.
- Haacke, E. M., Duhaime, A. C., Gean, A. D., Riedy, G., Wintermark, M., Mukherjee, P., et al. (2010). Common data elements in radiologic imaging of traumatic brain injury. *Journal of Magnetic Resonance Imaging*, 32(3), 516–43; Epub 2010/09/04.
- Duhaime, A. C., Gean, A. D., Haacke, E. M., Hicks, R., Wintermark, M., Mukherjee, P., et al. (2010). Common data elements in radiologic imaging of traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 91(11), 1661–6; Epub 2010/11/04.
- Bigler, E. D., & Maxwell, W. L. (2011). Neuroimaging and neuropathology of TBI. *NeuroRehabilitation*, 28(2), 63–74; Epub 2011/03/31.
- Marshall, L. F., Eisenberg, H. M., Jane, J. A., Luerssen, T. G., Marmarou, A., & Faulkes, A. (1991). A new classification of head injury based on computerized tomography. *Journal of Neurosurgery*, 75, 14–20.
- Saatman, K. E., Duhaime, A. C., Bullock, R., Maas, A. I., Valadka, A., & Manley, G. T. (2008). Classification of traumatic brain injury for targeted therapies. *Journal of Neurotrauma*, 25(7), 719–38; Epub 2008/07/17.
- 32. Eisenberg, H. M., Gary, H. E., Jr., Aldrich, E. F., Saydjari, C., Turner, B., Foulkes, M. A., et al. (1990). Initial CT findings in 753 patients with severe head injury. A report from the NIH Traumatic Coma Data Bank. *Journal of Neurosurgery*, 73(5), 688–98; Epub 1990/11/01.
- 33. Ono, J., Yamaura, A., Kubota, M., Okimura, Y., & Isobe, K. (2001). Outcome prediction in severe head injury: Analyses of clinical prognostic factors. *Journal of Clinical Neuroscience: Official Journal of the Neurosurgical Society of Australasia*, 8(2), 120–3; Epub 2001/08/04.
- 34. Servadei, F., Murray, G. D., Teasdale, G. M., Dearden, M., Iannotti, F., Lapierre, F., et al. (2002). Traumatic subarachnoid hemorrhage: Demographic and clinical study of 750 patients from the European brain injury

consortium survey of head injuries. *Neurosurgery*, *50*(2), 261–7; discussion 7–9; Epub 2002/02/15.

- 35. Maas, A. I., Hukkelhoven, C. W., Marshall, L. F., & Steyerberg, E. W. (2005). Prediction of outcome in traumatic brain injury with computed tomographic characteristics: A comparison between the computed tomographic classification and combinations of computed tomographic predictors. *Neurosurgery*, 57(6), 1173–82; discussion -82; Epub 2005/12/07.
- Huang, Y. H., Deng, Y. H., Lee, T. C., & Chen, W. F. (2012). Rotterdam computed tomography score as a prognosticator in head-injured patients undergoing decompressive craniectomy. *Neurosurgery*, 71(1), 80–5; Epub 2012/03/03.
- Chesnut, R. M. (2000). Evolving models of neurotrauma critical care: An analysis and call to action. *Clinical Neurosurgery*, 46, 185–95; Epub 2000/08/17.
- Nelson, D. W., Nystrom, H., MacCallum, R. M., Thornquist, B., Lilja, A., Bellander, B. M., et al. (2010). Extended analysis of early computed tomography scans of traumatic brain injured patients and relations to outcome. *Journal of Neurotrauma*, 27(1), 51–64; Epub 2009/08/25.
- 39. Hukkelhoven, C. W., Steyerberg, E. W., Habbema, J. D., Farace, E., Marmarou, A., Murray, G. D., et al. (2005). Predicting outcome after traumatic brain injury: Development and validation of a prognostic score based on admission characteristics. *Journal of Neurotrauma*, 22(10), 1025–39; Epub 2005/10/22.
- 40. Jacobs, B., Beems, T., van der Vliet, T. M., van Vugt, A. B., Hoedemaekers, C., Horn, J., et al. (2013). Outcome prediction in moderate and severe traumatic brain injury: A focus on computed tomography variables. *Neurocritical Care*, 19(1), 79–89; Epub 2012/11/10.
- Narayan, R. K., Maas, A. I., Servadei, F., Skolnick, B. E., Tillinger, M. N., & Marshall, L. F. (2008). Progression of traumatic intracerebral hemorrhage: A prospective observational study. *Journal of Neurotrauma*, 25(6), 629–39; Epub 2008/05/22.
- Bigler, E. D., Abildskov, T. J., Wilde, E. A., McCauley, S. R., Li, X., Merkley, T. L., et al. (2010). Diffuse damage in pediatric traumatic brain injury: A comparison of automated versus operator-controlled quantification methods. *NeuroImage*, 50(3), 1017–26; Epub 2010/01/12.
- 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; Epub 2010/11/05.
- 44. 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; Epub 2011/02/04.
- Takao, H., Abe, O., & Ohtomo, K. (2010). Computational analysis of cerebral cortex. *Neuroradiology*, 52(8), 691–8; Epub 2010/05/19.
- 46. Lee, S. H., Lee, S. T., Kim, B. J., Park, H. K., Kim, C. K., Jung, K. H., et al. (2011). Dynamic temporal change of cerebral microbleeds: Long-term follow-up MRI study. *PLoS One*, 6(10), e25930; Epub 2011/10/25.
- 47. Colbert, C. A., Holshouser, B. A., Aaen, G. S., Sheridan, C., Oyoyo, U., Kido, D., et al. (2010). Value of cerebral microhemorrhages detected with susceptibility-weighted MR Imaging for prediction of long-term outcome in children with nonaccidental trauma. *Radiology*, 256(3), 898–905; Epub 2010/08/20.
- Robinson, R. J., & Bhuta, S. (2011). Susceptibilityweighted imaging of the brain: Current utility and potential applications. *Journal of Neuroimaging*, 21(4), e189–204; Epub 2011/02/02.
- 49. Luccichenti, G., Giugni, E., Peran, P., Cherubini, A., Barba, C., Bivona, U., et al. (2010). 3 Tesla is twice as sensitive as 1.5 Tesla magnetic resonance imaging in the assessment of diffuse axonal injury in traumatic brain injury patients. *Functional Neurology*, 25(2), 109–14.
- 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. *AJNR. American Journal of Neuroradiology*, 24(6), 1049–56; Epub 2003/06/19.
- Ashwal, S., Babikian, T., Gardner-Nichols, J., Freier, M. C., Tong, K. A., & Holshouser, B. A. (2006). Susceptibility-weighted imaging and proton magnetic resonance spectroscopy in assessment of outcome after pediatric traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 87(12 Suppl 2), S50–8; Epub 2006/12/05.
- 52. Rivara, F. P., Koepsell, T. D., Wang, J., Temkin, N., Dorsch, A., Vavilala, M. S., et al. (2011). Disability 3, 12, and 24 months after traumatic brain injury among children and adolescents. *Pediatrics*, *128*(5), e1129– 38; Epub 2011/10/26.
- 53. Tong, K. A., Ashwal, S., Obenaus, A., Nickerson, J. P., Kido, D., & Haacke, E. M. (2008). Susceptibilityweighted MR imaging: A review of clinical applications in children. *AJNR. American Journal of Neuroradiology*, 29(1), 9–17; Epub 2007/10/11.
- 54. Van Boven, R. W., Harrington, G. S., Hackney, D. B., Ebel, A., Gauger, G., Bremner, J. D., et al. (2009). Advances in neuroimaging of traumatic brain injury and posttraumatic stress disorder. *Journal of Rehabilitation Research and Development*, 46(6), 717–57; Epub 2010/01/28.
- 55. Shenton, M. E., Hamoda, H. M., Schneiderman, J. S., Bouix, S., Pasternak, O., Rathi, Y., et al. (2012). A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging and Behavior*, 6(2), 137–92; Epub 2012/03/23.
- Hunter, J. V., Wilde, E. A., Tong, K. A., & Holshouser, B. A. (2012). Emerging imaging tools for use with traumatic brain injury research. *Journal of Neurotrauma*, 29(4), 654–71; Epub 2011/07/27.

- Niogi, S. N., & Mukherjee, P. (2010). Diffusion tensor imaging of mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 25(4), 241–55; Epub 2010/07/09.
- Gasparetto, E. L., Rueda Lopes, F. C., & Domingues, R. C. (2011). Diffusion imaging in traumatic brain injury. *Neuroimaging Clinics of North America*, 21(1), 115–25, viii; Epub 2011/04/12.
- Jang, S. H. (2011). A review of diffusion tensor imaging studies on motor recovery mechanisms in stroke patients. *NeuroRehabilitation*, 28(4), 345–52; Epub 2011/07/05.
- 60. Benson, R. R., Meda, S. A., Vasudevan, S., Kou, Z., Govindarajan, K. A., Hanks, R. A., et al. (2007). Global white matter analysis of diffusion tensor images is predictive of injury severity in traumatic brain injury. *Journal of Neurotrauma*, 24(3), 446–59; Epub 2007/04/04.
- Kubal, W. S. (2012). Updated imaging of traumatic brain injury. *Radiologic Clinics of North America*, 50(1), 15–41; Epub 2011/11/22.
- 62. Garnett, M. R., Blamire, A. M., Corkill, R. G., Cadoux-Hudson, T. A., Rajagopalan, B., & Styles, P. (2000). Early proton magnetic resonance spectroscopy in normal-appearing brain correlates with outcome in patients following traumatic brain injury. *Brain: A Journal of Neurology, 123*(Pt 10), 2046–54; Epub 2000/09/27.
- Holshouser, B. A., Tong, K. A., & Ashwal, S. (2005). Proton MR spectroscopic imaging depicts diffuse axonal injury in children with traumatic brain injury. *American Journal of Neuroradiology*, 26(5), 1276–85; Epub 2005/05/14.
- 64. Aaen, G. S., Holshouser, B. A., Sheridan, C., Colbert, C., McKenney, M., Kido, D., et al. (2010). Magnetic resonance spectroscopy predicts outcomes for children with nonaccidental trauma. *Pediatrics*, 125(2), 295–303; Epub 2010/02/04.
- Ashwal, S., Holshouser, B. A., Shu, S. K., Simmons, P. L., Perkin, R. M., Tomasi, L. G., et al. (2000). Predictive value of proton magnetic resonance spectroscopy in pediatric closed head injury. *Pediatric Neurology*, 23(2), 114–25; Epub 2000/10/06.
- 66. 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), 801–11; Epub 2006/08/31.
- 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), 104–14; Epub 2003/04/18.
- Holshouser, B. A., Ashwal, S., Luh, G. Y., Shu, S., Kahlon, S., Auld, K. L., et al. (1997). Proton MR spectroscopy after acute central nervous system injury: Outcome prediction in neonates, infants, and children. *Radiology*, 202(2), 487–96; Epub 1997/02/01.
- Hunter, J. V., Thornton, R. J., Wang, Z. J., Levin, H. S., Roberson, G., Brooks, W. M., et al. (2005). Late proton MR spectroscopy in children after traumatic

brain injury: Correlation with cognitive outcomes. *American Journal of Neuroradiology*, 26(3), 482–8; Epub 2005/03/12.

- Makoroff, K. L., Cecil, K. M., Care, M., & Ball, W. S., Jr. (2005). Elevated lactate as an early marker of brain injury in inflicted traumatic brain injury. *Pediatric Radiology*, 35(7), 668–76; Epub 2005/04/15.
- Yeo, R. A., Phillips, J. P., Jung, R. E., Brown, A. J., Campbell, R. C., & Brooks, W. M. (2006). Magnetic resonance spectroscopy detects brain injury and predicts cognitive functioning in children with brain injuries. *Journal of Neurotrauma*, 23(10), 1427–35; Epub 2006/10/06.
- Ciumas, C., Montavont, A., & Ryvlin, P. (2008). Magnetic resonance imaging in clinical trials. *Current Opinion in Neurology*, 21(4), 431–6; Epub 2008/07/09.
- 73. Wang, L. E., Tittgemeyer, M., Imperati, D., Diekhoff, S., Ameli, M., Fink, G. R., et al. (2012). Degeneration of corpus callosum and recovery of motor function after stroke: A multimodal magnetic resonance imaging study. *Human Brain Mapping*, *33*(12), 2941–56; Oct 22 [Epub ahead of print]. Epub 2011/10/25.
- 74. Zhang, S., & Li, C. S. (2011). Functional connectivity mapping of the human precuneus by resting state fMRI. *NeuroImage*, 59(4), 3548–62; Epub 2011/11/26.
- Vogel, A. C., Power, J. D., Petersen, S. E., & Schlaggar, B. L. (2010). Development of the brain's functional network architecture. *Neuropsychology Review*, 20(4), 362–75; Epub 2010/10/27.
- 76. Stroman, P. W., Bosma, R. L., Kornelsen, J., Lawrence-Dewar, J., Wheeler-Kingshott, C., Cadotte, D., et al. (2012). Advanced MR imaging techniques and characterization of residual anatomy. *Clinical Neurology and Neurosurgery*, 114(5), 460–470; Epub 2012/02/14.
- 77. Groswasser, Z., Reider, G., II, Schwab, K., Ommaya, A. K., Pridgen, A., Brown, H. R., et al. (2002). Quantitative imaging in late TBI. Part II: Cognition and work after closed and penetrating head injury: A report of the Vietnam head injury study. *Brain Injury*, *16*(8), 681–90; Epub 2002/08/09.
- Sherer, M., Stouter, J., Hart, T., Nakase-Richardson, R., Olivier, J., Manning, E., et al. (2006). Computed

tomography findings and early cognitive outcome after traumatic brain injury. *Brain Injury*, 20(10), 997–1005; Epub 2006/10/19.

- Yuh, E. L., Cooper, S. R., Ferguson, A. R., & Manley, G. T. (2011). Quantitative CT improves outcome prediction in acute traumatic brain injury. *Journal of Neurotrauma*, 29(5), 735–46; Epub 2011/10/06.
- 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 and Rehabilitation*, 85(10), 793–806; Epub 2006/09/26.
- Schiff, N. D. (2006). Multimodal neuroimaging approaches to disorders of consciousness. *The Journal* of Head Trauma Rehabilitation, 21(5), 388–97; Epub 2006/09/20.
- Caeyenberghs, K., Leemans, A., Heitger, M. H., Leunissen, I., Dhollander, T., Sunaert, S., et al. (2012). Graph analysis of functional brain networks for cognitive control of action in traumatic brain injury. *Brain*, *135*(Pt 4), 1293–307; Epub 2012/03/20.
- Reed, B. R., Eberling, J. L., Mungas, D., Weiner, M., Kramer, J. H., & Jagust, W. J. (2004). Effects of white matter lesions and lacunes on cortical function. *Archives of Neurology*, 61(10), 1545–50; Epub 2004/10/13.
- 84. Gauthier, L. V., Taub, E., Mark, V. W., Barghi, A., & Uswatte, G. (2012). Atrophy of spared gray matter tissue predicts poorer motor recovery and rehabilitation response in chronic stroke. *Stroke*, 43(2), 453–7; Epub 2011/11/19.
- Kononen, M., Tarkka, I. M., Niskanen, E., Pihlajamaki, M., Mervaala, E., Pitkanen, K., et al. (2012). Functional MRI and motor behavioral changes obtained with constraint-induced movement therapy in chronic stroke. *European Journal of Neurology*, *19*(4), 578–586; Epub 2011/11/02.
- Lotze, M., Beutling, W., Loibl, M., Domin, M., Platz, T., Schminke, U., et al. (2012). Contralesional motor cortex activation depends on ipsilesional corticospinal tract integrity in well-recovered subcortical stroke patients. *Neurorehabilitation and Neural Repair*, 26(6), 594–603; Epub 2011/12/06.

Part III

Intervention

## A Systematic and Evidence-Based Approach to Clinical Management of Patients with Disorders of Consciousness

Joseph T. Giacino, Christopher G. Carter, Carrie Charney, Denise Ambrosi, Matthew J. Doiron, Seth Herman, and Timothy Young

#### Abstract

Disorders of consciousness include coma, the vegetative state, the minimally conscious state and the post-traumatic confusional state. These conditions exist along a two-dimensional continuum comprised of arousal (i.e., wakefulness) and awareness (i.e., recognition of self and environment). Accurately characterizing and distinguishing these disorders early after onset is critically important as diagnosis is closely linked to prognosis and drives clinical decision-making. Unfortunately, published rates of misdiagnosed consistently approach 40 % with most of the error accounted for by failure to detect consciousness when it is preserved. Misdiagnosis may limit access to medical and rehabilitation services and lead to premature withdrawal of life-sustaining care. In this chapter, we describe a systematic, evidence-based framework for clinical management of patients with DoC. The primary aim is to demonstrate how a standardized, multi-tiered approach to assessment organized around a structured "care map" can be instituted in

J.T. Giacino, Ph.D. (🖂)

Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA 02115, USA

Department of Physical Medicine and Rehabilitation, Massachusetts General Hospital, Boston, MA, USA

MGH Institute for Healthcare Professionals, Boston, MA, USA e-mail: jgiacino@partners.org

C.G. Carter, Psy.D. Spaulding Rehabilitation Hospital, 300 First Avenue, Charlestown, MA 02129, USA

Harvard Medical School, Boston, MA 02115, USA

MGH Institute for Healthcare Professionals, Boston, MA, USA

C. Charney, M.S., CCC-SLP

D. Ambrosi, M.S., CCC-SLP • M.J. Doiron, B.A. Spaulding Rehabilitation Hospital, 300 First Avenue, Charlestown, MA 02129, USA

S. Herman, M.D. • T. Young, M.D. Spaulding Rehabilitation Hospital, 300 First Avenue, Charlestown, MA 02129, USA

Harvard Medical School, Boston, MA 02115, USA

Spaulding Rehabilitation Hospital, 300 First Avenue, Charlestown, MA 02129, USA

the rehabilitation setting to inform diagnostic, prognostic and treatment decisions, ultimately improving the consistency and effectiveness of care.

#### Keywords

Vegetative state • Minimally conscious state • Rehabilitation • Neuropsychology • Assessment • Outcome measure

There are approximately 18,000 new cases of severe traumatic brain injury (TBI) annually in the civilian population of the United States [1] and 200-300 more cases per year in active-duty military personnel. Many of those who survive severe TBI experience prolonged disorders of consciousness (DoC). In coma, the eyes remain continuously closed even when vigorous stimulation is applied, indicating that the arousal system is "offline" [2]. The failure to achieve a wakeful state eliminates any possibility of self or environmental awareness. This condition is self-limited and resolves when spontaneous eve-opening reemerges, almost always within 4 weeks of injury. The vegetative state (VS) is distinguished from coma by the reemergence of sleep-wake cycles (signaled clinically by spontaneous or elicited eye-opening); however, there are still no behavioral signs of self and environmental awareness [3]. VS is considered permanent after 12 months following TBI and after 3 months following non-traumatic causes [4]. In most cases, VS evolves into the minimally conscious state (MCS). MCS is characterized by the presence of at least one clearly recognizable behavioral sign of consciousness [5]. The diagnosis of MCS requires reproducible evidence of command-following, discernible yes-no responses, intelligible verbalization, or movements and affective behaviors provoked by relevant environmental stimuli that cannot be accounted for by reflexive activity. Some examples include manual object manipulation, visual tracking, and situation-specific emotional responses (e.g., smiling or crying in the presence of a family member). To meet existing diagnostic criteria for MCS, supportive behavioral evidence must be clearly discernible and reproducible on bedside examination. Emergence from MCS is established when there is clear evidence of reliable communication through verbal or gestural yes-no responses, or recovery of functional object use [5]. Following emergence from MCS, the next point along the continuum of recovery of consciousness is the posttraumatic confusional state (PTCS). PTCS is marked by temporal and spatial disorientation, distractibility, anterograde amnesia, impaired judgment, perceptual disturbance, restlessness, sleep disorder, and emotional lability [6]. During PTCS, 24-h supervision and assistance are required to ensure safety and com-

Behavior	PTCS	MCS	VS
Eye opening	Spontaneous	Spontaneous	Spontaneous
Attention	Impaired selective/sustained attention	Inability to focus/sustain attention	None
Response to pain	Defensive/anticipatory	Localization	Posturing/withdrawal
Movement	Goal-directed/appropriate object use	Automatic/object manipulation	Reflexive/patterned
Visual response	Object recognition	Object recognition/pursuit	Startle
Commands	Consistent	Inconsistent	None
Verbalization	Intelligible sentences	Intelligible words	Random vocalizations
Communication	Reliable yes-no	Unreliable yes-no	None
Affective response	Contingent	Contingent	Random

Table 1 Behavioral features associated with specific disorders of consciousness

pletion of routine self-care activities. Sherer and colleagues found that severity of confusion contributed significantly to ratings of employability at discharge from inpatient rehabilitation and productivity at 1 year post-injury [7]. Table 1 compares behavioral features of the four major DoCs.

Because there is no established objective test for conscious awareness, the determination of level of consciousness and corresponding diagnosis is based on a clinicians' subjective appraisal of elicited behavior. There is growing evidence, however, that clinicians frequently misjudge level of consciousness. Investigations consistently report that 30-40 % of patients believed to be unconscious on bedside examination actually retain conscious awareness [8-10]. This error rate is largely due to an obligatory over-reliance on behavior as a proxy for consciousness. Although behavioral observations are considered the "gold standard" in the evaluation of level of consciousness, behavioral signs can be misleading [11]. Reflexive behaviors may appear to be volitional while volitional responses may be masked by underlying sensory and motor impairments. In addition, behavioral output often fluctuates and a single observational period may be insufficient to capture evidence of conscious awareness. Nonetheless, diagnostic accuracy is critical to assure appropriate clinical management, establish an accurate prognosis, and provide appropriate information to caregivers. Misdiagnosis may limit access to medical and rehabilitation services and inappropriately influence end-of-life decision-making, including premature withdrawal of life-sustaining care.

The primary goals of rehabilitation for persons in the early phases of recovery from severe brain injury are to maintain medical stability, restore communication, and promote independence in self-care. An array of treatment interventions, including pharmacotherapy, physical management strategies, and structured sensory stimulation, are routinely administered in the inpatient rehabilitation setting to promote recovery of cognitive and motor functions. However, the absence of wellcontrolled treatment studies has slowed the development of standards of care to guide clinical decision-making regarding treatment selection. This has led some observers to describe the current approach to rehabilitation as a "black box" [12]. As a result, treatment interventions are often selected and applied in a "trial and error" manner, and the evaluation of treatment effectiveness is subject to observer bias. In the absence of objective data, treatment may be withdrawn prematurely or prolonged unnecessarily, hindering the recovery process and wasting limited resources.

Against this backdrop of diagnostic uncertainty and the prevailing "trial and error" approach to treatment, we describe a systematic, evidencebased framework for clinical management of patients with DoC. The primary objective is to demonstrate how a standardized approach to assessment can be instituted in the rehabilitation setting to inform diagnostic, prognostic, and treatment decisions. The importance of adopting an empirical approach to clinical care is underscored by recent published evidence indicating that individuals with DoC recover over a longer period of time than previously thought, and many regain the capacity to function independently [13–16].

#### Disorders of Consciousness Program Framework

The Spaulding Rehabilitation Network (SRN) Disorders of Consciousness Program was developed to provide a continuum of care specifically designed for individuals who have experienced severe acquired brain injury and have not yet regained the ability to follow instructions, communicate reliably, or perform basic self-care activities. The marked variability in the physical, cognitive, behavioral, and emotional sequelae of severe brain injury suggests that a one-size-fits-all model of rehabilitation is likely to be ineffective. In the remainder of this chapter, we describe a specialized 8-week program in which assessment and treatment procedures are standardized and administered systematically by a multidisciplinary neurorehabilitation team.

The 8-week SRN DoC Program is organized into three levels of care, each intended to address the clinical needs of patients functioning at different levels of consciousness. Program services are initiated and modified based on level-specific criteria. *Level I* focuses on individuals who have not yet recovered consciousness and whose level of functioning is consistent with coma or the vegetative state. Patients admitted to Level I are either unarousable or demonstrate fluctuations in arousal and display no command-following, purposeful movement, or communication ability. The Coma Recovery Scale-R (CRS-R) [17] (see description under section "Core Metrics") is the primary assessment measure used at this level to track neurobehavioral recovery and monitor response to interventions. Behavioral and pharmacologic protocols are commonly employed to facilitate arousal at this level. Level II focuses on patients in the MCS who show clear but inconsistent evidence of conscious awareness, are unable to communicate reliably, and require maximum assistance for basic care. The transition from Level I to Level II requires demonstration of at least one feature of MCS on three consecutive CRS-R exams. The CRS-R and Individualized Quantitative Behavioral Assessment (IQBA) protocols [18], which rely on single-subject research methodology to investigate case-specific questions, are the key assessment procedures used at this level. Therapies designed to foster response consistency, augmentative communication, and environmental control strategies are typically initiated at this level. Level III focuses on individuals in the posttraumatic confusional state. Patients in PTCS are alert and have regained the ability to communicate reliably, but remain confused and highly distractible, often with sleep disturbance, impulsivity, and agitation. Progression to Level III is achieved once reliable yes-no responses are demonstrated across three consecutive CRS-R exams. The primary assessment measure used in Level III is the Confusion Assessment Protocol (CAP) [6], which monitors seven cardinal signs associated with acute confusion (see description in section "Core Metrics").

#### DoC Program Care Map

In order to institute a systematic approach to care and maintain adherence to the program timeline, a specialized DoC Care Map was developed. The Care Map is divided into two sections. The discipline-specific section details the clinical services for which each rehabilitation specialist on the team is responsible. In contrast, the interdisciplinary section displays the activities that are shared by all team members. The Care Map specifies the timing of all assessment, treatment planning, and educational activities that are administered over the course of the 8-week program. The primary aim of the Care Map is to ensure that all components of the program are administered systematically and in accord with the pre-arranged timeline. Table 2 shows the interdisciplinary section of the DoC Program Care Map.

Assessment and treatment interventions are provided by a multidisciplinary team comprised of specialists in neuropsychology, physiatry, nursing, physical therapy, occupational therapy, speech language pathology, social work, case managers, and other specialists as appropriate. On admission to the DoC Program, participants undergo a standardized assessment carried out jointly by all members of the team. A comprehensive battery of "core metrics," referred to as the "DOC COMPASS" (i.e., Disorders of Consciousness COMPrehensive ASSessment Battery), is administered to establish a functional baseline across multiple domains. Some of the core metrics are administered by all members of the team, while others are assigned to particular disciplines, based on expertise. A fixed assessment schedule has been established with the frequency of administration varying by measure. Table 3 provides a summary of the core metrics and corresponding assessment schedule.

#### **DOC COMPASS**

#### **Core Metrics**

All patients admitted to the DoC Program undergo comprehensive assessment using a battery of core metrics that have been vetted for use in patients with DoC. Performance criteria have been established that determine when a particular core metric should be discontinued (e.g., when valid assessment is not possible) or transitioned to a "higher-level" measure (e.g., when ceiling

	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8
Assessment								
Initial team assessment	Х							
Family interview to obtain history	Х							
Neurobehavioral clinic		Х						
Clinical team meeting		Х			Х			
COMPASS administered	Х	Х	Х	Х	Х	Х	Х	Х
Specialized metrics			Х	Х	Х	Х	Х	Х
Final review of data								Х
Treatment								
Interdisciplinary team conference (ITC)	Х	Х	Х	Х	Х	Х	Х	Х
Implement initial treatment intervention(s)		Х	Х	Х				
Implement revised treatment intervention(s)					Х	Х	Х	Х
Establish transition/discharge plan						Х		
Implement transition/discharge plan							Х	Х
Neurobehavioral profile finalized for transition/discharge								Х
Family education								
Family orientation w/case manager, nurse manager, and program director	Х							
Meeting w/outreach coordinator	Х							
Family team meeting w/clinical team		Х				Х		
Family education seminar	Х	Х	Х	Х	Х	Х	Х	Х

#### Table 2 SRN disorders of consciousness program care map

Table 3 SRN DoC program assessment	schedule
------------------------------------	----------

Neurobehavioral measure	When to start administration	Frequency	When to discontinue	
Agitated Behavior Scale (ABS)	Admission	1× per nurse shift	3 consecutive days	
		$1 \times$ per therapy session	of scores $\leq 21$	
Coma-Recovery Scale-Revised (CRS-R)	Admission unless EMCS	2× per week	3 consecutive subscale scores of 4 for Auditory, 2 for Communication, and 3 for Arousal	
Confusion Assessment Protocol (CAP)	Admission if EMCS or upon discontinuation of CRS-R	1× per week	2 consecutive scores of not confused	
Galvenston Orientation Attention Test	Completion of the CAP if disorientation remained a symptom	1× per week	2 consecutive administration with Total Error points <25	
Disability Rating Scale (DRS)	Admission	1× per week	Never	
Functional Communication Measures (FCM)	Admission	Bi-weekly	Never	
Limb Movement Protocol (LMP)	Admission	1× per week	2 consecutive scores of 72	
Verbal Fluency	Consistent intelligible speech is present	1× per week	Never	
Medical Complication Checklist	Admission	1× per week	Never	

effects are apparent). The section below provides a brief summary of the core metrics in the *DOC COMPASS* that were selected to represent particular domains of function.

#### **Neurobehavioral Status**

*Coma Recovery Scale-Revised* (*CRS-R*): The CRS-R is a standardized measure of neurobehavioral function that has been widely used in dif-

ferential diagnosis, prognostic assessment, and outcome measurement in persons with DOC [10, 17, 19, 20]. The scale consists of 23 behavioral items that are weighted to reflect progressively increasing neurologic function. There are six subscales designed to assess arousal level, audition and language comprehension, expressive speech, visuoperceptual abilities, motor functions, and communication ability. Scoring is based on the presence or absence of operationally defined behavioral responses elicited by standardized stimulus presentation. The lowest items on each subscale represent brain stem reflexes, while the highest items reflect cognitively mediated behaviors. The CRS-R has been shown to be reliable and valid when administered by licensed medical and rehabilitation personnel [17, 19, 20]. The scale is completed on admission to determine diagnosis (e.g., VS or MCS), establish a neurobehavioral baseline, and identify level of care. Following baseline assessment, the CRS-R is administered twice weekly to monitor rate of recovery. CRS-R administration is discontinued in favor of the CAP when the criteria for emergence from MCS (i.e., consistent functional object use and/or functional communication) are met on three consecutive examinations.

*CAP*: The CAP is a compilation of items extracted and/or modified from existing standardized measures used to assess delirium, posttraumatic amnesia, and agitation [6]. The CAP includes seven subscales that assess level of cognitive impairment, disorientation, agitation, symptom fluctuation, sleep disturbance, decreased daytime arousal, and psychotic symptoms. In the SRN DoC Program, the CAP is initiated when CRS-R performance stabilizes at ceiling. The CAP is discontinued when fewer than four symptoms of confusion are present and there is no further evidence of disorientation on two consecutive examinations.

*Disability Rating Scale* (DRS): The DRS is the most widely used functional outcome scale in TBI research and practice. The scale monitors degree of disability and tracks change over time in patients recovering from coma [21]. Areas of

function assessed include arousal level (i.e., eye opening), motor responsiveness, communication ability, and cognitive ability for feeding, toileting, and grooming. General level of functioning and employability are also rated. DRS scores are obtained weekly by certified team members.

#### **Cognitive-Linguistic Function**

*Functional Communication Measures* (FCM): The FCMs were developed by the American Speech-Language Hearing Association to grade a variety of communication, swallowing, and cognitive abilities [22]. Performance is rated on a 7-point Likert scale. FCMs selected for use in the SRN DoC Program include Motor Speech, Spoken Language Comprehension, Spoken Language Expression, Swallowing, Augmentative-Alternative Communication, Fluency, Attention, and Memory. FCMs are completed by certified Speech Language Pathologists on admission, at week 4 and at week 8.

*Controlled Oral Word Association Test (COWAT)*: After intelligible speech is recovered (i.e., CRS-R Oromotor/Verbal subscale score of 3), the COWAT is added [23]. The COWAT assesses verbal initiation and fluency by instructing the patient to name as many words as possible within 60 s that begin with a designated letter of the alphabet. Three trials are administered and a total score is obtained. The COWAT is completed weekly by the Speech-Language Pathologist.

#### **Motor Function**

Limb Movement Protocol (LMP): The LMP was developed to track motor recovery in patients with DoC [24]. This measure focuses on upper extremity function and is designed to investigate functional movement sequences that involve use of objects (4 items) and social gestures (4 items). Three trials of each item are administered and the score is based on the accuracy, completeness, and consistency of the movement sequence executed. This protocol is administered weekly by the Occupational Therapist from admission through discharge or until the maximum score (i.e., 72) is achieved on three consecutive examinations.

#### **Medical Status**

Medical Complications Checklist (MCC): The MCC is an inventory of medical complications that are commonly observed in patients with posttraumatic DoC. The complications included on the list represent those found to have the highest incidence in a large cohort of patients with DoC enrolled in a TBI Model Systems-sponsored study [25]. The intent of the checklist is to track the number and duration of complications to help determine the influence of medical instability on rate of recovery and functional outcome in patients undergoing inpatient rehabilitation. Complications represented in the MCC include, cardiac, pulmonary, fluid/electrolyte/nutrition, infectious, neurological, endocrine, hematological, gastrointestinal, urological, orthopedic, pain, neurobehavioral, skin, and head/eyes/ears/nose/ throat. The MCC is completed weekly by the attending physician or resident.

#### **Specialized Protocols**

In addition to the core metrics, the DOC COMPASS includes a wide variety of specialized assessment protocols that are employed to address case-specific clinical questions. These individually tailored protocols are designed to complement the core metrics and are typically used to address more granular assessment questions pertaining to diagnosis, prognosis, and treatment. The Neuropsychology service meets with the rehabilitation team to develop the protocols, analyzes the results, and discusses their implications for treatment and long-term care needs with the rehabilitation team and family. Examples of specialized protocols include arousal monitoring procedures to gauge the length of time the patient maintains wakefulness, command-following protocols to help differentiate volitional from involuntary behavioral responses, and response consistency protocols to determine the consistency and accuracy of specific target behaviors (e.g., yes-no responses). All specialized protocols include scripted instructions for administration and corresponding forms for data collection. Data collection is conducted jointly by all members of the treatment team, regardless of discipline. Protocols are conducted during therapy sessions and typically require no more than 10 min for administration. Protocol adjustments are initiated by the therapy team as needed. Results are discussed at rehabilitation team meetings, during family conferences and are incorporated into reports submitted to the payor. Figures 1 and 2 depict examples of Arousal Monitoring and Yes– No Response Consistency protocols.

Clinical data generated by all measures included in the DOC COMPASS are uploaded to an online database and progress is monitored weekly during Interdisciplinary Team Conferences coordinated by the Case Manager. A "Comprehensive Neurobehavioral eProfile" is automatically generated for each patient in the program and updated each week. The eProfile demonstrates current performance as well as the trajectory of recovery across domains of function. Clinical benchmarks derived from the core metrics are employed to guide decision-making regarding the need for treatment modifications, program transitions, and recommendations for discharge. A case illustration showing an example of a completed *eProfile* is presented at the end of this chapter.

Treatment interventions are carried out in the same manner as the comprehensive assessment battery. That is, after establishing the primary treatment goals, standardized treatment methods are scripted and implemented by all members of the team. The treatment plan is reviewed in week 5 and modified as indicated, based on the data collected by the rehabilitation team. Figure 3 shows an example of a standardized treatment protocol intended to facilitate arousal.

#### Family Education and Support

Recognizing that family involvement will be essential to the long-term success of the survivor, family support is viewed as a vital component of the DOC program. While there is no standard approach to family support [26], research into self-identified family needs following TBI has identified consistent themes in terms of the supports families need: (1) the need for general

Patient:	Patient: Therapist:				
Date: Therapy:					
Time:		Medications			
Behavior	First 5 mins of tx session (0-5 mins) Duration of eyelid closure	Middle 5 mins of tx session (25-30 mins) Duration of eyelid closure	Last 5 mins of tx session (25-30 mins) Duration of eyelid closure	Total duration of eyelid closure	
1-60 seconds					
61-120 seconds					
121-180 seconds					
181-240 seconds					
241-300 seconds					
Total duration of eyelid closure					

#### AROUSAL MONITORING RECORD

**Procedure:** This protocol is designed to gauge arousal maintenance using a time sampling procedure. Arousal level should be monitored during the first, middle and last five minutes of each therapy session.

**Operational Definition of Underarousal**: An episode of underarousal begins when contact between the upper and lower eyelids is maintained continuously for longer than 3 seconds and ends when contact is released for longer than 3 seconds.

**Instructions:** During the first, middle and last 5 minutes of the therapy session, observe the status of the eyelids. Any time the eyelids are observed to close for at least 3 seconds, begin timing the length of time they remain closed. Stop timing when the eyelids remain open for at least 3 seconds. Continue recording episodes of sustained eye closure in this manner during the first, middle and last 5 minutes of the session. At the end of each 5 minute interval, record the *total length of time* the eyelids were closed during that period and enter it in the appropriate time block. Next, record the *total length of time* the eyes remained closed within and across each 5 minute interval.

**Fig. 1** Arousal Monitoring protocol. The Arousal Monitoring protocol includes scripted instructions for gauging the length of time arousal is maintained over a predefined time period. Arousal level is monitored using a time sampling procedure intended to sample the first,

information about brain injury, as well as specific information concerning their family member's injury, (2) guidance on how family members can be involved in care, and (3) the need for assistance in making sense of their experience [27– 29]. Based on these findings, several components have been built into the SRN DOC program to middle, and last 5 min of each therapy session. An episode of underarousal begins when contact between the upper and lower eyelids is maintained continuously for longer than 3 s and ends when contact is released for longer than 3 s

address these needs. The program helps prepare family and friends for the future by providing training in effective caregiving and advocacy strategies, while providing emotional support in adjusting to the new challenges in their lives.

Family support begins at the point of initial contact, prior to admission. Educational materials

#### Yes/No Comprehension

**Directions:** Administer runs of 6 paired yes/no questions, as outlined below, within the domains of personal information, orientation information and/or general knowledge. Please attempt administration of at least *one set* per tx session. Record pt's arousal as noted below, and record response (if any) occurring within 10 seconds of auditory stimulus.

Date:

Positioning during administration: \_\_\_\_\_ Did eyes remain open throughout administration (circle): YES / NO Was deep pressure stimulation provided (circle): YES / NO Did pt benefit from deep pressure (circle): YES / NO / NA

Personal Information Questions:

Stimulus:	Response? (+/-)	Accurate? (+/-)
Are you a man/male?		
Is your name*?		
Are you a*?		
Is your name***?		
Are you a woman/female?		
Are you a****?		
TOTAL:	/ 6	/ 6

enter name of patient

"enter an occupation other than patient's occupation

"enter a name other than patient's name

""enter patient's occupation

#### **Orientation Information Questions:**

Stimulus:	Response? (+/-)	Accurate? (+/-)	
Are we at a shopping mall?			
Is the year 2002?			
Are we in a hospital?			
Are you sitting in a bathtub?			
Is the year*?			
Are you sitting in a chair?			
TOTAL:	/ 6	/ 6	

enter current year

#### General Knowledge Questions:

Stimulus:	Response? (+/-)	Accurate? (+/-)
Is grass green?		
Is ice hot?		
Is a rock hard?		
Is grass red?		
Is ice cold?		
Is a rock soft?		
TOTAL:	/ 6	/ 6

**Fig. 2** Yes–No Response Consistency and accuracy monitoring protocol. After presenting a question concerning personal orientation, situational orientation and semantic knowledge, the examiner records whether a

discernible verbal or gestural "yes" or "no" response occurred, and whether or not it was accurate. Percent accuracy is determined for each type of question and for all questions collapsed **Sustained Attention Protocol** 

SRN Disorders of Consciousness Program

Patient:

Time:

Therapist:

**Objective:** Mr. A will independently sustain performance on a low cognitive load task for 10s continuously.

# Protocol Description:

This protocol is designed to facilitate recovery of sustained attention. Three different tasks will be administered requiring uninterrupted perform ance. Three trials of a single task will be conducted per session. Tasks can be modified as needed but should be characterized by low cognitive demands and should be able to be completed within a 10s timeframe. Protocol steps are as follow:

- Describe the task in simple terms.
   Request verbal reinstatement of the second sec
- Request verbal reinstatement of the task. Repeat until accurate or change task if 3 consecutive attempts are failed.
  - Initiate task.
- 4. Re-direct attention to task by calling out patient's name.
  - 5. Request verbal reinstatement of task.
    - a. If accurate, prompt to continue task.
- b. If maccurate, re-state task and rehearse until task is accurately repeated or 3 consecutive attempts are failed.
  - c. If accurately repeated, complete trial.
- On completion of steps 1-5, re-administer task instructions (repeat instructions 1x) and conduct a new trial but provide no assistance.
- In the table below, record whether the trial was completed without loss of set and without assistance, completed with verbal prompts to "Keep going" or failed (ig, set loss even with verbal prompting).

Fig. 3 Sustained Attention Training protocol. The protocol record shows detailed instructions for administration of an attention training protocol on the left flanked by a corresponding scoring grid on the right. The objective of this protocol is to improve the duration of time the patient remains on-task. A combination of verbal cuing, verbal

Persists on task for 11 or 10" w/o loss of requires set to "kee				
on task Fails to persist o but task for 10" ever prompts with verbal going" prompting				

reinstatement, and in vivo rehearsal strategies is utilized and performance is monitored using a standardized record sheet that tracks the frequency of occurrence of operationally defined target behaviors describing the program and staff are provided during a tour of the facility. Once admitted, a notebook containing a description of the program, the disciplines involved, basic information about brain injury, and information about support and educational resources is provided. The Case Manager and Social Worker meet with the family within the first 72 h of admission. The Case Manager helps orient the family to the facility, identifies immediate family issues or concerns, and begins to explore discharge options and family resources. It is important that this discussion begins at the point of admission so that realistic expectations for treatment goals and length of stay are established at the outset of treatment. The Social Worker evaluates the family's psychosocial status and needs and provides education about the DOC program and facility resources available to the family. Ongoing support is provided to the family, assisting with housing, obtaining outside counseling, encouraging healthy self-care habits, and coping strategies for family members, such as how to be an effective member of the treatment team. The Social Worker offers a family support group to facilitate meetings among family members for various patients and to address emotional stress experienced by family members. A mentor, often an experienced family member of a brain injury survivor, is available to meet with family members to provide further emotional support and practical advice. Both the Case Manager and the Social Worker help the family prepare for the next level of care which may involve identifying further treatment options, transitioning to a lower level of care, exploring funding options, and accompanying family members in visits to other facilities.

Team members participate in the educational process on an on-going basis by explaining their role in treatment and providing information about brain injury, DoC, assessment tools, and treatment approaches. Families are encouraged to attend therapy sessions and participate in the care of the patient where appropriate. Therapy staff and nursing staff provide formal training in how to care for and support individuals with brain injury. This may involve how to provide supervision while walking, feeding, or bathing instructions, encouraging use of a consistent communication system, or how to maintain a gastrostomy tube. This "hands-on" approach is the best way for family members to learn about brain injury in general, and their family member's needs, specifically. It is also the best way for them to acquire the skills they will need to care for their family member at home or to advocate for services for their family member if continued residential care is needed.

In Week 2 of the Program, the family meets formally with the interdisciplinary team. At this meeting, detailed information is provided about the results of the initial assessment, treatment objectives, procedures to be implemented, and expectations for the rehabilitation course. The 8-week timeline is strongly emphasized, as is the use of clinical benchmarks to guide decisionmaking regarding the selection of assessment and treatment procedures. Because of the timelimited nature of the DOC program, preparation for discharge and transition to the next stage of care and treatment starts at this meeting. The Case Manager meets with the family on a regular basis to keep the family informed about care and progress and when the time comes, assists with preparation for discharge to the next stage of care. The frequency of these meetings is determined on an individual basis, based on the support needs of the family. Families are made aware of treatment resources within the community (e.g., state chapter of the Brain Injury Association of America), in addition to resources available within the hospital (e.g., educational sessions, outpatient support groups). The team meets again formally with the family at the end of Week 5 to review progress and discuss plans for discharge from the program.

#### **Case Illustration**

To exemplify the application of a systematic, evidence-based approach to clinical management of patients with DoC, we provide a case illustration below.

*Medical History*: AB is a 24-year-old female who sustained a severe TBI with loss of consciousness

when she was struck by a car as a pedestrian. She had a Glasgow Coma Scale [30] score of 4 at the scene, indicating deep coma. Initial neuroimaging studies showed a subarachnoid hemorrhage which did not require neurosurgical intervention. An ICP bolt placed for pressure monitoring was discontinued on day 4. Follow-up MRI on day 8 revealed punctate hemorrhages in the posterior corpus callosum, right cerebellar region along the posterior falx and over the left posterior parietal lobe, consistent with grade II diffuse axonal injury. She also sustained significant polytrauma requiring multiple orthopedic surgeries. She regained spontaneous eye-opening on day 15 but remained poorly responsive. Active movement was noted in the left upper extremity, but there was no evidence of command-following or purposeful movement. The acute course was complicated by ventilatordependent pneumonia and recurring cardiorespiratory problems which eventually stabilized. She remained in a vegetative state for approximately 6 weeks after which she began to display automatic movements (e.g., nose-scratching, grasping the bedrail) followed by inconsistent movement of the right hand and toes to command. She was started on amantadine but this was discontinued as there was a concomitant increase in restlessness and stereotypical movements of the left arm.

On day 45, AB was transferred to the SRN Disorders of Consciousness Program for comprehensive inpatient neurorehabilitation. Repeat MRI of the brain revealed an extra-axial fluid collection, thought to be a subdural hygroma, overlying the left frontal lobe and a small focus of hyperattenuation in the left medial temporal lobe. The ensuing neurorehabilitation program was guided by the 8-week Care Map described below.

*Week 1*: The core measures of the *DOC COMPASS* were initiated by the rehabilitation team in week 1 to establish a baseline across functional domains. The opening score on the Coma Recovery Scale-Revised was 14, reflecting poorly sustained eye-opening, inconsistent command-following, visual pursuit, automatic motor behavior, unintelligible vocalizations, and no discernible yes–no responses (see CRS-R Profile in Table 4). Performance on the CRS-R

**Table 4** AB's CRS-R Profile on admission to the SRN Disorders of Consciousness Program

	Coma Recovery Scale-Revised			
Subscale	Best response	Score		
Auditory	Reproducible command- following	3		
Visual	Pursuit	3		
Motor	Automatic motor response	5		
Oromotor/verbal	Vocalization/oral movement	2		
Communication	None	0		
Arousal	Eye opening w/stimulation	1		
Total score		14		
CRS-R diagnosis		MCS		

confirmed her transition from VS to MCS and triggered implementation of the remaining core measures. Administration of the LMP by the occupational therapist resulted in an initial score of 38/72. Item analysis showed a high rate of partially executed movement sequences. AB's lack of verbal or gestural communication produced a Level 1 rating on the Spoken Language Expression subscale of the FCM. Global functional status as assessed by the DRS yielded a score of 26, which falls in the most severely disabled range of function. The results of the core metrics were presented at the biweekly Interdisciplinary Team Conference (ITC) to maintain communication across therapeutic disciplines and provide clinical status updates to the in-house case manager. To address the daytime fluctuations in arousal level, the Arousal Facilitation Protocol was administered by all team members at least once during each therapy session. In addition, simple cuing strategies were used to redirect attention and improve timeon-task during therapy sessions.

*Week 2*: In week 2, AB was evaluated by the Neuropsychology service in the Neurobehavioral Clinic. The purpose of the Clinic visit was to further investigate AB's current level of cognitive function, confirm the working diagnosis, establish the prognosis for further recovery, and help determine the primary treatment objectives. Examination findings replicated the behavioral signs of conscious awareness reported by the rehabilitation team, which included inconsistent command-following, visual pursuit, and auto-



#### Response Consistency/Accuracy Protocol

**Fig. 4** AB's performance on the Yes–No Response Consistency and Accuracy Monitoring protocol. The graph shows the percentage of trials in which any discernible yes–no response was detected following presentation of a question (*circles* on *dotted line*) over a 1-week interval. The solid lines indicate the percentage of accurate

matic motor responses. Episodes of intelligible verbalization were also elicited during this assessment. There was some evidence of leftsided sensory inattention and repetitive stereotypical movements of the right arm were again noted. Additional assessment of the right arm movements suggested these movements represented "frontal release behavior," likely reflecting loss of inhibitory control caused by the left frontal lesion noted on prior neuroimaging studies. It was recommended that the initial objectives of the rehabilitation program focus on establishing a reliable communication system and managing the right upper extremity motor disinhibition. At the end of week 2, the neurorehabilitation team met to discuss the program objectives, select the assessment and treatment procedures, and establish performance benchmarks. By the end of week 2, AB had begun to verbalize "yes" and "no." The emergence of yesno responses triggered the use of a specialized protocol for determining the consistency and accuracy of these responses to different types of questions (see Fig. 4). These data were used to help determine readiness for communication

responses to personal orientation (*diamonds*), situational orientation (*squares*), and semantic knowledge (*triangles*) questions. Results indicate that general response consistency improved from 65 to 100 %, while accuracy improved by approximately 10 % during the same interval across all three types of questions administered

training. Treatment continued to focus on improving arousal, attention, and response consistency. The initial family meeting was also completed in week 2 to familiarize the family with the 8-week DoC Program and to review the preliminary findings, treatment objectives, and treatment methods. The family was enlisted in the communication training protocol to maximize exposure to this intervention and promote generalization.

Weeks 3–5: During week 3, AB continued to progress. She achieved three consecutive scores of 23 (max=23) on the CRS-R, indicating emergence from MCS and that she was performing at the ceiling of the CRS-R. Consequently, the CRS-R was discontinued and the CAP initiated. AB also achieved the maximum score of 72 on two consecutive assessments with the LMP, triggering discontinuation of this measure not shown in Fig. 5. The DRS score decreased by 14 points between weeks 2 and 4, reflecting significant improvements in arousal level, motor functions, and performance in self-care activities. The initial CAP score showed five symptoms of confusion, including cognitive impairment, disorientation,





**Fig. 5** *DOC COMPASS eProfile* illustrating AB's performance across all 8 core measures. Panel (a) shows weekly scores on the CRS-R (broken down by specific subscales), LMP (broken down by response category), FCM, and Disability Rating Scale.









**Fig. 5** (continued) initially below the diagnostic cut-off (i.e., >20), and then cross into the clinical range before eventually stabilizing at a subthreshold level. These find-

agitation, symptom fluctuation, and sleep disturbance, supporting a diagnosis of posttraumatic confusional state. Elavil was titrated up to 40 mg to reduced restlessness and address ongoing daytime somnolence.

At week 5, a follow-up team meeting was held to review the data generated by the core metrics and specialized protocols. Apart from updating AB's clinical status, the discussion focused on her rate of recovery and the best options for continued care following discharge. Review of the yes–no protocol demonstrated 70–80 % accuracy across personal information, orientation, and general knowledge questions, signaling readiness for formal communication training. A communication training protocol was developed for pilot testing in weeks 6–8.

Weeks 6-8: At week 6, a follow-up family team meeting was held to review the degree and rate of progress across functional domains, and to discuss discharge recommendations. To aid the family's understanding of the clinical findings, data acquired from the DOC COMPASS were converted to graphics and presented as charts and figures. Discharge arrangements were initiated while the team continued to administer the core measures and specialized protocols during the last 2 weeks of the program. By week 8, AB presented with only one symptom of confusion on the CAP, indicating resolution of PTCS. Language recovery paralleled resolution of the confusional state as evidenced by improved FCM ratings across language areas and an increase in the verbal fluency score from 5 to 23 within a 4-week span (normal age-corrected mean=45). The Yes-No Response Consistency/Accuracy Protocol was discontinued at week 7 as she achieved 100 % accuracy across all three categories of questions. The total DRS score improved to 6, suggesting ongoing functional improvement and moderate residual cognitive and physical disability. AB's eProfile, shown in Fig. 5, depicts her performance within each functional domain across the 8-week program. AB was discharged home upon completion of the program with recommendations for 24-h supervision and outpatient speech, physical, and occupational therapies.

#### Conclusion

Severe brain injury is a complex neurobiologic disorder that can result in prolonged disturbance in consciousness. Attesting to the challenges associated with this condition, diagnostic error is common and treatment practices are highly variable across patients with similar problems. The prevailing "trial and error" approach to clinical management is inefficient, impedes evaluation of effectiveness, and slows accumulation of knowledge. In contrast, a systematic, data-driven approach offers a platform to administer assessment and treatment in a more objective manner, provides the opportunity to monitor progress in real time, and generates empirical data that can be used to inform best practices.

In this chapter, we describe an operational framework for clinical management of patients with DoC designed to inform clinical practice, in the context of clinical practice. In this model, the clinical setting serves as an in vivo laboratory, and the rehabilitation team functions singlemindedly, directing its efforts toward a set of common goals. The assessment process is interleaved with treatment, interventions are standardized, and common outcome measures employed throughout the rehabilitation course. This approach provides a clinical roadmap designed to improve consistency of care, generates objective evidence to inform diagnostic and prognostic decision-making, facilitates caretaker education, and expands the base of knowledge concerning TBI.

#### References

- Faul, M. X. L., Wald, M. M., & Coronado, V. (2010). *Traumatic brain injury in the United States: Emergency department visits, hospitalizations and deaths, 2002-2006.* Atlanta, GA: National Center for Injury Prevention and Control.
- Posner, J. B., & Plum, F. (2007). *Plum and Posner's diagnosis of stupor and coma* (4th ed., Vol. 14, 401p.). Oxford, England: Oxford University Press.
- Multi-Society Task Force on PVS. (1994). Medical aspects of the persistent vegetative state (1). *The New England Journal of Medicine*, 330(21), 1499–1508.

- Quality Standards Subcommittee of the American Academy of Neurology. (1995). Practice parameters: Assessment and management of patients in the persistent vegetative state (summary statement). *Neurology*, 45(5), 1015–1018.
- Giacino, J. T., Ashwal, S., Childs, N., Cranford, R., Jennett, B., Katz, D. I., et al. (2002). The minimally conscious state: Definition and diagnostic criteria. *Neurology*, 58(3), 349–353.
- Sherer, M., Nakase-Thompson, R., Yablon, S. A., & Gontkovsky, S. T. (2005). Multidimensional assessment of acute confusion after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 86(5), 896–904.
- Sherer, M., Yablon, S. A., Nakase-Richardson, R., & Nick, T. G. (2008). Effect of severity of post-traumatic confusion and its constituent symptoms on outcome after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(1), 42–47.
- Childs, N., Mercer, W., & Childs, H. (1993). Accuracy of diagnosis of persistent vegetative state. *Neurology*, 43(8), 1465–1467.
- Andrews, K., Murphy, L., Munday, R., & Littlewood, C. (1996). Misdiagnosis of the vegetative state: Retrospective study in a rehabilitation unit. *BMJ*, *313*(7048), 13–16.
- Schnakers, C., Vanhaudenhuyse, A., Giacino, J., Ventura, M., Boly, M., Majerus, S., et al. (2009). Diagnostic accuracy of the vegetative and minimally conscious state: Clinical consensus versus standardized neurobehavioral assessment. *BMC Neurology*, 9, 35.
- Giacino, J. T., Schnakers, C., Rodriguez-Moreno, D., Kalmar, K., Schiff, N., & Hirsch, J. (2009). Behavioral assessment in patients with disorders of consciousness: Gold standard or fool's gold? *Progress in Brain Research*, 177, 33–48.
- Dejong, G., Horn, S. D., Gassaway, J. A., Slavin, M. D., & Dijkers, M. P. (2004). Toward a taxonomy of rehabilitation interventions: Using an inductive approach to examine the "black box" of rehabilitation. *Archives of Physical Medicine and Rehabilitation*, 85(4), 678–686.
- Lammi, M. H., Smith, V. H., Tate, R. L., & Taylor, C. M. (2005). The minimally conscious state and recovery potential: A follow-up study 2 to 5 years after traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 86(4), 746–754.
- Katz, D. I., Polyak, M., Coughlan, D., Nichols, M., & Roche, A. (2009). Natural history of recovery from brain injury after prolonged disorders of consciousness: Outcome of patients admitted to inpatient rehabilitation with 1-4 year follow-up. *Progress in Brain Research*, 177, 73–88.
- Estraneo, A., Moretta, P., Loreto, V., Lanzillo, B., Santoro, L., & Trojano, L. (2010). Late recovery after traumatic, anoxic, or hemorrhagic long-lasting vegetative state. *Neurology*, *75*(3), 239–245.
- Nakase-Richardson, R. W. J., Giacino, J. T., et al. (2012). Longitudinal outcome of patients with disordered

consciousness in the NIDRR TBI model systems programs. *Journal of Neurotrauma*, 29(1), 59-65.

- Giacino, J. T., Kalmar, K., & Whyte, J. (2004). The JFK Coma Recovery Scale-Revised: Measurement characteristics and diagnostic utility. *Archives of Physical Medicine and Rehabilitation*, 85(12), 2020–2029.
- DiPasquale, M. C. W. (1996). J. The use of quantitative data in treatment planning for minimally conscious patients. *The Journal of Head Trauma Rehabilitation*, 11(6), 9.
- Schnakers, C., Majerus, S., Giacino, J., Vanhaudenhuyse, A., Bruno, M. A., Boly, M., et al. (2008). A French validation study of the Coma Recovery Scale-Revised (CRS-R). *Brain Injury*, 22(10), 786–792.
- Lovstad, M., Froslie, K. F., Giacino, J. T., Skandsen, T., Anke, A., & Schanke, A. K. (2010). Reliability and diagnostic characteristics of the JFK Coma Recovery Scale-Revised: Exploring the influence of rater's level of experience. *The Journal of Head Trauma Rehabilitation*, 25(5), 349–356.
- Rappaport, M., Hall, K. M., Hopkins, K., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: Coma to community. *Archives of Physical Medicine and Rehabilitation*, 63(3), 118–123.
- 22. American Speech-Language-Hearing Association. (2003). National Outcomes Measurement System (NOMS): Adult speech-language pathology user's guide. Rockville, MD: Author [Internet].
- Strauss, E., Sherman, E. M. S., Spreen, O., & Spreen, O. (2006). A compendium of neuropsychological tests: Administration, norms, and commentary (3rd ed., Vol. xvii). Oxford, England: Oxford University Press. 1216p.
- Schiff, N. D., Giacino, J. T., Kalmar, K., Victor, J. D., Baker, K., Gerber, M., et al. (2007). Behavioural improvements with thalamic stimulation after severe traumatic brain injury. *Nature*, 448(7153), 600–603.
- Whyte, J., Nordenbo, A. M., Kalmar, K., Merges, B., Bagiella, E., Chang, H., et al. (2013). Medical complications during inpatient rehabilitation among patient with traumatic disorders of consciousness. *Archives of Physical Medicine and Rehabilitation*, 94(10), 1877–1883.
- Sander, A. M. (2007). Brain injury and the family. *NeuroRehabilitation*, 22(1), 1–2.
- Sinnakaruppan, I., & Williams, D. M. (2001). Family carers and the adult head-injured: A critical review of carers' needs. *Brain Injury*, 15(8), 653–672.
- Gan, C., Campbell, K. A., Gemeinhardt, M., & McFadden, G. T. (2006). Predictors of family system functioning after brain injury. *Brain Injury*, 20(6), 587–600.
- 29. Bond, A. E., Draeger, C. R., Mandleco, B., & Donnelly, M. (2003). Needs of family members of patients with severe traumatic brain injury. Implications for evidence-based practice. *Critical Care Nurse*, 23(4), 63–72.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness. A practical scale. *Lancet*, 2(7872), 81–84.

## Behavioral Assessment of Acute Neurobehavioral Syndromes to Inform Treatment

#### Risa Nakase-Richardson and Clea C. Evans

#### Abstract

Behavioral and cognitive impairments after traumatic brain injury are commonly associated with poor community and social outcomes including high unemployment and decreased independent living. Few studies have investigated this important topic of rehabilitation care. Further, rehabilitation care providers have a paucity of training in dealing with challenging behaviors after brain injury. Given the breadth of this topic, this chapter focuses on behavior management on the inpatient rehabilitation unit. First, we present the evolution of neurobehavioral recovery with an emphasis on agitation. Next we present the behavioral principles of functional analyses to inform and develop an individualized treatment approach. Learning principles are reinforced with cases vignettes that highlight issues presented in the chapter. Finally, common considerations in working with team members, brain injury survivors, and family members are discussed.

#### Keywords

Behavior management • Behavior assessment • Treatment • Brain injury • Rehabilitation

R. Nakase-Richardson, Ph.D. (⊠) Department of Mental Health and Behavioral Sciences, James A. Haley Veterans Hospital, Tampa, FL, USA

University of South Florida Medical School, Tampa, FL, USA e-mail: Risa.Richardson@va.gov

C.C. Evans, Ph.D. Methodist Rehabilitation Center, Jackson, MS, USA

#### Background

Individuals with TBI can experience life-long changes in physical, behavioral, and cognitive functioning. Behavioral and cognitive impairments are commonly associated with poor community and social outcomes including high unemployment and decreased independent living. Behavioral impairments commonly include aggression, irritability, and poor social skills. Such impairments have been reported to be a better predictor of caregiver burden than severity of injury, physical impairments, or cognitive impairments alone [1–3]. Despite the frequency and impact of behavioral impairments after TBI, there is a paucity of research examining the efficacy of behavioral interventions for persons with TBI [4–6]. Carnevale and colleagues trained family members in behavior management techniques in the home and found that ongoing involvement with clinical staff was associated with better behavioral outcome [5]. Ongoing involvement of staff likely helped reinforce the principles of behavior management taught as part of the intervention.

Behavior Management Training for Rehabilitation Providers: Other care providers such as rehabilitation therapists and nurses who work with TBI survivors have reported a paucity of education regarding provision of non-pharmacological interventions to maximize compliance and treatment interventions when working with TBI survivors [7]. Further, the impact of behavior management education (BME) is unclear due to variable methods of education delivery, educational settings, and outcomes evaluated [6, 8]. Typical outcomes have included satisfaction with education, rating of commitment to change patient management practices, and patient outcomes (e.g., number of falls, restraint use, psychotropic medication use). To date, none of these outcomes have been shown to improve with BME for staff. Due to the patientspecific nature of behavioral intervention, global outcomes in group studies may mask individual responses to educational interventions. While it is challenging to determine the direct benefit of behavioral management education for patients, it is even more difficult to determine the associated benefit in decreased burden for care-providers. Further, behavior management paradigms lack a conceptual approach for how the interventions should differ across diverse neurological patients with varying deficits.

While studies of training in behavior management have shown limited or no benefit, direct studies of behavioral interventions show decreases in behavioral problems and improved community living success for persons with severe behavior problems [7, 9]. Despite this, rehabilitation providers who work with TBI survivors have reported a lack of education regarding behavior management therapeutic techniques and understanding of cognitive disorders to maximize compliance and treatment interventions [7]. Little is known about the educational needs and best practices used by other rehabilitation disciplines in different settings. Further, there is a paucity of understanding of how behavioral management techniques should be individualized for the unique cognitive profile of a TBI survivor in different stages of recovery (acute vs. post-acute). Given the breadth of issues surrounding behavior management of persons with TBI, we will focus the remainder of this chapter on the management of common acute neurorehabilitation issues.

#### Managing Agitation on the Inpatient Unit

On inpatient rehabilitation units, agitation is one of the most frequently observed behavioral problems after TBI [10, 11]. Agitation typically occurs during the period of posttraumatic amnesia (PTA) [12]. In the PTA period, patients expedisorientation, reduced rience ability concentrate, irritability, and inability to store memories [13, 14]. Agitated behaviors present as repetitive, non-goal directed, and unproductive. Approximately, 33-72 % of TBI patients exhibit agitation following recovery from the acute neurological condition of TBI, but this typically resolves on its own [15, 16]. Many behavioral characteristics are used to define agitation [17– 19]. Agitated behaviors range from more mild behaviors (e.g., nail-biting, foot-tapping, hand wringing, the inability to sit still, or pacing) to more severe behaviors (e.g., violent outbursts, including physical aggression, biting, spitting, screaming, self-harm, and sexually inappropriate comments or actions) [20, 21].

Therapists taking care of patients with acquired brain injury identify frustration in patients as the most common feeling before agitation. Patients also were reported to be distressed, anxious, scared, angry, and desperate prior to becoming agitated [22]. These losses of emotional control are influenced by brain damage. The limbic system (e.g., amygdala and hippocampus) and frontal lobe, which are commonly affected by TBI, are associated with emotional function. In the limbic system, the amygdala enhances response to fearful stimuli and identifies threat [23]. Damage to the amygdala produced deficits in identifying threat and fear, which can contribute to agitated behavior [24]. In addition, the amygdala functions to mediate impulses from the prefrontal cortex and the hypothalamus [25]. Therefore, when a patient has an injury to the amygdala, violent behavior and agitated/aggressive behavior are increased due to poorly regulated impulses [25]. Frontal lobe damage also results in a lack of control over emotionally charged behavior [26–28]. The frontal lobes regulate attention, provide continuity and coherence of behavior, and adjust behavior. Losses of these cognitive functions result in emotional lability and decreased inhibitory control [29]. In clinical research, patients who suffered orbitofrontal area damage showed emotional disturbances, such as a lack of impulse control, irritability, hyperkinesis, and mood changes (anxiety and depression) [30, 31]. In conclusion, agitation is associated with a lack of impulse control and/ or emotional lability that may result from injury to the limbic system and/or frontal lobes.

Cognitive impairment is another key antecedent of agitation and a component of posttraumatic confusion. Memory deficits, attention problems, loss of executive function and confusion and/or delirium are common after TBI [16, 32]. This decreased cognitive ability changes the patient's ability to recognize and appropriately respond to environmental stimuli. In the acute stage of recovery from TBI, most patients stay in an unfamiliar hospital or acute rehabilitation center. In the presence of cognitive impairment, an individual may be overwhelmed by external stimuli, such as TV and large crowds. They are more likely to be fearful and insecure while in an unfamiliar environment. This reaction occurs because cognitively impaired patients have to expend more effort to process these stimuli. Thus, patients are less able to cope with unfamiliar environments than those without TBI and can be easily exhausted by multiple stimuli. One study found a significant relationship between severe aggression and poor language function, highlighting receptive aphasia as an associated variable that can serve as an antecedent to agitation [33].

An important antecedent of agitation is external stimulation; noxious stimuli in environments, pain, and frustration often trigger agitated behavior [34]. Even regular structured activities, including hygiene programs, meal times, and toileting, can be perceived as overstimulating and lead to agitation during rehabilitation. In particular, physical activities, such as bathing and toileting, cause a patient to be agitated because these activities pressure a patient to initiate movement [35]. In addition, direct contact to a patient's body or staff entering a patient's private space may be perceived as invasive by the patient, leading to agitation.

The combative and assaultive characteristics of agitation can make caregivers feel a sense of threat while caring for the patient. In a 5-year outcome study of head injuries, Brooks et al. [36] found that 54 % of caregivers reported that they had experienced threats and violence while caring for agitated patients after TBI. Sometimes, agitated behavior can involve physical aggression, and it can cause caregivers real harm or injury. Agitation not only reduces the opportunity for patients to engage in rehabilitation but also becomes a major stressor for patients and their caregivers. Furthermore, prolonged agitation may reduce functional independence, induce a longer hospital stay, delay or prevent return to work, and disturb family dynamics and community integration [37–40].

In addition to threatening safety, agitation can be a burden and cause major stress to family and caregivers providing care to TBI patients [27]. A study of 79 TBI patients' families revealed that primary caregivers experienced high levels of psychological distress, such as anxiety, depression, fatigue, anger, and mood-disturbance [41]. Eighty-three percent of siblings experienced a high level of psychological distress while taking care of TBI patients with behavior disturbances [42]. Agitated behaviors may preclude patients participating in therapies and decrease the opportunity to engage in rehabilitation [43]. Because of a patient's combative and threatening behaviors, healthcare providers may be very likely to suspend therapies to avoid a dangerous situation. Furthermore, patients can be excluded from their therapy due to uncooperative behaviors. Therefore, the recovery process of agitated patients is slower, and additional visits for rehabilitation may be required [43]. Bogner and colleagues [37] found that longer periods of agitation in rehabilitation patients increase hospital length of stay. This extended length of hospital stay and additional visits for rehabilitation result in financial burdens on the patient's family and caregivers.

On rehabilitation units, pharmacological interventions are commonly used to manage agitation. Anticonvulsants, antidepressants, antipsychotics, beta-blockers, and neurostimulants have been shown to have varying success rates in reducing agitated behaviors after TBI [44]. Although pharmacological intervention can be beneficial, detrimental side effects can be more harmful than the untreated agitation symptoms. In addition, medication-induced drowsiness can interrupt patient participation in therapies [45, 46]. One important area for pharmacological intervention has been the sleep-wake cycle. Disruptions in sleep are a common symptom of posttraumatic confusion, with reports of up to 78 % of patients having difficulty sleeping at night or excessive somnolence during the day [18]. Medication interventions that regulate sleep after TBI and subsequently improve cognition and behavior are needed [47].

In addition to the pharmacological approach, physical restraints have been commonly used to control agitation. The use of physical restraints, such as bed rails, Posey vests, and 2-point and 4-point soft or hard restraints, often occurs during acute rehabilitation to protect agitated patients and caregivers. However, the use of physical restraints is related to minor injuries, such as sores and abrasions, increasing agitation, and more serious injuries, such as deep vein thrombosis and pulmonary embolism due to the immobilization of the patients [48, 49].

Intervening in the environment is a preferential approach for reducing agitation. Environmental stimulation, such as very loud noises and bright lights, can be a trigger for agitation. Simple environmental alterations, such as offering a private room and prohibiting TV watching, may be effective in reducing agitated behavior [44]. Another effective environmental alteration is providing a familiar environment, perhaps by bringing personal possessions from home to the hospital. Because people feel comfortable and safe in familiar environments, their agitated behaviors can decrease. This concept is consistent with the study by Willis and LaVigna [50]. According to the results of their study, cognitively impaired TBI patients were less agitated in a more familiar environment and were more agitated in a strange environment with new people [50]. Modification of stimulation by decreasing overstimulation and providing a structured environment has been a commonly used intervention to reduce agitation in many rehabilitation units.

#### Development of Brain Injury Behavior Management Educational Tools

Despite the high prevalence of agitation and behavioral challenges associated with it, rehabilitation service providers appear to be lacking in education in this area [51]. The discussion of behavior management approaches presented is based on workshops we have provided to care providers in a number of settings including inpatient rehabilitation, outpatient rehabilitation including comprehensive community integration programs, nursing homes, primary care clinics, and others. These educational workshops have included two modules. The first module, "Assessment of Behavioral Disorders" introduced the functional analysis technique with case vignettes for behavioral rehearsal of principles presented. Following each case



vignette, discussion highlighted how functional analyses directed management of the problem behavior with an emphasis on prevention rather than "reactionary" management approaches. Education about brain injury and the recovery process was emphasized throughout. We believe thorough knowledge of brain injury sequelae is a key feature of this behavior management approach. Finally, learning principles were briefly introduced to help staff conceptualize "techniques" for getting maximal compliance from patients.

During one workshop series with our own rehabilitation staff working within brain injury programs, we asked participants to complete surveys before and after the workshop. Prior to the workshop, participants' reported number of years of working with brain injury patients was positively correlated with confidence in treating patients with cognitive disorders (spearman rho=.52, p=.016) but not behavior disorders [51]. Following the workshop, our

seasoned brain injury staff (N=21), reported improved confidence in managing both behavior (Z=-2.97, p=.003) and cognitive issues (Z=-2.38, p=.017; see Fig. 1); although participants' confidence ratings remained higher in managing cognitive impairments relative to behavior impairments (see Fig. 1) [51]. When asked where providers received education about behavior management issues, both nursing and therapy staff reported local CEU opportunities and on-the job training as the common source most (Fig. 2) [51]. Unfortunately, formal education was the least frequent source of gaining knowledge for working with behaviorally disturbed brain injury survivors (see Fig. 2 [51]).

Below, we highlight some of the critical elements of the intervention, present case studies to rehearse concepts presented, and summarize with a conceptual framework of elements critical to understanding behavioral issues after TBI. These elements can be delivered in formal workshops on BME to facilitate caregiver understanding of working with this complex patient group. We welcome you to reproduce these materials for your own use.

#### Introduction to Functional Analyses

#### What is Functional Analysis?

Many resources exist for reviewing common problems and behavior management recommendations following brain injury; however, one of the most critical components to effective behavioral interventions is implementation of functional analysis prior to an intervention [52, 53]. Functional analysis establishes the function of a behavior by examining the "three-term contingency" (i.e., ABC) model (see Table 1).

An individualized approach to behavior management includes conducting functional analysis to identify the problem behavior, antecedents, and consequences [52, 53]. Functional analysis uses both direct observation and interviews with staff, family, and possibly patients. Once the problem behavior is defined, antecedents and consequences are identified. A hypothesis may be generated from the data gathered that potentially predicts when the behavior may occur (triggers) and why (consequences) that facilitate a behavior management recommendation. Once the triggers are identified, a response prevention approach is recommended rather than a "reactionary" approach. Behavior management interventions that focus solely on implementing consequences when a problem behavior occurs are often more time-consuming and stressful than prevention strategies. By preventing the problem behavior from occurring in the first place, patient, family, and staff benefit from a less stressful rehabilitation experience. An approach may look as follows:

- 1. Conduct functional analysis/A, B, C (using interview and observational data)
- 2. Predict: when and why
- 3. Test hypothesis and continue monitoring
- 4. Evaluate intervention and modify if necessary (collect data)
- 5. Retest hypothesis or go to hypothesis #2
- 6. Evaluate intervention (collect data)

To facilitate rehearsal of the functional analyses technique, we feel it is critical to implement it with actual case vignettes. Recipients of our workshop series have repeatedly stated that applying functional analyses to cases (behavioral rehearsal) had greatly facilitated learning the concepts. Prior to practicing with cases, we want to discuss some other behavior management constructs that are often misunderstood.

Consequences that facilitate learning of behaviors are categorized into reinforcement and punishment (see Table 2 for basic definitions) [52, 53]. Although each of these techniques could have several chapters written about them, we will simplify this section by focusing on these two global constructs. Simply put, effective reinforcement increases behavior and *effective* punishment decreases it [52, 53]. Key to these constructs is figuring out what is *effective* for each person. In our

A	The antecedents to a problem behavior	What are the people, places, events, time of day occurring <i>immediately</i> before the behavior?
В	The actual problem behavior itself	What does the behavior look like ( <i>topography</i> )? What is the frequency duration intensity?
С	The <i>consequences</i> are the things that immediately follow a behavior occurring (that may or may not influence the likelihood of recurrence)	What happened <i>immediately</i> after the behavior occurred. How did people react? What did the person get (good or bad)? What did the person avoid (good or bad)? What else changed?

 Table 1
 Three term contingency model (ABC)

Other key considerations: CONTEXT. Context includes aspects of a person's environment that do not happen immediately before or just after undesirable behavior but still have an effect on the behavior. Contextual factors can include the patient's diagnosis, medications, sleep cycle, diet, and the neurobehavioral course that follows

Technique	Definition
Reinforcement	Anything that increases the likelihood that the behavior will occur again
Punishment	Anything that decrease the likelihood that the behavior will occur again

 Table 2
 Contingency management techniques

experience, lay persons equate reinforcement with giving food, going on passes, and receiving some form of praise, without realizing that reinforcers vary by person. What is reinforcing for one patient may not be for another. Further, untrained persons may be less skilled in deconstructing problem situations where subtle reinforcement is involved in why a problem behavior occurred in the first place (as highlighted in case vignettes 1-2). Similarly, punishment is often construed as taking away of privileges or verbal rebuke. This may or may not be meaningful to all patients. Lay persons are less aware of how staff responses such as "ignoring" an inappropriate behavior, a technique known as withdrawal of reinforcement, can decrease likelihood of it occurring again. This is illustrated in Case 2. As we progress through the case vignettes, it will be important to understand the basic definition of reinforcement and punishment as we analyze the consequences of the behavior problems (see Table 2). The cases are actual behavior management consultations received during inpatient behavior management rounds or inpatient staffing reports. The details of the cases have been changed to protect the privacy of patients, family, and staff. As you read the cases, you may make use of the worksheet in the Appendix to practice recording antecedents, behaviors, and consequences of the problem scenarios.

#### Behavior Management Rounds Case 1

#### Complaint

During behavioral management rounds, the patient's occupational therapist (OT) and physi-

cal therapist (PT) (both females) suggested that a particular patient needed medication to control his behavior. They reported that the patient grabbed their breasts and buttocks during therapies. The speech language pathologist (SLP) attending rounds did not have this complaint. Nursing was not present and had not voiced this concern previously. Therapists also complained that the parents "just stood there" and did nothing to assist them in these moments.

#### Background

Patient was a 17-year-old male status post-severe TBI from an All-Terrain Vehicle accident 5 months earlier. He arrived for comprehensive rehabilitation at 3.5 months post-injury because of the severity of his TBI. He initially presented minimally conscious with episodes of dysautonomia requiring sedating medications to manage. Review of neuroimaging revealed diffuse axonal injury in bilateral frontal lobes, temporal lobes, and parietal lobes. Although he was nonverbal at the time of evaluation, he could point to yes/no cards to answer simple questions, which was a significant improvement in his responsiveness. When orientation was assessed this way, he was oriented to name and hometown only. He used a wheel chair for ambulation but was dependent in locomotion. He was also dependent in all aspects of self-care but was able to use his left hand to do gross motor tasks, such as holding cones in occupational therapy. Because his left hand was his only good arm, he also used it to point to yes/no visual cards. He was tall and large, requiring a two-person assist with all transfers. His parents were supportive and present throughout the day to accompany their son during all therapies and aspects of his medical care.

#### Psychologist's First Observation/Data Gathering Session

The patient was observed in a joint physical and occupational co-therapy session. The patient was noted to attempt grabbing behavior with his left hand only when working in close proximity (most of the time due to his physical status) to his young, female therapists. This commonly occurred when working on sitting balance on the mat in the gym. When the patient grabbed his PT, she verbalized "No, you shouldn't do that." She appeared to be embarrassed which resulted in nervous laughter during her redirection of the patient. The parents were present but did not respond to his inappropriate behavior. Subsequent interview with the family revealed that they were embarrassed, but did not know how to intervene. They were eager to assist but wanted to avoid interrupting the therapy session and thought that the therapists knew how to manage the behavior. They reassured the psychologist that the patient was raised in a Christian home and did not act this way before. Both mother and father denied inappropriate sexual behavior directed at them; however, the mother reported that she thought she had walked into his hospital room while the patient was touching himself with his left hand. Nursing staff subsequently reported that the male patient would occasionally grab them when assisting with transfers. During interviews and direct observation, it was noted that the behavior only occurred with female staff. He never attempted to inappropriately grab male hospital staff.

#### **Results of Functional Analyses**

#### **Define Problem Behavior**

- Inappropriate sexual behaviors: grabbing of PT/OT breasts and buttocks
- Duration: persists until patient redirected or the stimulus (person) is further away from the patient

#### Antecedents

 OT and PT sessions *during therapeutic exercises* (sitting balance—using arm to grab cones, transfers, and other times when therapists were in close proximity)

- When *female* staff were on his left side with reach of his left arm
- Upon interview with nursing staff, he would reportedly grab some of the female nurses during transfers

#### Consequences

- No overt reaction from parents.
- Therapists showed embarrassment (flushed face, nervous laughter), and told patient "no" in casual voice and continued with sessions.

#### Hypothesis

Patient is showing dis-inhibition (poor impulse control) when presented with stimuli of sexual nature. His limited motor control allows him to grab when in close proximity. This is occurring in his two therapies in which he has close contact with OT and PT. It is not happening in SLP because he sits at a desk when working with SLP. He reportedly has engaged in this behavior with female nursing staff during transfers (close proximity). Persons with severe TBI can exhibit poor behavioral control that is expressed with sexual gestures (verbal and nonverbal). The response may represent a form of environmental dependency in which he reacts to stimuli without conscious awareness that he is doing so. These cognitive-behavioral impairments are observed following frontal lobe injury consistent with this patient's history. It is our clinical experience that this behavior can occur among young male TBI survivors and that it commonly resolves during the recovery process.

#### **Treatment Approach**

1. We *involved* and *educated the family* about this symptom. We informed therapists that *family were embarrassed* and had no idea what to do in this situation. They were hoping the therapists would guide them in responding to the behavior. We discussed with both therapists and family that this *behavior can be common* and has nothing to do with who the patient was before his injury. We highlighted that it is likely a transient symptom of his *neurological injury* (i.e., poor impulse control and possible hypersexuality).

- 2. To facilitate managing this behavior, we adopted *response prevention strategies*. Since the antecedents were female staff in close proximity to his left side (good side), we asked dad to hold the patient's left hand when not being therapeutically addressed in "high risk" situations. We also recommended that the patient use his left hand in activities that were incompatible with grabbing of female staff (i.e., holding dad's hand; holding therapy devices, etc.).
- 3. If the behavior occurred, we planned to involve dad or mom in responding to the behavior in a "firm" tone of voice and *redirecting the patient* to appropriate therapeutic tasks. Caution was taken to avoid embarrassing the patient in front of others.
- 4. We asked therapists not to laugh or display behavior that could be perceived as reinforcing (he may have enjoyed that he made them laugh—even though it was a nervous laugh). We asked them to rehearse a firm "no" response and redirect patient to therapeutic activities while avoiding embarrassing the patient (which could escalate a situation).
- 5. We also asked staff to facilitate teaching the patient's father how to transfer him at bedside since he was present every day. This allowed female nursing staff to have an additional male person to assist with transfers. Dad was often on the left side.

#### Results

The strategy was effective, highlighting the importance of identifying antecedents, we prevented the behavior a majority of the time by having the patient engage in a behavior incompatible with grabbing. Dad or male nursing staff handled all transfers throughout the day. Collectively, these served as response prevention techniques, since we prevented the behavior by accurately identifying antecedents. Over the next month, the patient improved neurologically, including improved orientation. As he improved neurologically, the inappropriate grabbing was no longer an issue.

#### Behavior Management Rounds Case 2

#### Complaint

PT and OT reported that a patient attempted to strike them several times with his fists. He had significantly injured one of the OT technicians and she was placed on medical leave due to injury to her hand. Nursing reported similar problems during transfers. The patient would become agitated, but had not hit any of the nursing staff to date.

#### Background

The patient is a 28-year-old male hospitalized for comprehensive rehabilitation for TBI sustained approximately 2 years earlier. Acute records were unavailable, but follow-up neuroimaging revealed significant encephalomalacia resulting in ventriculomegaly. When discharged from acute hospitalization, he received minimal follow-up for problems such as severe spasticity. He received no PT, OT, or speech therapy (ST) as an outpatient. Most of his days were spent sitting in a wheelchair in front of a television at home. He lived with his family due to his inability to live independently. Upon readmission to rehabilitation, he was found to lack spontaneous speech, but would answer some questions with verbal responses if persistently cued. He was oriented to his name, hometown, and date of birth, but gave his age incorrectly (he gave his age at time of injury). He was disoriented in all other spheres due to severe memory impairments. When asked about his reason for hospitalization, he did not know he was in a hospital and would not provide further responses. When asked about his impairments, he did not acknowledge any deficits including the inability to walk or functionally use

his extremities. We felt this patient was unable to develop new memories (anterograde amnesia) which explained his inability to respond accurately to orientation questions and information about his current situation. He also demonstrated significant anosognosia (awareness impairment) which explained his poor understanding of his currently physical and cognitive status. Of note, his family was not present during his rehabilitation stay.

## Psychologist's First Observation/Data Gathering Session

The patient was observed during physical therapy in the main gym. Given the severity of his spasticity, the focal activity was stretching of his legs. During this activity, the patient would attempt to voice "STOP" when stretching began. When he did this, the therapist would respond "we have to stretch your legs." Patient would again voice "STOP!" The therapist continued to stretch him and the patient subsequently began to swing his arms in an attempt to strike the therapist. The therapist was clearly frustrated and displayed this in her tone of voice. The therapist responded that since he did this, he was not going to get to watch television that evening and he would have to go back to the nursing station immediately and miss therapy at that time as punishment. The patient was immediately brought back to the nursing station and not allowed to watch television that evening.

#### **Results of Functional Analyses**

#### **Define Problem Behavior**

• Hitting/Striking of therapists

#### Antecedents

- PT and OT sessions
- Stretching of spastic limbs
- Patient yelled "stop"
- Therapist ignored patient's request to stop stretching him

#### Consequences

- Therapist used frustrated tone to react to patient's behavior
- Stopped therapy session and taken to nursing station
- No television that evening

#### Hypothesis

The patient is severely cognitively impaired (poor memory for new information and disoriented) and shows poor awareness for the nature of his deficits and the importance/relevance of therapy activities. Spasticity is a painful medical condition and the focus of his therapies. In order to facilitate further independence with ambulation, this needs to be addressed therapeutically. However, it appears that his *perception is that he* is being hurt by someone. He wants to stop the painful therapeutic activities when they occur because he does not see the relevance (secondary to anosognosia). Of note, prior to the hitting episode observed, he did engage in a "more appropriate" behavior (i.e., yelling stop) that was "ignored" by his PT.

Patient was never violent with nursing but would yell out loud during transfers. It was suspected that his spasticity resulted in pain during nursing transfers. Since transfers are brief and time-limited activities, the patient did not escalate to violent behavior to stop the painful activity (i.e., transfers) with nursing.

#### **Treatment Approach**

- We educated his physical therapist about the patient's cognitive status and poor awareness for his impairments (thus poor understanding of his therapy sessions). We asked the therapist to "remind" the patient of the purpose of his therapy sessions at the start of each session and beginning of new exercises in each session. This facilitated awareness of his physical impairments and relevance of therapy and exercises. Due to his memory impairments, he needed the repeated cues to facilitate remembering why he needed therapy.
- We also educated his therapists about the importance of nonverbal communication with patients. We asked her to avoid using negative nonverbal

communication in working with this patient. He would likely respond to those cues more so than lengthy verbal directions/explanations.

- 3. We further educated the therapists to be responsive (i.e., reinforce: a consequence that results in a behavior likely to occur again) to the patient's appropriate behaviors (i.e., verbal communication of yelling "stop") when he wanted to stop painful exercises. We asked her to stop if he reported pain or yelled "stop." By responding (reinforcing) his more appropriate response, we hopefully avoided him escalating to a hitting response to stop the painful activity.
- 4. We also educated the therapist about the appropriate use of punishers (something that occurs that decreases the likelihood of a behavior occurring again) in behavior management. Although she felt that stopping therapy and precluding TV that night was a form of punishment, she actually reinforced the hitting behavior. Since the goal of his behavior (i.e., hitting) was to stop the painful activity, the ending of his therapy reinforced (increased the likelihood that it will occur again) the hitting behavior. A concern was that he would go directly to hitting behaviors next time, since it was effective in stopping the painful activity moreso than yelling "stop." We emphasized the importance of immediate consequences for influencing behavior (to increase or decrease it) rather than remote events. Although the therapist recommended the patient not be allowed to watch television that evening, it was too remote in time to influence his behavior. Further, he would likely not remember the situation that resulted in loss of television privileges due to his severe memory impairments.
- 5. We recommended *other strategies* to help the patient engage in therapeutic activities that might be painful. We asked her to consider a *distraction technique*. For this patient, we used a kitchen timer and chose the duration of the painful stretching activity (e.g., 5 s) *with him.* We recommended using that time interval regardless for the first activity. We recommended the patient count and look at the timer during the painful activity. For the next set, we asked the patient to increase the time any

amount as long as it is longer than the first time interval (e.g., 6 s). By giving the patient some level of control, we hoped he would engage in necessary therapeutic activities.

6. Finally, we asked his PT/OT to engage in nonpainful activities with the patient so that they avoided always being associated with painful activities.

#### Results

A combination of these strategies worked; however, the therapist was already experiencing negative feelings towards the patient that was challenging for her to overcome. She had little experience in dealing with this type of emotional reaction to her patients in her formal training. After lengthy discussion, we attended therapy sessions with her to model reinforcement of desirable behaviors (i.e., verbal praise) and demonstrate use of distraction techniques. She developed some co-treatment sessions with the SLP, so that the patient was engaged in distracting tasks during some of her therapy sessions. As the patient's spasticity improved with serial stretching and casting, he reported less pain during these activities. No further incidents of violence occurred in therapy and the yelling stopped with nursing.

#### Behavior Management Rounds Case 3

#### Complaint

Elopement in Progress (current issue) and Noncompliance with Vitals (subsequent complaint by nursing).

#### Background

Patient was an 18-year-old male who sustained a severe TBI in a motor-vehicle collision. His initial GCS was 4T in the emergency department and neuroimaging revealed intraparenchymal contusions in the left frontal, parietal, and temporal regions. He was admitted for comprehensive rehabilitation approximately 3 weeks post-injury. Upon admission, he was ambulatory and could move all extremities. He was nonverbal with flat affect. His Mississippi Aphasia Screening Test Score was 0/100 with a suspicion of global aphasia consistent with the site of his brain injury. He did not follow single-step verbal commands for any staff member. However, he would follow most visual commands demonstrated to him that were simple in complexity. At times, he would attempt to follow visual commands but moved incorrectly (suspected apraxia). On occasion, he was observed to pick up a telephone and say hello despite being nonverbal in all other situations. He was also noted to hit buttons on the remote control when lying in bed and looking at the TV. He did not interact with staff. He often held his head down when others tried to engage him. His GOAT score was -8 due to non-responding to orientation items or visual stimuli indicating choices for orientation items. His agitated behavior scale score was 27 (high degree of motor restlessness and inattention). During his first few days on the unit, he was noted to not eat much on meal trays brought to his room. Later, he was found to have trouble manipulating utensils/objects consistent with an apraxic presentation that is also consistent with the site of his injury. Although he was physically capable of toileting independently, he would have accidents or would be noted to urinate in the trash can in his room.

# Psychologist's Observation/Data Gathering Session

The psychologist was on the unit when the patient was observed in the elevator lobby of the locked unit with approximately 10 security and staff members surrounding the patient. During this incident, the patient was yelling un-intelligible phrases and backing into the lobby wall. As staff stood around, he would hit the doors of the elevators but did not hit the call button. Although the elevators required a passcode to active the call button, an elevator opening on the floor would allow an exit. An elevator eventually opened and the patient got on the elevator. Staff held the ele-

vator on the floor. Patient did not attempt to hit other floor buttons. He would pace the interior of the elevator and occasionally hit the walls. He never attempted to hit staff. When an alarm sounded indicating the elevator had been held on the floor too long, the patient got off the elevator and went back to his room. Security and floor staff backed off to avoid frightening the patient. When he got back to his room, a nursing assistant (on loan from another floor and unfamiliar with the patient) was waiting for him. Psychologist followed him into his room and observed nursing assistant state in a frustrated tone "Are you going to cooperate with me now and let me take your vitals?" (while standing with her hands on her hip) The patient sat down on his bed and held his head down. He did not look at the nursing assistant. He did not cooperate with her vitals exam. Psychologist learned that this was the situation taking place prior to his elopement. He did not "cooperate" with vitals exam, which led to increased frustration by the nursing assistant trying to finish her rounds.

#### **Results of Functional Analyses**

#### **Define Problem Behavior**

- Elopement from unit
- "Uncooperative" with vitals exam

#### **Situational Antecedents**

- Vitals exam at bedside
- Unfamiliar staff
- Verbal instruction used to comply with vitals assessment
- Frustration in voice of clinical provider

#### **Patient Antecedents**

- Receptive language problem (poor comprehension of verbal statements made to him).
- Apraxia (inability to use objects accurately and consistently). It is our clinical experience that automatic behaviors are most preserved (picking up phone and saying hello; holding remote control). Many staff observed him pick up the phone when it rang or hit buttons on remote and assumed he was more capable of

other complex motor behaviors. His apraxia was more pronounced in tasks requiring use of utensils (explaining his initial incomplete meals), going to urinal accurately and consistently, using elevator buttons (knowing how to call the elevator, etc.)

- Acute confusional state (disorientation, agitation, inattention, etc.).
- Lability—more pronounced in delirium/ confusion and likely intensified his frustrated response to vitals examination—also observed with frontal lobe lesions.

#### Consequences of His Being Uncooperative

- Patient escalated quickly in situation where he may have perceived someone being upset with him. Although he did not understand the content of the nursing assistant's instructions during vitals exam (due to his aphasia), we believe that he was capable of interpreting nonverbal communication (such as tone of voice) as the right hemisphere had no lesion abnormalities. Her frustrated tone of voice (which likely escalated with continued noncompliance) may have facilitated his exaggerated response (frustration) to a situation he did not understand. To escape someone communicating negatively with him, he left the room in an agitated state resulting in the elopement attempt.
- An important note, his elopement attempt ceased when the elevator noise was uncomfortable to him and he went back to his room. The elevator remaining on the floor in combination with an aversive elevator sound prompted the patient to exit the elevator and return to the floor.

#### **Hypothesis**

Patient likely did not understand the instructions of nursing when asking him to cooperate with vitals exam. He escalated due to nonverbal communication style of staff suggesting anger and frustration.

#### **Treatment Approach**

- 1. *Involve* and *educate* all staff about patient's cognitive deficits.
- 2. Adopt *response prevention strategies*. Help staff with communication style and imple-

mentation of nonverbal communication with him. For example, using a calm voice and letting patient see staff face when interacting with him helped to get his attention. Use of gestures while speaking to him calmly helped to get his cooperation.

 Educate staff about cognitive and behavioral deficits of this patient and provide strategies for maximizing patient cooperation given the patient's unique neurologic profile. Ask staff to pass information along from shift to shift.

#### Results

The strategies worked. Staff implemented strategies recommended with the patient. Patient continued to make progress and resolved from his acute confusional state with no further episodes of elopement.

#### **Concluding Remarks about Cases**

Although each of these cases highlight patient scenarios that are challenging and potentially dangerous to patients and staff, they reference the importance of an individualized treatment approach (*ideographic*) rather than a uniform (nomothetic) response (i.e., such as giving sedating medications to all violent patients). Figure 3 below highlights the complexity of behavioral issues following TBI. Their manifestation can often result from an interaction of environment with a specific patient's impairments occurring in the context of brain injury. Although several books highlight information about common behavioral issues and provide education about brain injury, they do not emphasize all of the elements necessary to understand a particular behavioral issue for a specific patient. To do this, it is necessary to conduct a functional analysis including patient TBI characteristics (e.g., cognitive functioning, awareness, etc.). Functional analysis promotes enhanced understanding by considering patient and environmental contributions to the problem behavior. This improved understanding can lead to the development of new treatment strategies.

Key components to implementing behavior management interventions with TBI patients



**Table 3** General recommendations for population specific groups

Target group	General recommendations
Team members	Be consistent
	Do not expect a quick fix
	Everything is relative
	Educate yourself about course of illness
	Measure behavior (small changes are improvement)
	Communicate with everyone
	Debrief with the Team psychologist neuropsychologist
	Be aware of burnout issues
TBI survivors	Do not talk down to patient
	Focus on behavior not person
	Undesirable behavior not undesirable
	person
	Monitor your own response (emotions, behavior) to him her
	Explain course of illness and do so repeatedly if necessary
Family	Educate
members	Schedule family conferences
	Schedule regular team meetings
	Be aware of unspoken family
	embarrassment
	Identify and intervene with ineffective coping strategies
	Explain importance of consistency (generalizability)

include understanding the course of acute recovery following brain injury, knowing patient-specific impairments, and identifying how the environment contributes to challenging behavior issues following brain injury. Table 3 highlights common pointers for rehabilitation staff in working with team members, TBI survivors, and family members.

#### Appendix: Functional Analysis Sample Worksheet



#### References

- Harris, J. K., Godfrey, H. P., Partridge, F. M., & Knight, R. G. (2001). Caregiver depression following traumatic brain injury (TBI): A consequence of adverse effects on family members? *Brain Injury*, 15, 223–238.
- Kreutzer, J. S., Gervasio, A. H., & Camplair, P. S. (1994). Patient correlates of caregivers' distress and family functioning after traumatic brain injury. *Brain Injury*, 8, 211–230.
- Jackson, D., Turner-Stokes, L., Murray, J., Leese, M., & McPherson, K. M. (2009). Acquired brain injury and dementia: A comparison of carer experiences. *Brain Injury*, 23(5), 433–444.
- Warchausky, S., Kewman, D., & Kay, J. (1999). Empirically supported psychological and behavioral therapies in pediatric rehabilitation of TBI. *The Journal of Head Trauma Rehabilitation*, 14, 373–383.
- Carnevale, G. J., Anselmi, V., Johnston, M. V., Busichio, K., & Walsh, V. (2006). A natural setting behavior management program for persons with acquired brain injury: A randomized controlled trial. *Archives of Physical Medicine and Rehabilitation*, 87, 1289–1297.
- Beaulieu, C., Wertheimer, J. C., Pickett, L., Spierre, L., Schnorbus, T., Healy, W., et al. (2008). Behavior management on an acute brain injury unit: Evaluating the effectiveness of an interdisciplinary training program. *The Journal of Head Trauma Rehabilitation*, 23(5), 304–311.
- Blosser, J. L., & DePompei, R. (1991). Preparing education professionals for meeting the needs of students with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 6, 73–82.
- Powers, J. S., Gordon, S. M., & Elgin, J. (2008). Disruptive behaviors in dementia: Promoting nonpharmacologic intervention with staff education. *Federal Practitioner*, 25(3), 28–39.
- Feeney, T. J., Ylvisaker, M., Rosen, B. H., et al. (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*, 61–75.
- Sandel, M. E., & Mysiw, W. J. (1996). The agitated brain injured patient. Part 1: Definitions, differential diagnosis, and assessment. *Archives of Physical Medicine and Rehabilitation*, 77(6), 617–623.
- Weir, N., Doig, E. J., Fleming, J. M., Wiemers, A., & Zemljic, C. (2006). Objective and behavioural assessment of the emergence from post-traumatic amnesia (PTA). *Brain Injury*, 20(9), 927–935.
- Mysiw, W. J., Corrigan, J. D., Carpenter, D., & Chock, S. K. L. (1990). Prospective assessment of posttraumatic amnesia: A comparison of the Galveston Orientation and Amnesia Test and the Orientation Group Monitoring System. *The Journal of Head Trauma Rehabilitation*, *5*, 65–72.

- Corrigan, J. D., Mysiw, W. J., Gribble, M. W., & Chock, S. K. (1992). Agitation, cognition and attention during post-traumatic amnesia. *Brain Injury*, 6(2), 155–160.
- Forrester, G., Encel, J., & Geffen, G. (1994). Measuring post-traumatic amnesia (PTA): An historical review. *Brain Injury*, 8(2), 175–184.
- Kadyan, V., Mysiw, W. J., Bogner, J. A., Corrigan, J. D., Fugate, L. P., & Clinchot, D. M. (2004). Gender differences in agitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 83(10), 747–752.
- Nakase-Thompson, R., Sherer, M., Yablon, S. A., Nick, T., & Trzepacz, P. T. (2004). Acute confusion following traumatic brain injury. *Brain Injury*, 18(2), 131–142.
- Brooke, M. M., Patterson, D. R., Questad, K. A., Cardenas, D., & Farrel-Roberts, L. (1992). The treatment of agitation during initial hospitalization after traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 73(10), 917–921.
- van der Naalt, J., van Zomeren, A. H., Sluiter, W. J., & Minderhoud, J. M. (2000). Acute behavioural disturbances related to imaging studies and outcome in mild-to-moderate head injury. *Brain Injury*, 14(9), 781–788.
- Levin, H. S., & Grossman, R. G. (1978). Behavioral sequelae of closed head injury. A quantitative study. *Archives of Neurology*, 35(11), 720–727.
- Fugate, L. P., Spacek, L. A., Kresty, L. A., Levy, C. E., Johnson, J. C., & Mysiw, W. J. (1997). Definition of agitation following traumatic brain injury: I. A survey of the Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation. Archives of Physical Medicine and Rehabilitation, 78(9), 917–923.
- Mysiw, W. J., Jackson, R. D., & Corrigan, J. D. (1988). Amitriptyline for post-traumatic agitation. *American Journal of Physical Medicine & Rehabilitation*, 67(1), 29–33.
- Pryor, J. (2005). What cues do nurses use to predict aggression in people with acquired brain injury? *Journal of Neuroscience*, 37(2), 117–121.
- 23. Dolan, R. J. (2002). Emotion, cognition, and behavior. *Science*, 298(5596), 1191–1194.
- Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., & Damasio, A. R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*, 269(5227), 1115–1118.
- Tonkonogy, J. M. (1991). Violence and temporal lobe lesion: Head CT and MRI data. *Journal of Neuropsychiatry & Clinical Neurosciences*, 3(2), 189–196.
- 26. Patrick, P. D., & Hebda, D. W. (1997). Management of aggression. In J. Leon-Carrion (Ed.), *Neuropsychological rehabilitation: Fundamentals, innovations, and directions* (pp. 431–451). Delray Beach: St Lucie Press.
- Silver, J. M., & Yudofsky, S. C. (2004). Aggressive disorder. In J. M. Silver, S. C. Yudofsky, & R. E. Hales (Eds.), 2004 Neuropsychiatry of traumatic brain injury (pp. 313–353). Washington DC: American Psychiatric Press.
- Sanchez-Navarro, J. P., Martinez-Selva, J. M., & Roman, F. (2005). Emotional response in patients with frontal brain damage: Effects of affective valence and information content. *Behavioral Neuroscience*, *119*(1), 87–97.
- Demark, J., & Gemeinhardt, M. (2002). Anger and it's management for survivors of acquired brain injury. *Brain Injury*, 16(2), 91–108.
- Grafman, J., Vance, S. C., Weingartner, H., Salazar, A. M., & Amin, D. (1986). The effects of lateralized frontal lesions on mood regulation. *Brain*, *109*(Pt 6), 1127–1148.
- Phillips, M. L., Drevets, W. C., Rauch, S. L., & Lane, R. (2003). Neurobiology of emotion perception I: The neural basis of normal emotion perception. *Biological Psychiatry*, 54(5), 504–514.
- 32. Stuss, D. T., Binns, M. A., Carruth, F. G., Levine, B., Brandys, C. E., Moulton, R. J., et al. (1999). The acute period of recovery from traumatic brain injury: Posttraumatic amnesia or posttraumatic confusional state? *Journal of Neurosurgery*, 90(4), 635–643.
- Alderman, N., Knight, C., & Morgan, C. (1997). Use of a modified version of the Overt Aggression Scale in the measurement and assessment of aggressive behaviors following brain injury. *Brain Injury*, 11(7), 503–523.
- Corrigan, J. D. (1989). Development of a scale for assessment of agitation following traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 11(2), 261–277.
- Fields, RB, Coffey, CE (Eds.) (1994). Traumatic brain injury. In: *Textbook of geriatric neuropsychiatry*. Washington, DC: American Psychiatric Press, pp. 479–506.
- Brooks, N., Campsie, L., Symington, C., Beattie, A., & McKinlay, W. (1986). The five year outcome of severe blunt head injury: A relative's view. *Journal of Neurology, Neurosurgery & Psychiatry*, 49(7), 764–770.
- 37. Bogner, J. A., Corrigan, J. D., Fugate, L., Mysiw, W. J., & Clinchot, D. (2001). Role of agitation in prediction of outcomes after traumatic brain injury. *American Journal of Physical Medicine & Rehabilitation*, 80(9), 636–644.
- Brooke, M. M., Questad, K. A., Patterson, D. R., & Bashak, K. J. (1992). Agitation and restlessness after closed head injury: A prospective study of 100 consecutive admissions. *Archives of Physical Medicine* and Rehabilitation, 73(4), 320–323.
- Eslinger, P. J., Grattan, L. M., & Geder, L. (1995). Impact of frontal lobe lesions on rehabilitation and recovery from acute brain injury. *Neurorehabilitation*, 5, 161–185.

- Nott, M. T., Chapparo, C., & Baguley, I. J. (2006). Agitation following traumatic brain injury: An Australian sample. *Brain Injury*, 20(11), 1175–1182.
- Perlesz, A., Kinsella, G., & Crowe, S. (2000). Psychological distress and family satisfaction following traumatic brain injury: Injured individuals and their primary, secondary, and tertiary carers. *The Journal of Head Trauma Rehabilitation*, 15(3), 909–929.
- 42. Orsillo, S. M., McCaffrey, R. J., & Fisher, J. M. (1993). Siblings of head-injured individuals: A population at risk. *The Journal of Head Trauma Rehabilitation*, 8, 102–115.
- Lequerica, A. H., Rapport, L. J., Loeher, K., Axelrod, B. N., Vangel, S. J., Jr., & Hanks, R. A. (2007). Agitation in acquired brain injury: Impact on acute rehabilitation therapies. *The Journal of Head Trauma Rehabilitation*, 22(3), 177–183.
- 44. Lombard, L. A., & Zafonte, R. D. (2005). Agitation after traumatic brain injury: Considerations and treatment options. *Archives of Physical Medicine and Rehabilitation*, 84(10), 797–812.
- Corrigan, J. D., & Mysiw, W. J. (1988). Agitation following traumatic head injury: Equivocal evidence for a discrete stage of cognitive recovery. *Archives of Physical Medicine and Rehabilitation*, 69(7), 487–492.
- 46. Zafonte, R. D. (1997). Treatment of agitation in the acute care setting. *The Journal of Head Trauma Rehabilitation*, 12(2), 78–81.
- 47. Larson, E. B., & Zollman, F. S. (2010). The effect of sleep medications on cognitive recovery from traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 25(1), 61–67.
- Hem, E., Steen, O., & Opjordsmoen, S. (2001). Thrombosis associated with physical restraints. *Acta Psychiatrica Scandinavica*, 103(1), 73–75.
- Laursen, S. B., Jensen, T. N., Bolwig, T., & Olsen, N. V. (2005). Deep venous thrombosis and pulmonary embolism following physical restraint. *Acta Psychiatrica Scandinavica*, *111*(4), 324–327.
- Willis, T. J., & LaVigna, G. W. (2003). The safe management of physical aggression using multi-element positive practices in community settings. *The Journal* of *Head Trauma Rehabilitation*, 18(1), 75–87.
- 51. Nakase-Richardson, R., Evans, C. C., Thors, C., Constantinidou, F., Wertheimer, J., et al. (2009). Evaluation of a multidisciplinary program intervention for managing cognitive and behavioral sequelae following brain injury. *Archives of Physical Medicine and Rehabilitation*, 90(10), e34.
- 52. Ferster, C. B. (1965). Classification of behavioral pathology. In L. Krasner & L. P. Ullmann (Eds.), *Research in behavior modification* (pp. 6–26). New York: Holt, Rinehard, & Winston.
- Goldfried, M. R., & Pomeranz, D. M. (1968). Role of assessment in behavior modification. *Psychological Reports*, 23, 75–87.

# Rehabilitation of Memory Problems Associated with Traumatic Brain Injury

# Angelle M. Sander and Laura M. van Veldhoven

#### Abstract

Memory problems are reported by the majority of persons with traumatic brain injury (TBI) and have a negative impact on their everyday independence and functioning. Research shows that training in compensatory memory strategies is effective for improving memory functioning in persons with TBI; however, no one strategy can meet the needs of all people with TBI. The current chapter provides an overview of the type of memory problems typically observed in people with TBI, as well as a review of the evidence for cognitive rehabilitation of memory. A systematic, individualized approach to training in compensatory memory strategies is then presented with an emphasis on ecological validity and integrating the preferences and resources of people with TBI and their caregivers. Two case examples are presented to illustrate the implementation of this approach to memory strategy training.

#### Keywords

Memory • Memory rehabilitation • Compensatory memory strategies • Memory training

A.M. Sander, Ph.D. (🖂) Baylor College of Medicine and Harris Health System, Houston, TX, USA

Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA e-mail: Angelle.Sander@memorialhermann.org

L.M. van Veldhoven, Ph.D. Baylor College of Medicine, Houston, TX, USA

# **Background and Purpose**

Impaired memory has long been known as one of the most frequently reported changes following traumatic brain injury (TBI) [1]. Significant changes in memory after TBI have been documented on neuropsychological measures, subjective measures, quality of life measures, and both short-term and long-terms outcomes [2–5]. Impaired memory has wide-reaching implications for community integration and participation, as it can disrupt ability to find and maintain work, live independently, manage household chores, and manage finances. Memory impairment can also have an impact on social activities, since forming and maintaining relationships depends partially on ability to make and keep dates for social activities, recall information about others' likes and dislikes, and discuss previous shared interactions. Poor performance on objective measures of memory after TBI has been associated with increased disability, unemployment, major depression, and decreased social autonomy [6–9]. Effective interventions for memory difficulties after TBI are crucial for improving quality of life and community participation after TBI.

The purpose of the current chapter is to provide clinical neuropsychologists with an understanding of the rehabilitation of memory impairments after TBI. The chapter will begin with an overview of the types of memory problems typically observed in people with TBI, as well as the way that these problems manifest in everyday functioning. This will be followed by a review of the evidence base for rehabilitation of memory after TBI. The second half of the chapter will focus on clinical implementation of training in compensatory memory techniques, including practical concerns when conducting this type of training. As the evidence base to date relates primarily to rehabilitation of memory in a post-acute setting, this chapter will focus on rehabilitation of memory impairments in individuals who have emerged from posttraumatic amnesia (the period of disorientation and inability to form continuous day-to-day memory that often accompanies the acute stage of recovery from TBI). Therefore, memory disturbance associated with acute confusion will not be addressed in this chapter. Readers interested in this topic can refer to the chapter by Mark Sherer and Joe Giacino in this volume.

# Typical Types of Memory Problems After TBI

Severe retrograde amnesia is not a common problem following TBI. The person with injury may be unable to recall events immediately preceding the injury, or sometimes during the entire day to several days before injury; however, recall of information that was well-learned prior to injury is typically intact [10]. This includes autobiographical information, life events, well-learned facts (such as historical information) and procedures. In contrast, anterograde amnesia is common following TBI. People with TBI can have difficulty with the acquisition/encoding [11], storage [12], and/or retrieval [13-15] of new information. Many people with TBI are able to retain information that they did encode or learn, even after a delay; however, the overall amount of material that they were able to learn is often impaired, leading to deficient recall compared to normative data. At least one study suggested that impaired acquisition accounted for poor retrieval of information, as retrieval was equivalent for people with TBI and healthy controls, after equating the groups on learning acquisition performance [16]. Another study, conducted with veterans with TBI, found three clusters of people with TBI regarding memory impairment: a consolidation deficit cluster with impaired overall learning and recall, but adequate retention of the information learned; a retention deficit cluster with impaired free and cued recall of information learned; and a retrieval and control deficit cluster, showing impaired free recall of information, improvement with cued recall, presence of intrusion errors, and presence of false positive errors during auditory recognition memory [17]. These performance clusters were considered to be consistent with the theoretical memory constructs of consolidation, retention, and retrieval.

Learning and recall difficulties after TBI are often related to impairments in attention, organization, and/or processing speed. For example, someone with impaired attention may be distracted by competing internal or external information when attempting to learn material. Persons with TBI have been shown to have increased susceptibility to interference during recall [18] and to have difficulty distinguishing between intrusions and target material [19, 20]. Difficulty distinguishing between target words and distracters during auditory recognition memory has also been reported [21]. Impaired organization can impact learning by making it difficult for someone to impose structure to assist with learning, such as categorizing information into related groups or relating it to information that they already know. For example people with TBI have been shown to have difficulty making spontaneous use of semantic organization strategies, although they are able to process information semantically and benefit when provided with semantic cues [11, 19, 22]. A person with impaired processing speed will likely have difficulty keeping up with information that is presented, making it difficult to encode it for later use.

Impairments in encoding or retrieval often occur for visual/spatial information, as well as for verbal information. This can lead to problems in finding their way around in everyday life [23]. Modality-specific memory impairments (e.g., intact recall of verbal information with impaired recall of visual information, or vice-versa) are rare following TBI, due to the diffuse axonal injury and coup-contrecoup injuries that are the hallmark of TBI and that often impact bilateral frontal and temporal lobes; however, they can occur and have been noted to particularly impact acquisition and storage [24].

One of the most common memory difficulties following TBI is impaired prospective memory [10, 25, 26]. Prospective memory refers to the ability to remember to perform a task in the future, such as remembering to call for lunch in 15 min or to call for an appointment on a certain day [27, 28].

# Manifestation of Memory Problems in Everyday Life

While formal assessment can provide a partial picture of memory functioning, it cannot provide a complete picture of the everyday strengths and weaknesses that people with TBI exhibit. Memory performance in a structured testing situation may not correspond with everyday memory functioning. Clinical experience has indicated that people who perform well in the structured testing situation may have difficulty learning and recalling information in less structured, daily settings. Conversely, people who perform poorly on traditional memory tests may use environmental cues or compensatory strategies that enable successful memory functioning in daily life. Therefore, an important part of any memory assessment should be informal assessment of the perceptions of the person with injury regarding their everyday memory functioning. Obtaining a family member or close other's perception of the person with injury's memory abilities can also be helpful, due to the possibility of impaired awareness of deficits in the person with injury. Research has indicated that people with TBI are often inaccurate reporters of memory problems [29, 30].

Our clinical and research experiences in the area of TBI have indicated that memory problems typically fall into one of the following categories: difficulty keeping track of belongings; forgetting what needs to be done (e.g., appointments to attend, chores to complete, questions to ask of a physician or teacher); forgetting how to get to places; forgetting what people have told them or what they have learned (including peoples' names); and forgetting how to do something/procedures. Prospective memory failures are the most common memory problems reported by persons with TBI (and their family members) in daily life [10, 26]. These memory failures involve forgetting to keep track of tasks that need to be accomplished at a certain point in time, such as when to take medication, when to pick children up from activities, and when to pay bills. This type of memory difficulty can be particularly devastating for persons with TBI, since being independent relies on the ability to remember when to initiate activities. It also has health and safety consequences, since forgetting to take medications at a certain time is a manifestation of impaired prospective memory.

# Review of Interventions for Memory Problems

Interventions for memory problems can be classified as restorative or compensatory, based on their aims [10]. The goal of restorative treatments is to restore memory functioning through systematic training and repetitive drills. This method of intervention is based on the theory that memory abilities can be restored through practice. The use of computerized games or programs designed to "exercise" the memory skills of the person with injury is an example of a treatment targeting restoration. Unfortunately, there is minimal empirical evidence to support the effectiveness of interventions aimed at restoring memory.

A second approach to improving memory after brain injury involves training in the use of compensatory strategies to circumvent existing memory problems [10]. This approach is based on the premise that memory function does not have to be restored in order for functional memory to improve. Instead, performance on everyday memory tasks can improve if the person learns effective strategies to serve in place of lost memory abilities. Compensatory strategies used can be classified as internal or external. Internal strategies include the use of visual imagery techniques, rehearsal and repetition, chunking or organization strategies, and self-instructional strategies. Training in use of internal memory strategies has been shown to be effective for improving performance on neuropsychological tests of memory [31, 32]; however, generalization to functional memory tasks is not clear. Furthermore, the use of internal strategies may tax the cognitive resources of persons with TBI, who often have concomitant impairments in attention, information processing speed, and executive functions. For this reason, the use of internal strategies may work best for those with mild memory impairment.

The use of external memory aids can circumvent some of the difficulties inherent in use of internal strategies. They involve the use of an external aid to remind people to perform tasks, attend appointments, etc. Studies investigating the use of external memory strategies can be classified into two broad categories: paper-based aids and electronic aids. The majority of paper-based interventions center on the utilization of a journal, notebook, or planner for compensation [33–35]. Use of memory notebooks or daily journals has been shown to be effective for reducing everyday memory failures [36, 37]. One study indicated that training in memory notebook usage was superior to the use of internal rehearsal strategies, particularly for persons with more severe memory impairment [36]. Ownsworth and McFarland found that addition of self-instructional training to the use of a daily diary resulted in a reduction in everyday memory failures.

While use of memory notebooks and other paper-based aids are readily available at a relatively low-cost, they are not optimal for all persons with TBI. Many people forget to use the strategy, requiring frequent cueing by family members. The time required to train in the use of a memory notebook can be quite intensive, and consistent repetition is often needed [37]. Based on their systematic reviews, Cicerone and colleagues concluded that external strategies are most effective for persons with mild memory impairment, and often do not work for persons with severe memory impairment.

In recent years, the rapid increase in portable technology has offered the possibility of using electronic external memory aids. For example, the use of voice recorders or mobile phones has been investigated as a means of compensating for memory problems. These devices have the benefit of being able to provide external cues, such as alarms, to remind a person of when to complete a task or attend an appointment. While many of the electronic aids have been investigated using case study designs and small sample sizes of less than 20 participants, the results of these preliminary investigations indicate that these technologies hold promise as memory compensation strategies [38–42]. Such technologies have shown success in helping people with TBI to meet everyday memory goals (e.g., remembering to take medications, remembering tasks to be completed) and remembering therapy goals.

In summary, a variety of compensatory memory strategies have been found to be successful for persons with brain injury. Based on their systematic reviews of the literature on cognitive rehabilitation, Cicerone and colleagues concluded that memory strategy training, using internalized or externalized strategies, is a practice standard for persons with mild memory impairment due to TBI [43–45]. They recommended use of external strategies, with direct application to functional activities, as a practice guideline for people with severe memory deficits after TBI or stroke. This latter recommendation was made because people with severe memory deficits often have difficulty learning to use strategies, and also forget to use those that they learned. They also have difficulty generalizing strategies learned for one task to another task. Therefore, learning a very specific strategy to help them with a specific functional task promotes greater success.

# Special Considerations for Memory Strategy Training

While the concept of training a person to use a compensatory memory strategy may appear to be common sense, there are some special issues to consider. Attention to these issues can maximize success of the person with TBI in learning the strategy and applying it to functional activities in their daily lives.

# Use of Ecologically Valid Training Tasks

The importance of using ecologically valid tasks when training in use of compensatory memory strategies has been emphasized [43, 44, 46]. Ecologically valid training tasks imply that the strategies used to instruct the individual on how to achieve a target behavior have direct relevance for his or her everyday life. In a review of instructional techniques to be used with neurogenic memory impairments, 27 % of the 51 studies reviewed by Ehlhardt and colleagues utilized ecologically valid tasks, and of these studies, 100 % reported positive findings [46]. Further, their review suggested that an individual's learning is facilitated when the selected tasks or information has inherently functional value. Simply put, persons with brain injury are more likely to consistently use memory strategies that they perceive as being relevant to real-world goals and tasks. Scherer and colleagues have emphasized the importance of matching compensatory technologies for cognitive difficulties with the specific needs and preferences of users [47]. For example, younger people may be more comfortable using portable electronic/digital technology, such as mobile phones and iPads. Some older people may be uncomfortable with any type of computerized technology. These comfort levels must be accounted for when choosing a memory strategy.

In partial response to the need to use ecologically valid training tasks, the concept of contextualized rehabilitation for persons with brain injury has gained increasing attention in recent years [48, 49]. Contextualized rehabilitation refers to treatments provided within the daily context of the individual. While traditional rehabilitation occurs in a treatment facility, often using simulated real-world tasks, contextualized rehabilitation is incorporated into the real-world everyday activities of the person with injury. In this approach, there is an emphasis on arranging the environment to help the person with injury to become more successful in everyday activities. Training of significant others, including parents and coworkers, is an important part of this approach. Training persons with TBI in the use of compensatory strategies (digital technology) in the home setting was conducted by Gentry with positive impact on everyday memory tasks and community integration outcomes [50]. The conduct of compensatory memory training in the home and community settings appears to hold promise as a means of increasing successful use of memory strategies in everyday life; however, the comparative effectiveness of contextualized training relative to standard training has yet to be investigated.

# Involvement of Caregiver or Significant Other in Training

As indicated in the section on interventions, training in the use of memory strategies can sometimes require much repetition and can tax the cognitive resources of people with TBI. In addition, the generalizability of the training to other daily tasks and situations is unclear. Inclusion of a caregiver or significant other in the training can help to circumvent these problems. Caregivers can provide reminders for the person with TBI to use the memory strategy regularly, help them to identify situations in which the strategy is needed, and assist with application of the strategy in new situations. Caregivers should be encouraged to initially provide regular prompts to use the strategy. These prompts can be faded over time as the person becomes more independent in using the strategy. Caregivers should also be trained to assist the person with injury in practicing the strategy frequently, in all situations in which it might be helpful. This consistent practice can result in the strategy use becoming habit, which can gradually lead to increased independence for the person with injury. Caregivers can also serve as an important source of reinforcement and encouragement, particularly during the initial stages of learning a strategy, when the person with injury may have frequent failures that may tempt them to abandon the strategy. Finally, caregivers are in a unique position to help the person with injury to identify the situations in which memory strategies are needed, as they are often intimately familiar with their daily routines and responsibilities. They can also be helpful in identifying potential obstacles to strategy use, so that the therapist can assist with problem-solving.

#### **Emphasis on Process**

Even the most individualized, ecologically valid memory strategy will not work for every memory problem that a person encounters in their daily lives. This is the problem with some traditional approaches used in rehabilitation programs, where a single compensatory strategy (e.g., memory notebook usage) is taught to all clients with TBI. People are most likely to have success with daily memory functioning if they (and their caregivers) learn a process for systematically approaching everyday memory problems. This process can then be applied to novel situations, where the particular memory strategy that they learned in rehabilitation may not work.

Training clients to ask themselves the following questions when they approach a new situation can be helpful: (1) "Will I have anything that I will need to remember in this situation?" (2) "What kind of information will I need to remember in this situation?" (3) "Will the memory strategy that I usually use work in this situation?" or "What things might get in the way of me using a strategy to remember information in this situation?" Teaching the person with injury and caregiver to systematically ask these questions when approaching new situations can help them become accustomed to solving new memory problems on their own, beyond the end of therapy. Therapists can help teach this approach through use of role plays and verbal problemsolving. An example is that the person may need to attend a doctor's appointment on a certain day. He may identify that he will need to ask the doctor questions about the side effects of a medication that he has been taking. The therapist may ask the person to consider whether the strategy he typically uses will work in this situation. Assuming that the person uses a memory notebook or electronic organizer, the person could be prompted to enter the questions into this tool. The therapist can then help him to identify potential obstacles to using the strategy. For example, he may carry the notebook or organizer in a bag and may forget to look at it while with the doctor. The therapist could help him to plan to carry the notebook or organizer in his hands. Another potential obstacle is that he could become distracted by the doctor's questions and forget to use the strategy. The therapist could help him to problem solve solutions, such as placing a sticker on the front of the notebook or organizer to remind him to look inside for the question. Alternatively, he might periodically ask himself if there is anything he needs to ask the doctor, in order to cue himself to look at the notebook. Obviously, these solutions will not work for everyone and may not work the first time. The important thing is to engage the person with TBI in the process of systematically planning and solving everyday memory problems, in the variety of situations in which they might occur.

# A "How-to" Guide for Memory Compensation

In our experience, there are several steps necessary for maximizing successful use of compensatory memory strategies and for promoting effective generalization. These steps are described below. The steps begin following a comprehensive formal neuropsychological assessment of memory.

# Step 1: Gathering of In-Depth Information on Who, What, When, Where, and How

Moving straight from neuropsychological test performance to recommending a memory strategy is not likely to be successful. As with all people, persons with TBI vary in the extent to which they used memory strategies before injury, their everyday memory needs, their comfort level in using certain strategies, and their resources to support strategy use. Clinicians must have a comprehensive understanding of all of these issues in order to successfully train a person to use a memory strategy. We have seen the frustrating and disappointing outcome of a memory compensation strategy that was poorly developed due to inadequate information gathered by the clinician. Information that is important to memory compensation selection and development can fall into several categories, including:

- Specifics about the memory problem: the who, what, when, where, and how
- Reactions, both positive and negative, to potential compensation strategies
- Available resources
- Family/significant other/caregiver support and willingness to be involved
- Previous compensation efforts

By gathering information that fits into these categories, the clinician will be able to understand and identify which approach will best meet the memory needs of the person with injury, and potentially avoid problems that could deter the person's ability to be consistently successful in use of the memory strategy. While not directly reported in the literature, experienced clinicians are aware that success is limited when taking a memory compensation approach "off the self" and delivering it to the person with injury, without understanding their needs or preferences. Only by fully understanding the specifics of the memory problem and the contributing factors that facilitate and hinder memory compensation efforts, can the clinician devise an informed plan for intervention.

# Step 2: Choose a Compensatory Memory Strategy (with Input of the Person with TBI and Caregiver)

Based on the information gathered during step 1, the clinician can hypothesize which memory strategy may best fit the client's needs and available resources; however, the final choice of strategy should be made with input from the client and involved caregiver. Acceptance of the strategy by the person with injury is crucial to successful implementation. Some people have strong negative reactions to using compensatory memory strategies, believing that they are a "crutch" and will prevent their memory abilities from improving. Education is especially important for these people. Open discussion about their comfort level in using the strategy, and any anticipated obstacles to use, helps to ensure ecological validity and to involve the client in the thinking process, which can be crucial for continued compensation of memory problems once the intervention ends. Even a clinician with years of experience in compensatory memory training may not be successful if pushing a strategy that a particular client is not comfortable with. We view the memory rehabilitation process as a partnership, with both therapist and client working together to develop and implement memory compensation strategies. A list of some possible strategies is shown in Table 1. We have found that it is helpful to provide clients with a list of possible strategies to begin discussion, and we have provided a description that can be offered to clients for each strategy. This list is not meant to be exhaustive, and therapists should be open to clients proposing novel strategies that meet their needs.

#### Step 3: Setting Up the Strategy

Once a strategy has been agreed upon, the therapist should help the client and caregiver to set

Compensatory memory strategy	Brief description of strategy
Memory notebook	A place where all important information to be remembered is written down. A memory notebook can be divided into different sections (e.g., daily calendar; things to do; addresses and phone numbers) based on individual needs. It can also be referred to as a planner or organizer
Cell phone or smart phone	Blackberry, iPhone, and other mobile phones have alarms that can be set to sound at a particular time and date as a reminder of appointments, things to do, etc. Some phones also have calendars and a place for notes
Computer	Like a memory notebook, a computer can be a place where all important information is kept. A calendar program on a computer can be used to keep track of important appointments or tasks to accomplish (e.g., paying bills, submitting paperwork)
Checklist	A list of items that need to be purchased, tasks that need to be completed, steps in a task or procedure, or anything else that needs to be remembered and can be formatted like a checklist (i.e., each step or item can be checked off as it is completed)
Memory station	A specific place in the home where frequently used items are kept or where belongings are kept that need to be taken when the person leaves home
Pill box	A box with separate compartments that can hold medications to be taken and that can be labeled as to day and time of dosage
Digital voice recorder	Can be used to keep track of information when writing it down is too difficult or not preferred. For example, when parking your car at the grocery store, you can read your car's location into the voice recorder and after grocery shopping, play back what you recorded to quickly locate your car

 Table 1 Examples of compensatory memory strategies

up the strategy in a way that will maximize success. Even if a client is already using a strategy, it may need to be set up differently or reorganized. A good example is use of a pill box to keep track of medications. Many pill boxes are set up by day, with all the medications for one day placed in the same compartment (e.g., "Monday" compartment). This can be problematic if there is more than one dose of the same medication per day, or if there are multiple medications being taken in one day, but at different times. Persons with impaired memory may forget which of the medications to take at which time or may become confused and take medications twice. The pill box should be set up according to dosage times. For example, all of the medications for the Monday a.m. time slot should be in one compartment, the noon time slot in another, and the p.m. dosage in yet a different compartment. This strategy may also need to be supplemented by use of an alarm to cue the person with TBI to go to the pill box to take the medication. This is just one example of how traditional strategies may need to be altered to fit the needs of individual clients. Setting up strategies in the context in which they will be used (e.g., home, community) can be very beneficial.

#### Step 4: Demonstrating the Strategy

Simply explaining strategy use to a person with TBI and/or their caregiver is not usually sufficient. Even the most well-established and easyto-follow verbal and written instructions can be difficult to implement once the individual with injury has left the rehabilitation treatment setting and returned to his or her home or community. The clinician should teach the memory compensation strategy to the client in short, discrete steps and provide reinforcing feedback. It is imperative to know that the person with injury adequately understands how the memory compensation strategy is implemented and when to use it. This can best be done by breaking the strategy down and showing the person with injury in a "handson," ecologically valid manner (i.e., working

with materials that will be used by the individual with injury and in situations that resemble the real-world situations as closely as possible). This can allow the clinician to identify obstacles to successful strategy implementation and address the problems immediately. It is important to provide reinforcing feedback as the person with injury implements the strategy. People often become discouraged when the strategy does not work right away, but successful use of strategies requires repeated practice. Providing verbal reinforcement and encouragement can help to build a client's confidence that he or she can master the strategy.

# Step 5: Simulated and Real-World Practice

Guided practice of compensatory memory strategies is important for clients to acquire the skills necessary to use them independently. Initially, this practice can occur through role-played situations with the therapist; however, much of the meaningful practice must occur in real-world situations, such as in the client's home or community. Assigned practice of strategies in these real-world environments is crucial to success. The therapist should help the person with injury to identify specific opportunities for practice in their real-world environments. The strategies should be practiced a minimum of once per day, but optimally more frequently. Providing clients with a written reminder to practice (e.g., calendar, list) can be beneficial, but involving caregivers to prompt them is also helpful. If naturally occurring opportunities for daily practice are not readily available during the course of a client's day, the clinician should assist with development of role-plays that can be used to practice strategies. In our clinical practice and experience, daily and repeated practice of the memory compensation strategy is necessary for the strategy to become a habit. Once use of the memory strategy becomes a habit, the memory problem that it is designed to address will have less of an impact on the person's daily functioning. This is the ultimate objective of memory rehabilitation.

#### Step 6: Follow-up Support

Use of compensatory memory strategies often must evolve over time to meet changing needs in real-world environments. Follow-up support is important for helping to maintain the skills learned during therapy. This support can be provided by telephone or in person. During these support sessions, the therapist can determine if the client is using the memory compensation strategy on an ongoing basis, if he or she is encountering any problems, and if the strategy needs to be adapted to meet the demands of a new situation. The therapist can assist with problemsolving and tweaking of the memory strategy. Whenever possible, the person with injury and their caregiver should be engaged in the problemsolving process. As noted in the section on emphasizing process, this can help them learn how to generalize the memory compensation strategy to fit other needs or needs that have not yet emerged. In some situations, development of a new strategy may be necessary.

## **Case Examples**

We will now present two case examples that illustrate in detail how to apply the principles and concepts discussed in this chapter. These case examples are based on our clinical experiences in working with individuals with TBI in both a postacute rehabilitation setting and in an ongoing clinical trial investigating contextualized memory compensation training in participants' homes.

# Case Example No. 1: Remembering to Take Medications

Bob was a 24-year-old male who sustained a severe TBI as a result of a motor vehicle accident. At the time of initial clinical assessment with Bob, he was 6 months post-injury and had not returned to productive work outside the home. He was safe to stay home alone, but had moved in with his girlfriend for continued support and assistance with meeting his needs. During clinins uled medication time to remind h

cal interview, Bob expressed several concerns related to his memory; chief among them was his ability to remember to take his medications as directed. He was taking numerous medications at multiple times throughout the day and was not using a pill box. Because his girlfriend and family recognized the potential for Bob to miss a medication dose or take the wrong medication, his girlfriend and brother were actively involved in monitoring his medications. His brother, who was responsible for his daytime medications, frequently called him or stopped by his apartment to ensure that his medications were taken as prescribed. This was creating a strain for both Bob and his brother. Bob felt that he was being treated like a child and his brother was having difficulty meeting his demands at work because of his involvement in his brother's care. At the end of the initial clinical interview and assessment, it was agreed that the first treatment goal would be for Bob to become independent with his medications.

#### **Information Gathering**

To figure out how to best intervene in Bob's difficulty with remembering to take his medications as directed, in-depth information was needed. Questions, like those shown in Table 2 below, were asked of Bob and his brother during his first inhome appointment:

From these questions it was learned that Bob was taking five different medications and the dosage of one of them changed every couple of days. He took medications both in the morning and in the evening, and for two of the medications, he took them both in the morning and evening. Bob kept his medications lined up on the dining room table. Once he had taken the medication, he would turn a medication bottle around, so that the label could not be seen. He kept his medications in the dining room so that he saw them frequently, which reminded him to take them, and he preferred to take his medications with food (it should also be noted that Bob did not have small children in his home, so there were no worries about keeping medications in a spot where a child would not be able to access them). Bob's brother called him 5 min prior to a scheduled medication time to remind him to take his medication, and his girlfriend would remind him to take his medication in the evening if she did not see him take them on his own. Bob's brother and girlfriend would periodically count the number of pills in a bottle to ensure that Bob was taking his medication as prescribed. When his medications were close to running out, his girlfriend would call in the prescription refill and pick them up from the pharmacy.

Bob did not have a pill box, but was interested in and willing to buy one. He believed that he could fill his pill box on his own, but was willing to include his brother and girlfriend in the beginning. He was also open to his brother and girlfriend initially monitoring his use of the pill box to ensure that he took his medications, but felt that their monitoring would not be needed for very long. Bob was also interested in using his cell phone to set-up reminders of when to take his medication. He always carried his cell phone in his pants' pocket or on a table next to him, so he did not feel that wearing a digital watch with a reminder was necessary. He also did not like to wear a watch. Bob was not interested in having written reminders posted in the home because he did not want guests to know he had a hard time remembering information. He also felt like posted reminders would lead him to feeling as if he was being treated like a child.

Prior to injury and currently, Bob felt comfortable with his knowledge of how his cell phone worked, and he believed he could independently program reminders on it. His brother had a similar phone to Bob's and could be a resource for programming reminders, if needed. Bob's brother was willing to be involved in Bob's therapy in the hopes that he could become more independent and need less help from him. Bob's girlfriend was also interested in being involved in his therapy, but did not have a work schedule that would allow for it. Bob's brother and girlfriend frequently spoke, and he and Bob felt like they could adequately pass along information to her so that she too could help Bob with memory compensation efforts.

Before injury, Bob did not use any strategies to help him with his memory. He felt like he had

 Table 2
 Information gathering for Case No. 1

Specifics about the memory activity

- · How many medications is Bob taking?
- At what times must each of the medications be taken (including multiple times for some medications)?
- · Where does Bob keep his medications?
- How does Bob keep track of whether or not he has taken his medications (including whether a caregiver prompts him)?
- Where is he when he needs to take his medications (e.g., at home, out in the community)

Previous compensation efforts

- What, if any, previous compensation efforts have been tried by Bob and his family to help with medication management?
  - Were these attempts successful, and why or why not?
  - Are there elements of these previous strategies that they would like to incorporate into development of a new strategy? Can the existing strategy be modified to be more effective?
- What, if any, strategies did Bob use to help with memory prior to his injury?

Reactions to potential compensatory strategy

- Is Bob open to using a pill box to manage his medications? If not, what are his concerns?
- Would Bob prefer to use an electronic device or paper-and-pencil-based tools to help him remember to take medications?
- Is Bob open to involving his family in helping him to learn a compensatory memory strategy?
- Available resources
- Does Bob already own a pill box?
- Does Bob have a cell phone?
  - Does the phone have text messaging or an alarm feature?
  - Does Bob or his family have access to the Internet to program text reminders to be sent to his phone?
  - Does Bob have an alarm clock or other device (e.g., digital watch with an alarm feature) that could be used to alert him that it is time to take medications?
  - -What types of paper-and-pencil materials does he have that could be used to develop a strategy?

Family member/caregiver support

- · How do Bob's brother and girlfriend ensure that he is taking his medications as prescribed?
- · Does Bob need help getting refills on his medications?
- Does Bob's family believe that he would need supervision to fill his pill box?

How would family members, if at all, be willing to help Bob to learn a strategy to remind him to take his medications?

been able to remember appointments, important information, and anything else without any external aids.

#### Strategy Development

Review of cognitive rehabilitation literature suggests that a strategy focused on the use of an external aid with direct application to a functional activity would be best for a person with a severe TBI [43–45]. Within this context and clinical experience, it was hypothesized that a pill box, in conjunction with external reminders to take medications, would be a best-fit memory compensation strategy for Bob's goal of remembering to take his medications as prescribed. Based on Bob's preferences, text messages or reminders sent from his cell phone would be used as external reminders of when to take his medications. This strategy was presented to Bob and his brother and met with approval. Bob felt like the use of his cell phone in the strategy was in line with his preferences, and using his cell phone helped him to feel "normal since people use their cell phones for all sorts of things." His brother felt that he and Bob's girlfriend could easily support Bob in the use of the strategy.

To identify the specifics of how the strategy would work, Bob and his brother wrote down a list of all Bob's medications, their dosages, and the times the medications needed to be taken. Together, they programmed Bob's cell phone to send him a reminder message that consisted of the instruction "take AM MEDS" or "take PM MEDS," depending on which time of day it was. With the reminders in place, Bob and his brother filled his pill box, using the medication list they had created and paying close attention to the particular medication that changed three times during the week. Bob chose to keep his pill box on the dining room table since he had a strong memory of the pills being there and it did not pose a risk to anyone in the household to keep them there. Bob also decided he would like to keep a box of crackers next to his pill box so that he would have food to eat when it was time to take his medications.

In terms of a procedure to follow, it was decided that after hearing and reading the reminder message, Bob would stop whatever he was doing, get a glass of water and then sit down at the dining room table, take the pills out of the appropriate place in the pill box, eat a few crackers, and then take his medications. At the end of the week, Bob would refill his pill box. For the first 2 weeks, Bob would fill the pill box with either his brother or girlfriend. After that, he would fill it on his own, with his brother or girlfriend checking his work until everyone felt confident in Bob's ability to independently fill his pill box. A plan for Bob to take over calling in his own prescription refills was deferred initially; however, Bob indicated that once he was filling his pill box with complete independence, he believed he could make his girlfriend aware of the need to call in his prescription refills.

# Teaching of Strategy and Assignment of Practice

To ensure that Bob could carry out the compensation strategy that was developed, the therapist first modeled the strategy in short, discrete, concrete steps to Bob and his brother. Sitting in a chair in his living room, the therapist made a mock cell phone alert sound and upon hearing the sound, picked up Bob's cell phone and said, "my phone has a reminder message on it that says 'take AM MEDS,' I am going to get up out of this chair and get a glass of water. The therapist then

walked into the kitchen, with Bob and his brother following behind her watching, and got a glass of water. The therapist then said, "I have a glass of water and I am now going to go sit at the dining room table." She then went and sat at the dining room table and said, "I am taking the medications out of the AM slot for Monday." She then took the medications out of the indicated spot. The therapist then said, "I am going to eat a few crackers and then I'll take my medications." Having gone through the entire strategy step-bystep for Bob and his brother, the therapist asked if they had any questions before helping to guide Bob through the same trial run. Without much difficulty, Bob went through the strategy and the therapist positively reinforced his efforts.

Bob needed to take medications twice a day, so the therapist did not see a need to assign him any artificial practice. Practice of the strategy was already set up through the twice daily reminders that were programmed in Bob's phone. With Bob in agreement, Bob's brother would call him 15 min after his morning reminder had gone off on his phone to ensure that he had taken his medications. His girlfriend would be home in the evening to observe if Bob followed through with the reminder to take his evening medications. Bob, his brother, and the therapist felt comfortable with the plan for continued use and practice of the memory compensation strategy.

#### **Follow-up Support**

During a follow-up visit, Bob and his brother reported successful use of the strategy. As a precaution, the therapist asked Bob to role-play use of the strategy. During this role-play, the therapist noticed that Bob's pillbox was full despite it being the middle of the week. She queried Bob about this and Bob reported that he filled in the pill box each night. The therapist, Bob, and his brother discussed the pros and cons of filling the pill box each day or once a week. Together they identified that it was better to fill the pill box once a week so that Bob could immediately know where he was at with his pills during a week. Particularly due to the medication with dosage changes, the potential for confusion and inaccurate taking of medications was high. No other difficulties or problems with this strategy were identified during subsequent visits with Bob and his brother.

# Case Example No. 2: Remembering Important Information, such as Details About Upcoming Events and Appointments

Bill was a 56-year-old, married male who sustained a moderate TBI as a result of a fall from a ladder. At the time of the initial clinical encounter, Bill was 3 months post-injury and very eager to go back to work. Bill was a successful regional salesperson for a major company and was worried about losing his customers in an extremely competitive market. By 3 months post-injury, Bill's recovery had progressed quickly and the rehabilitation team had begun to discuss his return-to-work plan. One of the biggest challenges for Bill to cope with when he returned to work was going to be deficits in his short-term memory that had been observed functionally and on neuropsychological testing. Bill often forgot details about upcoming events and times of appointments and meetings. As part of Bill's job, he would need to remember times and dates of meetings with customers, specifics about their needs as customers, and information about past and future orders. The goal of treatment with Bill was to help him learn a compensation strategy for remembering important information.

#### Information Gathering

In-depth information was first needed in order to figure out how to best intervene on Bill's difficulty with remembering important information. Questions, like those shown below in Table 3, were asked of Bill during his first session:

Bill reported that he frequently forgot details about the date and time of a meeting and/or appointment, questions he wanted to ask at meetings, and some of the answers to his questions. Bill noticed that he forgot important information when attending medical appointments on his own and meeting with the Human Resources (HR) Department at his job regarding his return to work. He would become aware that he forgot about an appointment or important information when his wife asked him about how a meeting went or about paperwork that he needed to complete. Bill would then become embarrassed when he needed to reschedule an appointment or request additional information. He felt he had a "really good memory" before his accident and did not need to write down information in order for him to remember it. Prior to injury, he often took phone calls while driving to appointments with customers and was able to remember information that was discussed. He felt like he had been successful in his work because he could remember a great deal of information in his head.

Bill had been given a memory notebook during his inpatient hospitalization, but when working with the outpatient rehabilitation team, Bill expressed doubts and concerns about using it. Bill wanted to use his Blackberry to help him to remember important information. He felt that the memory notebook he was given was too cumbersome to carry around, and the sections in it did not meet his needs. He felt that using a Blackberry would demonstrate that he is "current with the times." He already had to carry a Blackberry as his cell phone for work and was able to sync it with his computer to prevent loss of the information should he lose the phone. The Blackberry had text messaging, reminder, and note-taking features. Bill did not remember where he stored the manual for his Blackberry, but felt that he had a decent amount of knowledge about its features, and a close friend of his could serve as a resource for Blackberry-related questions.

Prior to injury, Bill was not "using his Blackberry to its full potential," as he did not make use of its many features, but he recently entered appointments into the calendar on his phone. While his entry of his appointments was accurate, he still missed appointments because Bill preferred to keep his pants pockets empty and often left his Blackberry on the dresser in his bedroom when he was home. If Bill was not in his bedroom when the alarm sounded on his Blackberry, he did not hear it and missed the appointment. He tended to only check the calendar 
 Table 3
 Information gathering for Case No. 2

Specifics about the memory activities and problems

- What type of information does Bill typically forget?
- Are there particular situations or settings in which Bill is more likely to forget important information?
- · How does Bill identify when he has forgotten information?
- · How often does forgetting information create a problem for Bill?

Previous compensation efforts

- What, if any, previous compensation efforts have been tried by Bill to help him remember important information?
   Does Bill think that his previous compensation efforts have been helpful? Why or why not?
  - What elements of previous compensation, if any, would he like to incorporate into the new strategy?
- Did Bill use strategies to help with his memory before injury? If yes, what were the strategies and what were they used for?

Reactions to potential compensation strategies

- Would Bill prefer to use an electronic device or paper-and-pencil-based tools to help him remember important information?
  - If he has an electronic device preference, what are Bill's views on using a cell phone, digital voice recorder, or computer to help him to remember important information?
  - If he has a paper-and-pencil preference, what are his views on having reminders posted on the walls of his home or office to help him to remember important information?

Is Bill willing to involve family members and/or coworkers in helping him to learn a compensation strategy?

Available resources

- Does Bill have a cell phone?
  - Does his phone have a text messaging or reminder feature?
  - Does he have access to an internet program that would allow him to send reminder text messages to his phone?
  - Does he use the phone for taking notes or recording personal information?
  - Does he have access to the manual for his phone or access to someone who can help him learn to use the different features that could be used to compensate for memory problems?
- · Does Bill have a digital voice recorder?
- · Does Bill have a computer?
- · Does Bill have a paper-and-pencil-based planner?
- · Is Bill willing to buy any of the above resources?

Family member/caregiver support

- Are any members of Bill's family willing to be involved in helping him to learn how to compensate for his memory difficulties?
- Are Bill's supervisors and coworkers open to being involved in helping him to learn how to compensate for his memory difficulties?

on his phone in the morning, so he would frequently forget appointments that occurred later in the day.

Bill had a very supportive wife, who had been frequently involved in his treatment; however, a recent increase in her demands at work had resulted in her being less able to attend treatment sessions with Bill. She requested that the treatment team call her with updates and suggestions for helping Bill. Bill was reluctant to involve any coworkers in his treatment. He feared that his employer and colleagues would second-guess his abilities and readiness to return. He expressed willingness to revisit the issue of involving coworkers if he could not improve his performance independently, with use of compensatory memory strategies.

#### Strategy Development

The treatment team felt that he could benefit from learning a strategy that could be applied in multiple situations. Based on Bill's initial compensation attempts and his clear preference to utilize his Blackberry, the team agreed to focus on using his Blackberry instead of his memory notebook to help him to remember important information. Based on the initial information gathered, it appeared that Bill had two main types of information to remember-appointments and information that related to his appointments. As a starting point, Bill made a list of all of the times and dates of standing customer appointments. After the list was finished, he went back through the list and noted the information that he would need to ask at each of the various appointments. Bill then entered each appointment into his calendar on his Blackberry. His calendar had a feature where he could include information in a "notes section" for each appointment. In that space he entered the questions that he needed to ask during the appointment. Bill felt comfortable with how he had programmed his Blackberry, but agreed to go over what he had done with his friend to see if he had done it correctly or if there was an easier or better way to do it.

We identified that Bill would review the notes for an appointment once he parked his car at the appointment, just prior to the start of the appointment, and at the conclusion of the appointment, to ensure that he had covered all of his questions. Bill would also keep his Blackberry readily available to him during the appointment, so that he could make notes about the question's response in the same calendar entry and enter in new appointments if needed. We also decided that Bill should program a reminder alarm to ring on his phone every hour to prompt him to review his calendar, as checking his calendar only once a day had previously caused problems. Upon hearing the reminder alarm, Bill would review his calendar for the day. In the evening, he would review the calendar for the subsequent morning so that he would have time to do any necessary preparation work. Bill also agreed to buy a holster, so that he could keep the Blackberry on the waistline of his pants.

## Teaching of Strategy and Assignment of Practice

Bill first practiced responding to the hourly alarm reminder to check his calendar. In response to a simulated alarm sound, the therapist talked Bill through picking up his phone, scrolling through his calendar, and then returning his phone to the holster on the waistline of his pants. She positively reinforced Bill as he adeptly demonstrated this strategy. To practice using his Blackberry during an appointment, they role-played how he would use the Blackberry for a doctor's appointment on the subsequent day. First, the therapist modeled how to review the questions he had for the appointment, while in his car. She stated, "I just parked my car and I am now going to take out my Blackberry and refresh my memory of the questions that I want to ask the doctor. Let's see, my appointment is at 3 pm, oh here is the entry and in the notes Section I see that I wanted to ask about my test results and my blood pressure reading during my last appointment. Now that I am done reviewing my notes, I am going to put my phone back in its holster." The therapist then said, "Okay, I am now in the waiting room of the doctor's office and I am going to review my notes for this appointment again," and modeled taking the Blackberry off of its holster, scrolling through the entry and muttering information to herself. Next, the therapist said, "Now Bill, let's assume the visit is over and I asked my questions, but I need to review my notes for this appointment one last time before I leave the doctor's office, so that I am sure I got the information I needed." The therapist then removed the Blackberry from its holster once again and modeled using it. She then said, "One more step we should practice together would be to enter in a new appointment. So let's assume I need to stop by the front desk of the doctor's office on my way out and schedule my next appointment." She then demonstrated asking about a follow-up appointment and entering that information into the Blackberry. Following the therapist modeling of the entire strategy, she guided Bill through that same role-play and provided positive reinforcement as he proficiently went through it.

The real-world practice assigned to Bill would be to frequently review his calendar according to the hourly reminder alarms. At the time of training, he did not have appointments on a daily basis, as he had not returned to work yet. Bill agreed that daily practice of the memory compensation strategy was necessary in order for him to be comfortable with it prior to returning to work. Role-play scenarios were established, which could be used on the days when he did not have an appointment. Bill entered these role-play scenarios into his Blackberry before leaving the therapist's office at the conclusion of his appointment. At the end of the session, Bill and the therapist called his wife to review the strategy that had been developed, as well as the plan for daily practice. She expressed understanding of the strategy and intent to help Bill practice.

#### Strategy Follow-up

During Bill's next appointment, he reported that he had no difficulties practicing using his Blackberry to remember important information, but he had thought of a situation in which using his Blackberry would be difficult-receiving a call from a customer while driving. As previously mentioned, prior to injury, Bill would take calls from customers while driving and remember the information that was discussed. Bill recognized that if he took the call while driving, he probably would not remember all of the information that was discussed unless he took notes in his Blackberry. This would be unsafe to do while driving. The therapist guided Bill in discussing the pros and cons of letting his phone go to voicemail while driving. He decided to let his phone go to voicemail and then check his voicemail once safely parked. At that time, he would immediately enter the important information into his Blackberry and/or return the phone call. This sequence of actions was then role-played to solidify it for Bill.

#### Summary

Everyday memory problems are frequent following TBI, particularly in the areas of learning new information and prospective memory. Training in compensatory memory strategies is a practice standard for persons with mild memory impairment due to TBI, and use of external strategies, with direct application to functional activities, is a practice guideline for people with severe memory deficits after TBI. A systematic, yet individualized approach to training persons with TBI to use compensatory memory strategies is most likely to be successful for improving everyday memory functioning. Engagement of the person with TBI and a family member or caregiver in the process of choosing, developing, and implementing memory strategies helps to increase generalizability to real-world settings. When possible, training should be conducted within the real-world setting where it will be used (e.g., home or community). Accounting for individual needs, resources, and preferences, and providing follow-up support for strategy usage, is most likely to ensure success.

Acknowledgment Preparation of this chapter was partially supported by U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020.

#### References

- Levin, H. S., Grossman, R. G., Rose, J. E., & Teasdale, G. (1979). Long-term neuropsychological outcome of closed head injury. *Journal of Neurosurgery*, 50, 412–422.
- Van Zomeren, A. H., & Van Den Berg, W. (1985). Residual complaints of patients two years after severe head injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 48,* 21–28.
- Kaitaro, T., Koskinen, S., & Kaipio, M. L. (1995). Neuropsychological problems in everyday life: A 5-year follow-up study of young severely closedhead-injured patients. *Brain Injury*, 9, 713–727.
- Ponsford, J. L., Olver, J. H., & Curran, C. (1995). A profile of outcome: 2 years after traumatic brain injury. *Brain Injury*, 9, 1–10.
- Hoofien, D., Gilboa, A., Vakil, E., & Donovick, P. J. (2001). Traumatic brain injury (TBI) 10–20 years later: A comprehensive outcome study of psychiatric symptomatology, cognitive abilities and psychosocial functioning. *Brain Injury*, 15, 189–209.
- Tate, R. L. (1997). Beyond one-bun, two-shoe: Recent advances in the psychological rehabilitation of memory disorders after acquired brain injury. *Brain Injury*, *11*, 907–918.
- Cifu, D. X., Keyser-Marcus, L., Lopez, E., Wehman, P., Kreutzer, J. S., Englander, J., et al. (1997). Acute predictors of successful return to work 1 year after traumatic brain injury: A multi-center analysis. *Archives of Physical Medicine and Rehabilitation*, 78, 125–131.
- Mazzaux, J. M., Masson, F., Levin, H. S., Alaoui, P., Maurette, P., & Barat, M. (1997). Long-term neuropsychological outcome and loss of social autonomy after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 78, 1316–1320.

- Rappaport, M. J., McCullagh, S., Shammi, P., & Feinstein, A. (2005). Cognitive impairment associated with depression following mild and moderate traumatic brain injury. *Journal of Neuropsychiatry & Clinical Neurosciences*, 17, 61–65.
- Sohlberg, M. M., & Mateer, C. A. (2001). Cognitive rehabilitation: An integrative neuropsychological approach. New York: The Guilford Press.
- Goldstein, F. C., Levin, H. S., & Boake, C. (1989). Conceptual encoding following severe closed head injury. *Cortex*, 25, 541–554.
- Crosson, B., Novack, T. A., Trenerry, M. R., & Craig, P. L. (1988). California Verbal Learning Test (CVLT) performance in severely head-injured and neurologically normal adult males. *Journal of Clinical and Experimental Neuropsychology*, 10, 754–768.
- Brooks, D. N. (1976). Weschler Memory Scale performance and its relationship to brain damage after severe closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 39*, 593–601.
- 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, 675–684.
- Kear-Caldwell, J. J., & Heller, M. (1980). The Weschler Memory Scale and closed head injury. *Journal of Clinical Psychology*, 36, 782–787.
- DeLuca, J., Schultheis, M. T., Madigan, N. K., Christodoulou, C., & Averill, A. (2000). Acquisition versus retrieval deficits in traumatic brain injury: Implications for memory rehabilitation. *Archives of Physical Medicine* and Rehabilitation, 81(10), 1327–1333.
- Curtiss, G., Vanderploeg, R. D., Spencer, J., & Salazar, A. M. (2001). Patterns of verbal learning and memory in traumatic brain injury. *Journal of International Neuropsychological Society*, 7, 574–585.
- Stuss, D. T., Ely, P., Hugenholtz, H., Richard, M. T., LaRochelle, S., Poirier, C. A., et al. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. *Neurosurgery*, *17*, 41–47.
- Crosson, B., Novack, T., Trenerry, M. R., & Craig, P. L. (1989). Differentiation of verbal memory deficits in blunt head injury using the recognition trial of the California Verbal Learning Test: An exploratory study. *The Clinical Neuropsychologist*, *3*, 29–44.
- 20. Paniak, C. E., Shore, D. L., & Rourke, B. P. (1989). Recovery of memory after severe closed head injury: Dissociations in recovery of memory parameters and predictors of outcome. *Journal of Clinical and Experimental Neuropsychology*, 11(5), 631–644.
- Hannay, H. J., Levin, H. S., & Grossman, R. G. (1979). Impaired recognition memory after head injury. *Cortex*, 15, 269–283.
- Goldstein, F. C., Levin, H. S., Boake, C., & Lohrey, J. H. (1990). Facilitation of memory performance through induced semantic processing in survivors of severe closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, *12*(2), 286–300.
- 23. Shum, D. H., Harris, D., & O'Gorman, J. G. (2000). Effects of severe traumatic brain injury on visual

memory. *Journal of Clinical and Experimental Neuropsychology*, 22(1), 25–39.

- Vanderploeg, R. D., Curtiss, G., Schinka, J. A., & Lanham, R. A., Jr. (2001). Material-specific memory in traumatic brain injury: Differential effects during acquisition, recall, and retention. *Neuropsychology*, 15(2), 174–184.
- Mateer, C. A., Sohlberg, M. M., & Crinean, J. (1987). Perceptions of memory function in individuals with closed-head injury. *Journal of Head Trauma Rehabilitation*, 2, 74–84.
- Roche, N. L., Fleming, J., & Shum, D. H. (2002). Selfawareness of prospective memory failure in adults with traumatic brain injury. *Brain Injury*, 16, 931–945.
- Cockburn, J. (1995). Task interruption in prospective memory: A frontal lobe function? *Cortex*, 31, 87–97.
- Cockburn, J. (1996). Failure of prospective memory after acquired brain damage: Preliminary investigation and suggestions for future directions. *Journal of Clinical and Experimental Neuropsychology*, 18, 304–309.
- Sunderland, A., Harris, J. E., & Baddeley, A. (1983). Do laboratory tests predict everyday memory. *Journal* of Verbal Learning and Verbal Behavior, 22, 341–357.
- Boake, C., Freeland, J. C., Ringholz, G. M., Nance, M. L., & Edwards, K. E. (1995). Awareness of memory loss after severe closed-head injury. *Brain Injury*, *9*, 273–283.
- Thoene, A. I., & Glisky, E. L. (1995). Learning of name-face associations in memory impaired patients: A comparison of different training procedures. *Journal of International Neuropsychological Society*, 1, 29–38.
- Milders, M., Deelman, B., & Berg, I. (1998). Rehabilitation of memory for people's names. *Memory*, 6, 21–36.
- Sohlberg, M. M., & Mateer, C. A. (1989). Training use of compensatory memory books: A three stage behavioral approach. *Journal of Clinical and Experimental Neuropsychology*, 11, 871–891.
- Donaghy, S., & Williams, W. (1998). A new protocol for training severely impaired patients in the usage of memory journals. *Brain Injury*, 12, 1061–1076.
- Ownsworth, T. L., & McFarland, K. (1999). Memory remediation in long-term acquired brain injury: Two approaches in diary training. *Brain Injury*, 13, 605–626.
- Zencius, A., Wesolowski, M. D., & Burke, W. H. (1990). A comparison of four memory strategies with traumatically brain-injured clients. *Brain Injury*, *4*, 33–38.
- Schmitter-Edgecombe, M., Fahy, J. F., Whelan, J. P., & Long, C. J. (1995). Memory remediation after severe closed head injury: Notebook training versus supportive therapy. *Journal of Consulting and Clinical Psychology*, 63, 484–489.
- Van den Broek, M. D., Downes, J., Johnson, Z., Dayus, B., & Hilton, N. (2000). Evaluation of an electronic aid in the neuropsychological rehabilitation of prospective memory deficits. *Brain Injury*, 14, 455–462.

- Wade, T. K., & Troy, J. C. (2001). Mobile phones as a new memory aid: A preliminary investigation using case studies. *Brain Injury*, 15, 305–320.
- Wright, P., Rogers, N., Hall, C., Wilson, B., Evans, J., et al. (2001). Comparison of pocket-computer memory aids for people with brain injury. *Brain Injury*, 15, 787–800.
- 41. Hart, T., Hawkey, K., & Whyte, J. (2002). Use of a portable voice organizer to remember therapy goals in traumatic brain injury rehabilitation: A withinsubjects trail. *The Journal of Head Trauma Rehabilitation*, 17, 556–570.
- 42. Stapleton, S., Adams, M., & Atterton, L. (2007). A mobile phone as a memory aid for individuals with traumatic brain injury: A preliminary investigation. *Brain Injury*, 21, 401–411.
- Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., et al. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation*, 81, 1596–1615.
- 44. Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., et al. (2005). Evidence-based cognitive rehabilitation: Updated review of the literature from 1998 through 2002. Archives of Physical Medicine and Rehabilitation, 86, 1681–1692.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., et al. (2011). Evidence-based

cognitive rehabilitation: Updated review of the literature from 2003 through 2008. Archives of Physical Medicine and Rehabilitation, 92, 519–530.

- 46. Ehlhardt, L. A., Sohlberg, M. M., Kennedy, M., Coelho, C., Turkstra, L., et al. (2008). Evidence-based practice guidelines for instructing individuals with neurogenic memory impairments: What have we learned in the past 20 years? *Neuropsychological Rehabilitation*, 18, 300–342.
- Scherer, M., Schneider, B., Cushman, L., & Wong, T. (2008). Assessing the match of person and cognitive support technology. *Archives of Physical Medicine* and *Rehabilitation*, 89, E9.
- Ylvisaker, M., Hanks, R., & Johnson-Greene, D. (2002). Perspectives on rehabilitation of individuals with cognitive impairment after brain injury: Rationale for reconsideration of theoretical paradigms. *The Journal of Head Trauma Rehabilitation*, 17, 191–209.
- 49. Braga, L. W., Da Paz, A. C., & Ylvisaker, M. (2005). Direct clinician-delivered versus indirect familysupported rehabilitation of children with traumatic brain injury: A randomized controlled trial. *Brain Injury*, 19, 819–831.
- Gentry, T., Wallace, J., Kvarfordt, C., & Lynch, K. B. (2008). Personal digital assistants as cognitive aids for individuals with severe traumatic brain injury: A community-based trial. *Brain Injury*, 22, 19–24.

# Rehabilitation of Attention and Executive Function Impairments

# Keith D. Cicerone and Kacey Little Maestas

#### Abstract

Disturbances of executive functions, including the executive control of attention, are recognized as among the most common, persistent, and debilitating consequences of traumatic brain injury (TBI) [1-6]. This chapter focuses on the application of empirically supported strategies for managing impairments of higher level attention and executive functions following TBI. We recognize that the clinical neuropsychology and cognitive rehabilitation literatures have typically considered attention and executive functions in relative isolation. However, there is considerable overlap and interdependence in the structure and function of higher-level aspects of attention and executive functions. Throughout the chapter, we use the term "attention-executive functions" to refer to executive functions (e.g., anticipating consequences, planning and organizing, initiating and sustaining activities) as well as skills associated with the executive control of attention (also referred to as supervisory, complex, or higher-level attention). Skills associated with executive control of attention include the ability to sustain attention in the face of distractions (selective attention), switch focus or mental sets (alternating attention), or manipulate and control information held online (working memory). The processes involving attention-executive functions are distributed throughout the frontal regions and connect with other frontal, posterior, and subcortical areas to exert executive (i.e., top-down) control over lower level, more modular, or automatic functions [7, 8]. The frontal lobes and interconnecting circuits are

K.D. Cicerone, Ph.D., ABPP-Cn (🖂) Neuropsychology and Cognitive Rehabilitation, JFK-Johnson Rehabilitation Institute, Edison, NJ, USA

Rutgers—Robert Wood Johnson Medical School, New Brunswick, NJ, USA e-mail: KCicerone@JFKHealth.org K.L. Maestas, Ph.D. Baylor College of Medicine, Houston, TX, USA

Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_10, © Springer Science+Business Media, LLC 2014

particularly vulnerable to focal and diffuse damage in TBI, which accounts for the frequency of deficits involving attention-executive functions in this population. Given the overlap in the structure and function of attentionexecutive processes, interventions targeting these processes are also intimately related.

The chapter begins with a review of attention-executive functions. We also provide a brief overview of recent empirically driven models of frontal lobe functioning, particularly as these frameworks relate to the conceptualization and remediation of attention-executive deficits following TBI. Next, we describe the nature of attention-executive functioning impairments and illustrate how associated impairments can manifest in individuals' everyday lives. This is followed by a review of interventions for attention-executive functioning, including empirical evidence that has culminated in recommendations for clinical practice. The remainder of the chapter is devoted to describing the application of metacognitive training, a class of interventions that have demonstrated empirical evidence of efficacy in ameliorating attention-executive impairments [9-12]. Based on our clinical experiences and judgments, we will emphasize key components of the described therapies with the intent of providing a greater understanding of the theory and application of metacognitive strategy training in the context of brain injury rehabilitation.

#### Keywords

Attention • Executive function • Frontal lobe • Brain injury • Cognition • Rehabilitation

• Rehabilitation

# **Overview of Attention-Executive Functions**

The term "executive functioning" is used traditionally to refer to a set of integrated higher-order processes that determine goal-directed and purposeful behavior. By supervising and coordinating underlying cognitive, behavioral, and emotional processes, executive functions allow the orderly execution of daily life activities. Executive functions include the ability to formulate goals, solve problems, anticipate the consequences of actions, plan and organize behavior, initiate relevant behaviors, inhibit irrelevant behaviors, and monitor and adapt behavior to fit a particular task or context [9]. Higher-level attentional processes responsible for allocating attention across more than one task, actively shifting attention between tasks, and selectively sustaining attention while inhibiting irrelevant information, also fall within the broad rubric of executive functioning [13, 14]. Working memory, defined as a process that allows the short-term storage, maintenance, and manipulation of information [15], is also intimately related to attentionexecutive functioning. The "executive" aspect of working memory is the ability to actively manipulate and control transitory information in the mind [16].

Attention-executive functions are also related to emotional and behavioral self-regulation, and metacognitive processes [14, 17]. Metacognition, or simply thinking about thinking, is a set of processes that involves *metacognitive knowledge or beliefs, self-monitoring,* and *self-control* that work together to enable self-regulation [12, 18]. Metacognitive knowledge is the moment-tomoment and more stable beliefs about one's cognitive abilities. The ability to self-monitor one's performance and use the resulting internal feedback to adapt to changes in the environment or task demands (a process labeled self-control) is also an integral aspect of metacognition. There is some debate as to whether metacognition is in fact a core "executive" function or rather reflects a distinct superordinate process at the highest level of the cognitive system [12, 18]. Nonetheless, there seems to be general consensus that metacognitive self-regulatory skills are mediated by the frontal lobes, are frequently disrupted following traumatic brain injury (TBI), and are critical to the execution of self-directed complex behaviors [12, 14, 17-25].

# Models of Attention-Executive Functioning Impairments Following TBI

Recent developments in cognitive neuroscience, aided by advances in brain imaging methodologies (e.g., diffusion-tensor imaging–DTI; functional magnetic resonance imaging–fMRI), have led to number of new empirically driven, theoretical perspectives of attention-executive functioning. We will briefly review these perspectives with an emphasis on their relevance to the nature and rehabilitation of attention-executive impairments following TBI.

Posner and Peterson [26] have described an approach to understanding attention-executive processes based on principles of large-scale distributed neural networks [27]. Cognitive-neuropsychological and neuroimaging studies have identified distinct attentional networks for alerting, orienting, and executive control [26, 28–30]. The alerting attention network, which is characterized by reciprocal connections between right frontal and parietal regions, involves the ability to reach and sustain preparedness for potential stimuli [31]. The orienting attention network includes the superior and inferior parietal

lobes, frontal eye fields, superior colliculus, and the pulvinar and reticular thalamic nuclei, and involves the ability to make covert shifts in attention [31]. The executive attention network involves the anterior cingulate, medial frontal cortex, and lateral prefrontal cortex, and underlies the capacity to use relevant information, ignore irrelevant information, and resolve conflicts among competing sources of information [31]. It is worth noting that the capacity for conflict resolution is a developmental process, which in early life evolves from the need for resolution (or delay) of emotional needs [32].

Niogi and colleagues [33] examined the operations of these attention networks in relation to structural connectivity of white matter tracts. They demonstrated distinct and separable relationships between attention components and structural connectivity, providing evidence of discrete networks. Alerting was related to the posterior limb of the internal capsule, which contains thalamic-parietal connections. Orienting was related to the splenium of the corpus callosum containing interhemispheric tracts connecting regions including the frontal eye fields and posterior parietal regions. The executive-conflict effect correlated with structural integrity of the anterior cingulate region, as expected. Niogi and colleagues also demonstrated an association between decreased performance of the executive network after TBI and reduced fractional anisotropy within the anterior corona radiata, including projections to the anterior cingulate gyrus [34].

The initial investigations of attentional networks were directed at establishing the efficiency and independence of functions involving alerting, orienting, and executive attention [35]. More recently, investigations have demonstrated consistent interactions among attention networks [36]. For example, the alerting and orienting system will typically operate together in realworld situations where a single event indicates both when and where a relevant stimulus occurs [30], suggesting that both of these networks serve a more general preparatory function [37].

This anticipatory function is likely a general, integral feature of higher cognitive processing, especially attention-executive processes [38].

Ghajar and Ivry [39] have described an anticipatory neural network involving the prefrontalinferior parietal areas that provides a feed-forward mechanism that reduces performance variability by generating a "predictive brain state." This framework suggests that through the generation of moment-to-moment predictions about the immediate future, and the comparison of these predictions with sensory feedback, individuals will be less distracted by irrelevant information which will facilitate goal-oriented behavior. Disruption of this network may represent a fundamental defect after TBI resulting in a variety of deficits, including impairments of sustained attention, poor self-monitoring of errors, and loss of goal-directed behavior.

The interaction among various aspects of attention and executive functioning has also been related to three distinct, separable anterior attentional systems by Stuss and his colleagues [40]. One attentional system maintains a general state of readiness to respond, mediated primarily by superior medical frontal structures. A second attentional system serves primarily to bias attention toward specific criteria that guide responsiveness. Dysfunction of these processes, associated with damage to the left dorsolateral prefrontal cortex, is related to deficits of task initiation and setting of response criteria. The third attentional system maintains the criteria for response selection, so that relevant aspects of the environment are consistently selected and responses to competing or irrelevant targets are inhibited. Dysfunction in this network, associated with damage to right dorsolateral frontal cortex, produces deficits in sustaining attention to criteria and conflict resolution. This characterization of an anterior attentional system has significant correspondence with the attentional networks described by Posner and Peterson [26, 41]; specifically, an attentional network devoted to maintaining alertness or readiness to respond, a second network related to orienting and setting criteria to respond, and a third network related to "executive" attentional processes of response selection and conflict resolution.

Peterson and Posner [41] more recently noted the evidence supporting two brain systems related to the orienting network, through an interaction of endogenous and exogenous stimulus control [42]. Corbetta and Shulman described segregated networks corresponding to top-down and stimulus-driven regulation of attention, related to dorsal and ventral frontal-parietal connections, respectively [42]. While initially related to specific aspects of visual attention, more recent evidence suggests that these networks are related to more general, dynamic roles of reorienting between internally directed activity and attention to the environment [37]. They also noted that the orienting function is not restricted to visual information and overlaps with information from other sensory modalities. The operations of the executive network have also been elaborated, with evidence of dual networks involved in top-down cognitive control [43, 44]. The first executive system involves a fronto-parietal network, distinct from the orienting network, that serves primarily to adapt control over performance by maintaining a stable response set (including the maintenance and prioritization of information in working memory), while a network related to the cingular-opecular connectivity (consistent with the original executive network) provides moment to moment control over performance, perhaps by providing a continuously updated account of predicted demands on cognitive resources [45, 46].

Stuss has also described a "revamped attentional model" of executive functions, based on the fractionation of frontal lobe regions corresponding to fundamental cognitive operations [8]. Three of these proposed processes and their anatomic relations correspond to the earlier formulation of an anterior attentional system [8, 40]. Within this new framework, damage to the superior medial frontal lobes is associated with decreased performance on a range of (apparently dissimilar) tasks due to a failure of "energization," or the initiation and sustaining of any response. For example, superior medial damage might manifest as slow response on speeded tasks. Two aspects of "executive functioning" are related to the dorsolateral frontal cortices (DFC), as in the earlier model. Damage to the left DFC is related to initial task setting and goal orientation, while damage to the right DFC suggests poor monitoring of ongoing performance, deficits in sustained attention, and increased intra-individual variability. These three attentional networks can be further defined by their connections with other areas of the brain. The first is a ventral system involving orbitofrontal cortex and limbic structures related to emotional and behavioral regulation. The second is a rostral and lateral system with bidirectional connections between prefrontal cortex and posterior cortices related to "metacognitive" processes (i.e., the ability to self-monitor and self-regulate), including shifting between attention directed at the environment and internal trains of thought [47, 48]. Stuss and colleagues propose that metacognitive functions-positioned at the highest level of the cognitive system-coordinate the integration of attention-executive cognitive information with emotional and motivational processes, which ultimately guides and enables complex, purposeful behavior [8, 49]. The distinction between a more dorsal fronto-cingular-parietal pathway associated with more deliberate, slow-to-respond, volitional behavior and a ventral pathway that is more reactive (to either environmental stimuli or basic emotional states) and associated with quicker-to-respond, automatic behaviors may be another general principle of the brain's organization of complex attention-executive functions [50]. This dissociation is also central to dualprocess approaches to cognitive-affective control [51–54] and provides a framework for metacognitive strategy training methods for affective and behavioral self-regulation [55, 56].

These empirically supported models suggest that the dissolution and restoration of cognitive functioning after TBI may be informed through an understanding of the efficiency and interaction of attention-executive networks, although this approach has to date had limited influence on our understanding of cognitive recovery or the development of rehabilitation interventions. Several authors have argued that these models have important implications for the rehabilitation of attention-executive impairments [14, 57]. Metacognitive processes are critical to the ability to use internal goals and desires to direct ones' thoughts and behaviors. Thus, metacognitive training, characterized by interventions to foster anticipation and planning, response monitoring, and self-evaluation, may be particularly beneficial in reestablishing top-down control over attention-executive cognitive, emotional, and behavioral functioning [9–12, 51, 57, 58]. Recent applications of metacognitive interventions have also emphasized the importance of addressing not only attention-executive cognitive functions, but also emotional/motivational, and behavioral functions, as these processes are anatomically and functionally related in the execution of complex, real-world behaviors [59–61].

# The Nature of Attention-Executive Impairments Following TBI

Deficits involving self-directed attention and executive cognitive functioning, behavioral and emotional regulation, and metacognitive functioning are interrelated and commonly impaired following TBI.

# Attention-Executive Cognitive Impairments

Impairments of task setting and response preparation can be associated with problems anticipating errors, analyzing situations, planning and executing solutions, and maintaining a flexible or pragmatic approach to tasks-all of which are common after TBI. Impairments in attentionexecutive function can impact memory through poor initial encoding and organization, or due to a failure to discriminate relevant from irrelevant (or current from past) information. These deficits are often expressed as a vulnerability to interference or false positive errors (or in extreme cases, confabulation). It is important to distinguish these "memory" problems due to breakdown in the attention-executive from a primary amnestic disturbance. Disturbances in task monitoring can result in difficulty sustaining attention, characterized by an increase in all types of errors as well as reduced awareness of errors. In addition, individuals with TBI often complain of being easily distracted, being unable to return to a task after distraction, problems "shifting gears" or switching back and forth between tasks, and problems doing more than one thing at a time. These problems are often most apparent under conditions of increased cognitive load or complexity, such as responding to rapid, externally paced information or responding to multiple simultaneous task demands. Problems with sustained attention may appear as momentary lapses of attention ("mindwandering") or as increased performance variability due to less effective allocation of attention resources and goal-maintenance during task demands, with some evidence that these types of attentional difficulties reflect different attentional networks [62].

# Impairments of Emotional and Behavioral Regulation

In the emotional realm, TBI-related dysfunction can result from damage to those areas of the brain that are responsible for inhibiting the limbic system and the direct expression of emotions. This can result in a loss or decrease in the ability to regulate or control one's emotions. This "release" of emotional expression is often manifested as emotional lability or pathologic affect. Emotional dysregulation after brain injury is characterized by precipitous onset and rapid dissipation (both reflecting the absence of cognitive mediation of affective responding). This can cause a person to feel more extreme or quickly alternate between emotional highs and lows, or to become overwhelmed when expressing emotions. These deficits stem from underlying brain dysfunction, which distinguishes them from those reactions that reflect a purely emotional reaction to perceived impairment that could be expected in many people without brain dysfunction, such as grief, sadness, anxiety, or frustration. Moreover, the nature of the underlying brain dysfunction distinguishes these emotional reactions from those seen secondary to mental illness and psychiatric disorders.

Behaviorally, persons with TBI often fail to think before they act and show corresponding impulsivity, disinhibition, hyperverbosity, poor emotional control, distractibility, and cognitive inflexibility. These "positive" symptoms are indications of behavior that is stimulus-bound or overly determined by the environment. This can lead to behaviors that are rigid, impulsive, and poorly thought out. Although these excessive thoughts, behaviors, and emotions are common, problems can also manifest as more "negative" symptoms that appear to be the opposite of "positive" symptoms. Negative symptoms can include difficulty initiating tasks, low drive or motivation, apathy, impersistence, or a spontaneity—a constellation of symptoms that sometimes is referred to as adynamia [56].

It is also important to note that individuals with TBI who exhibit problems with emotional and/or behavioral regulation often perform within normal limits on neuropsychological measures of attention-executive functioning, but exhibit significant attention-executive problems in their everyday lives [63]. One reason for this is that neuropsychological assessments are administered in highly controlled environments and involve at least some structured task instruction, whereas attention-executive deficits are most apparent in novel, unstructured, or ambiguous situations that are characteristic of real-life functioning.

#### Metacognitive Impairments

Deficits involving metacognitive self-regulatory skills can contribute to a host of cognitive, behavioral, and emotional problems that impair internally driven goal-directed behavior. Cognitively, people with TBI may have problems monitoring their own thoughts, and therefore lack the internal feedback that is necessary to regulate and guide self-directed behavior. For example, individuals may have problems identifying relevant goals and maintaining progress towards completing goals. Other people may appear to persist with a certain strategy even though it is not working. This may be due to a failure in recognizing the need to adjust an unproductive approach (i.e., poor self-monitoring) or because of difficulties altering thinking or behavior in response to changing task or environmental demands (poor self-control). Metacognitive deficits involving an inability to accurately predict and evaluate one's performance on a given task can manifest as inaccurate self-appraisals of cognitive abilities, an inability to set realistic goals, or unwillingness to accept feedback from others. Moreover, an inability to accurately predict and evaluate one's performance on a given task can contribute to poor self-efficacy as well as reduced motivation and persistence in the face of challenging tasks. Deficits in self-regulating behaviors and emotions can be reflected in the co-occurrence of "positive" symptoms (e.g., impulsivity, disinhibition, distractibility, poor emotional control) and "negative" symptoms (e.g., difficulty initiating tasks, low drive or motivation) in the same individual [56]. For example, when a person with metacognitive deficits lacks external stimulation, a lack of self-regulatory control can manifest as symptoms of low motivation, apathy, and a failure to engage in internally driven, goal-directed behaviors. However, when the same individual is externally stimulated, a lack of self-regulatory control and the concomitant release of lower level more automatic behaviors can manifest as symptoms of impulsivity, disinhibition, emotional lability, and inflexibility [56].

### **Real-World and Clinical Implications**

Taken together, attention-executive impairments can have far-reaching consequences for the person with injury and their family members. These deficits can pose significant barriers to a person's ability to perform functional activities in everyday life (e.g., preparing meals, managing money, using transportation) and live independently [64–67]. Attention-executive dysfunction can also compromise full participation across a number of meaningful life situations, including the ability to work, attend school, manage home responsibilities, and engage in social/leisure activities [66–68].

The presence of attention-executive impairments also presents a significant challenge to the rehabilitation process. They can undermine the acquisition and application of compensatory strategies for other cognitive impairments (e.g., memory notebook training). Furthermore, attention-executive dysfunction in particular may limit the extent to which a patient is able to execute compensatory strategies for other cognitive impairments independently [69], particularly in novel situations or environments [70]. Thus, attention-executive dysfunction can interfere with the success and generalization of treatments for other acquired cognitive deficits. Clearly, there is a strong case for rehabilitation interventions aimed at improving attention-executive functions for persons with TBI.

# Interventions for Attention-Executive Impairments

Two primary approaches to intervention have been investigated in the remediation of attentionexecutive deficits in person with TBI: direct training and metacognitive training. Consistent with the overall goals of cognitive rehabilitation, both approaches are directed at effecting cognitive change with the ultimate goal of improving patients' functioning in meaningful contexts, including functional activities, participation, and quality of life [9–11]. However, direct training and metacognitive training interventions differ with regard to the theoretical approach to treatment; the proposed mechanism of action; and the emphasis and delivery of treatment activities. In this section, we will review the rational, evidencebased literature, and clinical recommendations for direct training and metacognitive training interventions. Because most widely used direct training interventions involve commercially available software and treatment programs that are not in the public domain, we will not describe the specific application of this type of approach. Readers should consult the research studies referenced in this chapter for further information regarding proprietary treatment programs/materials.

#### **Direct Training Interventions**

Direct training (also referred to as processspecific training, cognitive remediation, restitution training, or restorative approaches) refers to a broad class of bottom-up interventions that aim to directly restore specific attention-executive processes. This type of intervention is based on the assumption that attention-executive processes can be isolated (e.g., working memory, selective attention, divided attention) and selectively damaged. By activating and stimulating discrete attention-executive processes through a stimulus drill approach, training is hypothesized to result in direct improvement of the selectively targeted attention-executive functions [58, 71–73]. In turn, it is predicted that patients will demonstrate improvements on untrained tasks that also involve the "trained" or targeted cognitive function, and ultimately, in their everyday life activities.

Training activities typically involve computerized or paper or pencil administration of repetitive drills or exercises (e.g., detecting targets in the presence of distracters, sorting words in alphabetical order) that are presented in a hierarchical manner so that tasks become increasingly difficult and remain challenging as patients demonstrate improvement [73]. Some direct training interventions target an individual skill, such as working memory or divided attention [74–79], while other interventions are more comprehensive and offer stand-alone exercises that target a range of attention-executive skills [75, 80]. For example, the Attention Process Training (APT), a commercially available program developed by Sohlberg and Mateer [81], is based on the author's clinical model of attention and includes exercises for five different types of attention that may be impaired following TBI: focused, sustained, selective, alternating, and divided.

The evidence-based literature is generally supportive of direct training interventions, with the caveat that this approach should be used in conjunction with metacognitive training [9–11, 58], which is detailed in the following section of this chapter. As we have discussed previously, deficits in metacognitive self-regulatory skills, including the ability to monitor and control one's thoughts and behaviors, are common after TBI. From a theoretical perspective, incorporating metacognitive training (e.g., feedback, self-monitoring, strategy training) should enhance the potential benefit of direct training by teaching individuals strategies to regulate their own thoughts and behaviors that can be applied outside the laboratory or clinical setting. Given the decontextualized nature of direct training (i.e., completing abstract exercises in the absence of meaningful contexts), metacognitive training that incorporates extensive practice in applying strategies in multiple real-world contexts is also critical to transfer and generalization of acquired skills. Research also supports the importance of combining direct attention training with metacognitive strategy training [58, 82, 83]. For instance, findings from a systematic review of the TBI direct attention training literature suggested that the most robust improvements following treatment were observed in studies that also included training in metacognitive strategies [58]. These recommendations have been echoed by Cicerone and colleagues who have emphasized that clinicians who use a direct training approach should use it in conjunction with a metacognitive training to promote the acquisition of specific compensatory strategies that can be applied in meaningful realworld situations [9-11]. As an example, the most recent version of the Attention Process Training program (APT-3) [84] also involves metacognitive self-regulation training as a critical component of treatment; however, it is important to note that the efficacy of APT-3 for persons with TBI has yet to be determined.

Due to advances in our understanding of neuroplasticity-that is, the ability of the nervous system to respond to internal and external stimuli by reorganizing its structure, functions, and connections, as well an increased emphasis on research that translates neuroplasticity research into clinical practice [85], a growing literature has investigated the efficacy of automated, computerized direct training programs to improve working memory in patients with brain injury [75, 76, 78, 79]. These studies have made explicit claims to be rooted in the mechanics and potential of neuroplastic changes in the brain [59]. In a seemingly opposite direction from the evidencebased clinical practice recommendations set forth by the Cognitive Rehabilitation Task Force of the American Congress of Rehabilitation Medicine (ACRM) Brain Injury-Interdisciplinary Special Interest Group (BI-ISIG) [9–11], this type of intervention has minimal therapist involvement, as well as no instruction related to strategies or application to real-world activities. Rather, these "neuroplasticity-based interventions" are based on the premise that direct training alone has the potential to "lead to a less diffuse pattern of cortical activity or redistribution of neural activities within the network areas," [75] which in turn will contribute to restoration of targeted attentionexecutive functions. However, it is important to note that many of the demands that are inherent in "neuroplasticity-based" computerized cognitive training are also critical aspects of therapistdirected interventions: active engagement in the treatment process, attention to the relevant aspects of the treatment situation, ongoing provision of feedback, adaptive adjustment of task difficulty based on prior performance, and a planned progression of treatment demands.

Similar to findings reported in the earlier direct training literature, initial evidence from these plasticity-focused studies has shown improvements on untrained attention-executive tasks [75, 76, 78, 79] and patient-rated cognitive symptoms [75, 76, 79]. Using fMRI, Kim and colleagues also demonstrated changes in cortical activation following computerized neuroplasticity based training in a sample of patients with TBI [75]. This is suggestive of training-related brain plasticity; however, this line of research is still in its infancy and implications regarding these training-induced patterns of cortical activation remain open to various interpretations [13, 59]. Chen and colleagues [13] have noted that increased cerebral activation can represent the recruitment of remote areas into a functional system, compensatory changes in brain activation, effects of effort and motivation, or maladaptive forms of cerebral plasticity. Decreased activation may represent a failure of recruitment, increased neural efficiency, or automatic processing. It is also unknown if changes in functional cerebral activation are related to improvements in patients' everyday lives.

Despite these initial positive findings, several factors may limit the applicability of automated,

computerized neuroplasticity-based training. First, the training has been described as "intense, demanding and tiring" [76] requiring participants to initiate and sustain about an hour a day of cognitive exercise, 5 days a week. Thus, this type of training may only be appropriate for patients with a high level of motivation, compliance, commitment, and stamina [59]. In addition, there is evidence that the treatment is most effective for patients with relatively preserved cognitive functioning, suggesting it may be inappropriate for patients with more severe impairments [76]. The lack of therapist involvement and the impersonal nature of the treatment may be inappropriate and unappealing for many patients. Lastly, from a theoretical perspective, automated, computerized direct training interventions are limited in that they addresses only attention-executive cognitive functions and fail to consider patients' emotional and behavioral concerns, which can influence the outcome of treatment (although it might be argued that some capacity for frustration tolerance is embedded in the training). Moreover, to the extent that direct training is capable of strengthening specific cognitive skills, a lack of explicit instruction in metacognitive strategies along with the decontextualized intervention (i.e., completing abstract exercises in the absence of meaningful contexts) suggests that individuals may have difficulty applying these skills outside of the laboratory or clinical setting.

In summary, the findings from direct training interventions have been generally positive; however, we stress that the use of direct training without therapist involvement and without training inmetacognitive strategies is not recommended. This is consistent with practice recommendations of the BI-ISIG Cognitive Rehabilitation Task Force that direct training should also involve metacognitive training to promote development of compensatory strategies and foster generalization to real-world tasks [9–11]. Based on emerging evidence regarding plasticity-based computerized training, the BI-ISIG Cognitive Rehabilitation Task Force also recommended that computer-based interventions may be considered as an adjunct to clinician-guided treatment for the remediation of attention deficits after TBI [86]. However, the Task Force emphasized that the sole reliance on repeated exposure and practice on computerbased tasks without involvement and intervention by a therapist is not recommended.

#### Metacognitive Training Interventions

A second broad approach to the remediation of attention-executive dysfunction involves metacognitive training-an umbrella term that applies to many top-down interventions that emphasize capacity for self-regulation. As we have discussed earlier, metacognitive self-regulatory processes, which are often impaired following TBI, involve the ability to monitor and control one's thoughts, emotions, and behaviors. Metacognitive processes allow for the execution of self-directed, complex behaviors by supporting a set of skills, including (1) identifying and setting goals, including anticipating task demands; (2) selfmonitoring and comparing performance with goals or outcomes; (3) making decisions to change one's behavior or select an alternative approach to a situation; and (4) executing the change in behaviors [12, 87]. The goal of metacognitive training interventions is to reestablish these metacognitive processes in an effort to improve a patient's ability to exercise control over his or her behaviors and the cognitive functions that support or inhibit them. Towards this end, the therapist attempts to remove obstacles that interfere with this self-control. These obstacles can be cognitive, emotional, or behavioral. Often they comprise all three. This is accomplished through the training and internalization of metacognitive strategies that involve direct, step-by-step instruction to teach individuals how to regulate their own behavior [12, 87, 88]. Rather than train specific skills, metacognitive strategies can be used in a variety of different situations, thus enhancing the potential for generalization of behavioral improvements to novel, real-world contexts [13, 56, 60, 82].

Although the primary goal of metacognitive training is to enhance a person's ability to internalize awareness and control over his or her behavior, treatment usually begins with external cuing of a general rule or principle for the particular metacognitive strategy. Accordingly, patients are taught the process of self-monitoring and self-control by means of education, modeling, external directions, instructional handouts, and cuing. Over time, through practice and implementation in a variety of settings, this external strategy can become internalized, under the self-generated direction of the patient, including the cuing, structure, and execution involved in the strategy. Thus, what begins as an external strategy becomes an internal strategy.

Metacognitive strategies may be relatively simple or complex. For example, training a patient in the use of a simple problem-solving sequence is a simple strategy that may be appropriate for those patients with moderate to severe impairment. Other metacognitive training interventions are more complex, often involving multiple steps or targeting multiple problems. For instance, complex interventions may involve simultaneous training in self-regulation of cognitive and emotional processes. Complex strategies have the advantage of providing a much wider range of therapeutic challenges and experiences for a patient and may allow for the training of a wider range of target behaviors and situations, covering a wider range of applicability, and allowing for greater generalization and transfer of training; however, these strategies may be usable only by those with relatively mild to moderate impairment.

In the TBI rehabilitation literature, the majority of metacognitive training interventions have focused on improving general problem-solving skills [89–92], or more specific activities that are involved in problem solving, including goal management [93–96]; planning and self-monitoring [55, 56]; problem solving that involves time pressure management [97, 98]; and attentional selfregulation [93, 96, 99]. A recent investigation of metacognitive strategy training conducted by Spikman and colleagues [92] was unique in that it involved a multifaceted approach targeting eight different skills including, self-awareness, goal setting, planning, self-initiation, selfmonitoring, self-inhibition, flexibility, and strategic behavior. Boelen and colleagues [57] noted that this intervention appears to address many of the components of Stuss' theoretical framework [8]. Moreover, the multifaceted intervention was successful in improving the amount and quality of daily life functioning across the domains of vocational functioning, social interaction, leisure activities, and mobility.

In addition to the cognitive aspects of problem solving, current interventions based on metacognitive strategy training have also addressed emotional aspects of behavioral regulation [60, 91]. In order to solve problems in a cognitively effective manner, individuals have to be able to control those emotional reactions that interfere with their ability to think clearly and effectively. These reactions are often associated with "negative self-talk" in the form of cognitive distortions or misattributions. Examples include: "I have to do a perfect job"; or "I'm a failure"; or "I have to be in complete control or it will be a disaster." Rath and colleagues [91] evaluated the effectiveness of an innovative intervention that focused on the development of emotional self-regulation strategies as the basis for maintaining an effective problem orientation, along with a "clear thinking" component that included cognitivebehavioral training in problem-solving skills. The intervention also involved a systematic process for analyzing real-life problems and roleplay of real-life examples of problem situations. The intervention group demonstrated improvements on measures of executive functioning, self-appraisal of "clear thinking," self-appraisal of emotional self-regulation, and objective observer-ratings of interpersonal problem-solving behaviors in naturalistic simulations.

In summary, there is substantial evidence to support the use of metacognitive training for persons with TBI [9–12, 57]. Based on the current evidence, the BI-ISIG Cognitive Rehabilitation Task Force of ACRM has also recommended, as a practice standard, metacognitive strategy training for deficits in executive functioning after TBI, including impairments of emotional selfregulation [11]. As detailed in a systematic review and meta-analysis, metacognitive training for problem solving of personally relevant activities has demonstrated significant effect sizes in impairment, activity, and participation outcomes compared to control treatments and was recommended to improve problem-solving deficits following TBI [12]. The BI-ISIG Cognitive Rehabilitation Task Force of ACRM has come to a similar conclusion and has recommended, as a practice guideline, training in formal problemsolving strategies and their application to everyday situations and functional activities during post-acute rehabilitation after TBI [11].

From a theoretical and clinical perspective, an important benefit of metacognitive training is that these strategies can be customized rather easily to address problems with attention-executive, cognitive, emotional/motivational, and behavioral functioning. Cicerone and colleagues [60] have suggested that metacognitive training directed at improving patients' self-regulation of both cognitive and emotional processes leads to increases in patients' self-efficacy beliefs, specifically in their confidence in managing residual cognitive and emotional symptoms. Improvements in perceived self-efficacy (and related concepts, such as maintaining a positive problem orientation) are related to positive outcomes [61, 91], particularly patients' subjective well-being and life satisfaction [100]. In addition, metacognitive training can be applied to clients' personally relevant goals, which will enhance acquisition and generalization of strategies. This is also important as training that has meaning to the client may increase motivation and satisfaction with treatment.

# Clinical Application of Metacognitive Training

In the section, we describe a general framework that is applicable to the majority of metacognitive training interventions. We also describe self-talk procedures—a simple metacognitive strategy that can be adapted to address multiple problems depending on the needs of the client. This is followed by a description of the metacognitive treatment of problem-solving deficits.

# A General Algorithm for Metacognitive Strategy Training

Most interventions in this area follow a common structure, which parallels the structure of executive functioning: the creation of an internal plan or representation in anticipation of a desired goal state; execution of a response or sequence of responses; and the use of feedback to compare the internal plan with the achieved outcome, and modification of one's plan accordingly. A general algorithm involving the following sequence of steps can be applied to many different metacognitive training interventions: (1) raising awareness of deficits; (2) anticipation and planning of the potential response to novel tasks or problemsolving situations; (3) implementation and selfmonitoring of selected responses; and (4) evaluation of the outcome of the response, comparing the outcome with the desired or anticipated outcome, and changing the approach of the task, if necessary, which then "resets" the sequence of operations.

While the specific language and aspects of interventions vary, this structure and process is common to most metacognitive and problemsolving interventions for impairments of attention-executive functioning and can serve as framework for understanding and implementing these various interventions. The general steps outlined below were utilized in studies by Von Cramon et al. [89], Fasotti et al. [97], Levine et al. [95], and Cicerone et al. [60] to address general problem-solving impairments. These steps were also utilized in the single-case studies of Ylvisaker and Feeney [101] and Dawson et al. [102], using a Goal-Plan-Do-Review strategy. Similar steps have also been utilized by Goverover et al. [103], and Cheng and Man [104] to address problems with self-awareness.

#### Awareness

In clinical practice, metacognitive training is often preceded by attempts to assess and foster the patient's awareness of deficits, identifying the relevant strategies and setting goals.

Effective treatment, particularly involving metacognitive training interventions, usually

requires assisting the patient to develop an awareness of the underlying impairments and their negative functional consequences. For those with awareness deficits, it is generally helpful to help a patient to recognize deficits by pointing out the discrepancies between self-perception and reality. Fleming and Ownsworth [21] also recommend: (1) selecting key tasks and environments in which awareness behaviors are most important within everyday activities and roles; (2) providing clear feedback and structured opportunities to help patients evaluate their performance, discover errors, and compensate for deficits; (3) using habit formation, when necessary, through repetition and procedural or implicit learning; and (4) providing education and environmental supports. Patients may be asked to predict explicitly the expected outcome of their behavior, e.g., predicted number of errors, accuracy, speed to completion, or some other aspect of success or failure. After completing the task, the patient is asked to compare his or her performance to the actual scores obtained on the task [103–105]. This allows for comparison of expected and observed outcomes, an activity that can facilitate awareness and improve self-monitoring.

Formal intervention should be considered for individuals with neurocognitive unawareness. Readers are referred to Chap. 12 of this book and also the Cognitive Rehabilitation Manual by Edmund Haskins and colleagues [106] for a thorough description of awareness interventions. Treatment to address awareness is an inherent aspect of metacognitive strategy training and should be incorporated into essentially all interventions, not only to foster increased awareness of deficits but also to foster awareness of the use and application of strategies. Crosson and colleagues [107] provide a framework for relating specific compensatory strategies to different levels of impaired awareness.

Engaging the patient in the process of overt goal setting is another aspect of this stage of treatment [108]. In practice, the setting of goals for rehabilitation will occur simultaneously with the clinical assessment of a patient's intellectual awareness of their identified "deficits" and their implications for daily functioning. Patients' intellectual awareness, goal orientation, and identification of compensatory strategies all represent aspects of *metacognitive knowledge* [107, 109]. The concept of metacognitive knowledge has also been extended to include self-efficacy beliefs [60, 109].

#### **Anticipate and Plan**

This aspect of metacognitive strategy training is grounded in the view that the anticipation of events in the environment, preparation of a response, and expectations about the consequences of behavior are central aspects of attention-executive functioning. To anticipate something makes it more likely that you will attend to it if and when it occurs (and to notice if it does not). The use of a prediction paradigm can be an explicit aspect of this process, not only to increase anticipatory awareness [107], but to establish a structure to mediate the patient's approach to the task [56]. Patients are assisted with the processes of *task setting*, including setting the appropriate criteria for stimulus relevance and preparing for stimulus selection, response selection, and response inhibition. Selfinstructional techniques are another practical application of these principles. For example, prior to initiating a given task, the patient is instructed to identify the demands associated with the task, and to plan the appropriate sequence of responses. A relatively simple self-instructional metacognitive strategy involves having the patient engage in a self-talk procedure by which patients are taught to "talk themselves through" tasks [55, 56]. This serves to prevent unwanted behaviors, while simultaneously encouraging planning and self-monitoring and attentional focus. Training occurs in three stages to promote the progressive internalization of verbal selfregulation. Training begins with overt verbalization (talking out loud), then transitions to faded verbal self-instruction (whispering), and finally to covert verbal mediation(inner talk).

Cicerone and Wood [55] found that this threestage, self-talk procedure described above resulted in reduced task-related errors and also eventual cessation of off-task behaviors. Additional instruction and practice in the application of the strategy was also associated with increased spontaneous use of the strategy in novel situations. Cicerone and Giacino [56] subsequently evaluated training using the same selftalk protocol with five patients with TBI and one patient with falx meningioma. All patients exhibited impaired planning and self-monitoring. Five of the six patients showed marked reduction of task-related errors and perseverative responses.

At a more complex level, patients are asked to conduct an activity analysis that anticipates not only characteristics of the task but the potential impact of one's cognitive abilities, emotional responses, and environmental supports on their performance of the task as well as examining their self-efficacy belief in relation to managing these cognitive, emotional, and task demands [60]. The anticipation of situations that are likely to cause "information overload" and preparation of a plan to manage these situations is an example of this process, applied to Time Pressure Management [98].

#### **Execute and Self-Monitor**

Next, the person implements his/her plan to perform the task, actively self-monitors performance and use of strategies throughout, all of which reflect the process of task-monitoring. In most situations, the demands to execute and selfmonitor the patient's behavior occur simultaneously and require some capacity to shift attention between the requirements of the task and monitoring one's internal train of thought. Emphasis is placed on the patient's capacity for online monitoring [109] and emergent awareness of performance [107]. Errors are most likely to occur during this process of task execution: errors due to failure to anticipate some aspect of a situation; errors related to inability to maintain a consistent response set; attentional lapses related to one's mind wandering, errors because things do not "go as planned"; errors because something "captured" one's attention or failed to gain one's attention; and errors related to emotional interference. It is not surprising that error management training can be an essential component of selfregulation during this phase, including the training of both metacognitive and emotional regulation

strategies [24, 60, 110, 111]. The use of "errorful learning"—although also more effortful—can be beneficial in situations with inconsistent mapping of stimulus–response contingencies, to foster strategy application in situations with varied demands, and to facilitate transfer of learning, including functional activities [112, 113]. The therapist may need to direct attention and support to the patient's online monitoring of errors or problems and application of compensatory strategies at the appropriate time and situations (aspects of *metacognitive experience*).

The principles identified earlier in relation to neuroplasticity also apply to metacognitive strategy training, such as the need to continually adapt the task to maintain an appropriate level of success and challenge. The therapist should determine the amount of structure and cuing provided at this level of intervention to facilitate online monitoring (e.g., awareness and correction of errors) and strategy application, with the patient assuming greater responsibility for independent performance over the progression of treatment. An approach to compensatory strategy training based on the instructional process in natural contexts [114] varies the level of assistance provided (rather than changing aspects of the task itself) as a basis for continually adapting task demands. The therapist serves a mediating function to encourage self-monitoring and strategy application [115]. This approach allows the therapist to control those elements of the task that are initially beyond the patient's capacity, reinforcing attention to those elements within the patient's range of competence and emphasizing the patient's need to recognize and adjust to varied demands as a common occurrence. Errors are expected to occur as the therapist transfers responsibility for the task to the patient, and these occurrences provide the principal basis for determining the patient's competence, adjusting the level of intervention, and providing feedback.

#### Self-Evaluate

Finally the person compares the effectiveness of their actions with the predicted effects and consequences and evaluates their performance (typically while incorporating feedback from the therapist and/or others). This metacognitive evaluation process can be directed at the patient's immediate performance, compared with prior expectations, and related to the relevance and implications of deficits (or strengths) to the person's daily functioning. Therapeutic feedback can serve as the basis for the patient's modifying their awareness, adjusting their goals, and identifying the need to adjust the nature and application of compensatory strategies. The process of self-evaluation thus feeds back to the processes of awareness and goal setting.

# Metacognitive Training for Problem-Solving Deficits

The treatment of problem-solving deficits using metacognitive strategies involves teaching patients to gain control over their cognitive processing by learning and following a formal problem-solving strategy. One example is the Goal-Plan-Do-Review sequence taken from Ylvisaker and Feeney (1998) [101]. However, it should be noted that any series of steps that reflect the evidence-based algorithm described earlier (i.e., Awareness, Plan, Execute, Self-Evaluate) can be used.

Patients are instructed to apply the Goal-Plan-Do-Review strategy to each new problem they face. Initially, patients are taught to complete a written, structured worksheet which outlines each step of the sequence. Through frequent repetition of this procedure across a range of tasks, patients can learn to apply the problemsolving strategy more effectively and quickly.

The long-term goal of problem-solving training is to enable the patient to become familiar and skilled with the problem-solving sequence that it is generalized to various situations with minimal external cuing. In effect, through practice and repetition it becomes an internalized strategy. For those patients who are unable to achieve internal control over their problemsolving attempts, an approach that relies on external cuing (e.g., external prompts from therapist or caregiver; using structured worksheets) may be necessary.

# Case Example Illustrating the Use of Metacognitive Strategy Training

EF is a 44-year-old woman who sustained a TBI when she fell and was kicked in the head by a horse. She sustained a loss of consciousness with hemorrhagic contusions in the left dorsolateral frontal and bilateral inferior medial and lateral frontal lobes as well as an intraparenchymal lesion in the anterior cingulate. She received a course of acute rehabilitation and was discharged to her home (with her husband and two adolescent daughters). She was working as an attorney prior to her injury, but did not return to work. Her family noted that prior to her injury she had "held the family together" but since then she was more emotionally labile and would sometimes perseverate on an activity, such as asking family members repeatedly what they wanted for dinner and obsessively cleaning up after her husband and children. Two years after her injury she was offered the opportunity to work at her former firm on a part time basis as a "legal assistant" doing background research for other attorneys. After returning to work she had difficulty finishing tasks, often getting involved in tasks with less priority. Her husband expressed his concern to her neurologist and she was referred for neuropsychological evaluation and treatment.

When first seen, she generally attributed her problems on her job to having an "unreasonable amount of work," but she did acknowledge having difficulties because she would lose track of what she was doing while working on the computer or reading files, "train of thought goes off and then I lose everything," and she would "get stuck" and not be able to figure out another way of doing something. Neuropsychological evaluation was notable for intact processing speed but marked decline with dual task demands. On basic tasks of sustained attention she responded rapidly but missed targets, particularly on the later trials. On more complex tasks she still responded quickly but had difficulty establishing the correct set, and she would perseverate on an inappropriate sequence of responses early in the task. Her memory was average but she did exhibit increased susceptibility to both proactive and retroactive interference. The initial course of treatment was based on the use of verbal mediation as a form of self-instructional training, applied to a series of "transfer" problems (similar to the Tower of London or Tower of Hanoi) [55]. A baseline was established, in which it was noted that her latencies prior to starting each trial were quick, and unrelated to task difficulty, and she would initially make an extensive number of errors before realizing there was another, more efficient, solution to the task. In the initial stage of the intervention EF was instructed to verbalize the steps required to complete the task prior to any overt attempt at problem solution. She was prompted to anticipate or recognize errors during this process, and this was contrasted with her propensity to make errors without recognizing them during her baseline task performance. EF initially found this process "uncomfortable" and "more difficult," which was interpreted in relation to her need to "slow down" and adopt a more conscious, deliberate approach that contrasted with the difficulties she encountered due to her automatic, impulsive responding. Following her verbalization she was asked to complete the task, while again verbalizing the steps (and verbally noting any errors). She was provided multiple practice trials on the task following the same structure, while fading her overt verbal self-instruction. This process was used to formulate a more general (metacognitive) strategy reflecting her need to "stop, think and plan" (STP) while performing an activity, following the general algorithm described above. EF was then given the opportunity to practice the metacognitive strategy on multiple tasks having similar problem structure and demands but with varied content and varied levels of difficulty, to reinforce the near transfer of strategy use to a variety of situations. The intervention initially was applied to "artificial" tasks within the safety of the therapeutic environment; these same strategies were gradually applied to situations in EF's daily functioning, including her work responsibilities. During this process a distinction was made between "error recognition" and "error utilization," the latter emphasizing the practical (positive) value of recognizing errors as an opportunity to correct her

mistakes. Interventions to facilitate errormanagement included feedback, open-ended prompts ("Is there anything else you can do? Are you sure?"), and modeling "correct" performance and positive self-statements [24, 61]. Throughout this process, she was encouraged to identify past experiences where she had successfully carried out an activity and to relate these to her current demands [116]. The emphasis of treatment sessions shifted from table-top exercises to analyzing examples from her daily life where she experienced difficulty, and simulating these functional activities within the treatment setting. Her use of the "stop, think and plan" strategy was gradually expanded to include formal aspects of "goal-plando-review" [102] and time pressure management [98]. These sessions also provided the opportunity to address her moment-to-moment self-monitoring and error management in relation to her difficulties sustaining attention in her daily functioning, reflected in her subjective complaints that her "train of thought goes off" and she loses track of what she is doing, and she "gets stuck" on one thing and cannot figure out another way of doing something. She could identify that this was particularly evident when she was attempting to conduct research as part of her work, which typically involved managing multiple cases and requests for information under time demands. The apparent contradiction between the extremes of her being "easily distracted" and "getting stuck on one thing" was interpreted in relation to a common, underlying problem of "dysregulation" [56] reflecting her need to regulate her attention more effectively in a variety of situations. Treatment for this aspect of her functioning was again introduced by incorporating structured attention exercises and functional tasks within sessions, providing an opportunity for monitoring attention lapses and task-unrelated thoughts. She was educated regarding the relationship between sustained attention and error awareness [117, 118]. The method of "content-free" cuing [119] was introduced as a means of self-monitoring whether she was "doing what I need to be doing" at intermittent intervals during task performance. This procedure was again applied to a variety of tasks, with increasing functional application (e.g., using the Internet for research) and gradually transferred to strategy use during her daily activities (including programming an auditory cue to occur at random intervals on her Smartphone). During this progression EF was also assisted in identifying signs of emotional reactivity (which within sessions manifested as her feeling frustrated and overwhelmed) that interfered with task performance. The use of a Likert-like Cognitive Energy Scale (CES) was introduced as a method for emotional self-monitoring and self-regulation [60, 110]. Her emotional reactions and attentional lapses were reinterpreted from representing negative, disruptive events to representing "stop" cues that signaled the need for her to institute a compensatory strategy. Over time, she was able to employ the STP and CES as "on-line" cognitive and emotional strategies to "reset" her attention to relevant aspects of the situation and when she became distracted, "stuck," or overwhelmed.

The latter progression of treatment was directed at generalizing the use of strategies in her daily life. During this extended period of treatment she was given multiple opportunities to apply her strategies and increasing responsibility for recognizing the need and implementing strategies in her daily functioning [55]. The Goal-Plan-Do-Review strategy was used as a way for her to plan and organize her daily activities, such as prioritizing work tasks for each day and a weekly menu for her home. Treatment for behavioral and emotional regulation shifted emphasis from the routine use of specific problem solving and emotional regulation strategies, to reinforcing the more general psychological aspects of self-appraisal-maintaining a positive problem orientation and keeping emotional reactions from interfering with her functioning-that allowed her to establish an acceptable sense of identity [120]. After several months of treatment she demonstrated the ability to apply metacognitive strategies for practical problem solving, attention regulation, and emotional regulation. She was "less emotional" and less likely to perseverate on household tasks in her daily functioning, although she remained less sociable. She was able to complete her household and work responsibilities more effectively, although this remained more effortful and she continued to

function at a lower capacity than she did prior to her injury, and she had the advantage of a supportive environment both at home and at work. The improvements in her ability for self-regulation were accompanied by improvements in her selfefficacy and confidence that she could manage her cognitive and emotional symptoms.

This case illustrates the clinical application of evidence-based cognitive rehabilitation to a disturbance of the anterior attention-executive networks after a moderate-severe TBI. Although interventions are typically evaluated in isolation, in practice it is common to use multiple intervention methods simultaneously or in a progression [115], as seen in this case illustration. The patient exhibited significant problems with attention and executive functioning, but had other favorable prognostic signs including above average intelligence and education, relatively intact memory, at least an intellectual awareness of her problems, good motivation, and some capacity to recognize her mistakes and apply compensatory strategies, or to use a strategy in specific situations, and a supportive environment. Under these favorable circumstances, treatment based on metacognitive strategy training enabled her to regulate those aspects of her functioning that supported a positive identity.

Acknowledgement Preparation of this chapter was partially supported by U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020.

#### References

- Cicerone, K. D. (1996). Attention deficits and dual task demands after mild traumatic brain injury. *Brain Injury*, 10, 79–89.
- Draper, K., & Ponsford, J. (2008). Cognitive functioning ten years following traumatic brain injury and rehabilitation. *Neuropsychology*, 22, 618–625.
- Duncan, J. (1986). Disorganization of behaviour after frontal lobe damage. *Cognitive Neuropsychology*, 3, 271–290.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.
- 5. Luria, A. R. (1973). *The working brain*. New York: Basic Books.

- Shallice, T. (1982). Specific impairments of planning. *Philosophic Transactions of the Royal Society* of London B, 298, 199–209.
- Chen, A. J., & D'Esposito, M. (2010). Traumatic brain injury: From bench to bedside to society. *Neuron*, 66, 11–14.
- Stuss, D. T. (2011). Functions of the frontal lobes: Relation to executive functions. *Journal of International Neuropsychological Society*, 17, 759–765.
- Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., Bergquist, T. F., et al. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation*, 81, 1596–1615.
- Cicerone, K. D., Dahlberg, C., Malec, J. F., Langenbahn, D. M., Felicetti, T., Kneipp, S., et al. (2005). Evidencebased cognitive rehabilitation: Updated review of the literature from 1998 through 2002. *Archives of Physical Medicine and Rehabilitation*, 86, 1681–1692.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., et al. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation*, 92, 519–530.
- Kennedy, M. R., Coelho, C., Turkstra, L., Ylvisaker, M., Moore Sohlberg, M., Yorkston, K., et al. (2008). Intervention for executive functions after traumatic brain injury: A systematic review, meta-analysis and clinical recommendations. *Neuropsychological Rehabilitation*, 18, 257–299.
- Chen, A. J., Abrams, G. M., & D'Esposito, M. (2006). Functional reintegration of prefrontal neural networks for enhancing recovery after brain injury. *The Journal* of *Head Trauma Rehabilitation*, 21, 107–118.
- 14. Cicerone, K., Levin, H., Malec, J., Stuss, D., & Whyte, J. (2006). Cognitive rehabilitation interventions for executive function: Moving from bench to bedside in patients with traumatic brain injury. *Journal of Cognitive Neuroscience*, 18, 1212–1222.
- 15. Baddeley, A. (1992). Working memory. *Science*, 255, 556–559.
- Baddeley, A. D. (1986). Working memory. Oxford, England: Oxford University Press.
- Stuss, D. T. (2011). Traumatic brain injury: Relation to executive dysfunction and the frontal lobes. *Current Opinion in Neurology*, 24, 584–589.
- Kennedy, M. R., & Coelho, C. (2005). Selfregulation after traumatic brain injury: A framework for intervention of memory and problem solving. *Seminars in Speech and Language*, 26, 242–255.
- Chiou, K. S., Carlson, R. A., Arnett, P. A., Cosentino, S. A., & Hillary, F. G. (2011). Metacognitive monitoring in moderate and severe traumatic brain injury. *Journal of International Neuropsychological Society*, 17(4), 720–731.
- Ciurli, P., Bivona, U., Barba, C., Onder, G., Silvestro, D., Azicnuda, E., et al. (2010). Metacognitive unawareness correlates with executive function
impairment after severe traumatic brain injury. *Journal of International Neuropsychological Society*, *16*, 360–368.

- Fleming, J. M., & Ownsworth, T. (2006). A review of awareness interventions in brain injury rehabilitation. *Neuropsychological Rehabilitation*, 16, 474–500.
- Hart, T., & Evans, J. (2006). Self-regulation and goal theories in brain injury rehabilitation. *The Journal of Head Trauma Rehabilitation*, 21, 142–155.
- Ownsworth, T. (2010). A metacognitive contextual approach for facilitating return to work following acquired brain injury: Three descriptive case studies. *Work*, 36, 381–388.
- Ownsworth, T., Quinn, H., Fleming, J., Kendall, M., & Shum, D. (2010). Error self-regulation following traumatic brain injury: A single case study evaluation of metacognitive skills training and behavioural practice interventions. *Neuropsychological Rehabilitation*, 20, 59–80.
- 25. Ownsworth, T. L., Fleming, J., Shum, D., Kuipers, P., & Strong, J. (2008). A randomised controlled trial (RCT) for facilitating goal attainment and improving psychosocial function following acquired brain injury: Comparison of three intervention formats. *Journal of Rehabilitation Medicine*, 40, 81–88.
- Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, 13, 25–42.
- Mesulam, M. M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Annals of Neurology*, 28, 597–613.
- Fan, J., McCandliss, B. D., Fossella, J., Flombaum, J. I., & Posner, M. I. (2005). The activation of attentional networks. *NeuroImage*, 26, 471–479.
- Posner, M. I., Petersen, S. E., Fox, P. T., & Raichle, M. E. (1988). Localization of cognitive operations in the human brain. *Science*, 240, 1627–1631.
- Fan, J., Gu, X., Guise, K. G., Liu, X., Fossella, J., Wang, H., et al. (2009). Testing the behavioral interaction and integration of attentional networks. *Brain* and Cognition, 70, 209–220.
- Posner, M. I., & Fan, J. (2004). Attention as an organ system. In J. R. Pomerantz & M. C. Crair (Eds.), *Topics in integrative neuroscience: From cells to cognition*. Cambridge, England: Cambridge University Press.
- Posner, M. I., Rothbart, M. K., Sheese, B. E., & Tang, Y. (2007). The anterior cingulate gyrus and the mechanism of self-regulation. *Cognitive, Affective,* & *Behavioral Neuroscience,* 7, 391–395.
- 33. Niogi, S., Mukherjee, P., Ghajar, J., & McCandliss, B. D. (2010). Individual differences in distinct components of attention are linked to anatomical variations in distinct white matter tracts. *Frontiers in Neuroanatomy*, 4, 2.
- Niogi, S. N., Mukherjee, P., Ghajar, J., Johnson, C. E., Kolster, R., Lee, H., et al. (2008). Structural dissociation of attentional control and memory in adults

with and without mild traumatic brain injury. *Brain*, 131, 3209–3221.

- 35. Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, 14, 340–347.
- Callejas, A., Lupianez, J., & Tudela, P. (2004). The three attentional networks: On their independence and interactions. *Brain and Cognition*, 54, 225–227.
- Corbetta, M., Patel, G., & Shulman, G. L. (2008). The reorienting system of the human brain: From environment to theory of mind. *Neuron*, 58, 306–324.
- Teuber, H. L. (1964). The riddle of frontal lobe function in man. In J. M. Warren & K. Akert (Eds.), *The frontal cortex and behavior* (pp. 410–444). New York: McGraw-Hill.
- Ghajar, J., & Ivry, R. B. (2008). The predictive brain state: Timing deficiency in traumatic brain injury? *Neurorehabilitation and Neural Repair*, 22, 217–227.
- 40. Stuss, D. T., Binns, M. A., Murphy, K. J., & Alexander, M. P. (2002). Dissociations within the anterior attentional system: Effects of task complexity and irrelevant information on reaction time speed and accuracy. *Neuropsychology*, *16*, 500–513.
- Petersen, S. E., & Posner, M. I. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience*, 35, 73–89.
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience*, *3*, 201–215.
- Dosenbach, N. U., Fair, D. A., Cohen, A. L., Schlaggar, B. L., & Petersen, S. E. (2008). A dualnetworks architecture of top-down control. *Trends in Cognitive Sciences*, 12, 99–105.
- 44. Dosenbach, N. U., Fair, D. A., Miezin, F. M., Cohen, A. L., Wenger, K. K., Dosenbach, R. A., et al. (2007). Distinct brain networks for adaptive and stable task control in humans. *Proceedings of the National Academy of Sciences of the United States of America, 104*, 11073–11078.
- 45. Milham, M. P., Banich, M. T., Claus, E. D., & Cohen, N. J. (2003). Practice-related effects demonstrate complementary roles of anterior cingulate and prefrontal cortices in attentional control. *NeuroImage*, 18, 483–493.
- 46. Sheth, S. A., Mian, M. K., Patel, S. R., Asaad, W. F., Williams, Z. M., Dougherty, D. D., et al. (2012). Human dorsal anterior cingulate cortex neurons mediate ongoing behavioural adaptation. *Nature*, 488, 218–221.
- Burgess, P. W., Dumontheil, I., & Gilbert, S. J. (2007). The gateway hypothesis of rostral prefrontal cortex (area 10) function. *Trends in Cognitive Sciences*, 11, 290–298.
- Levine, B., Turner, G. R., & Stuss, D. T. (2008). Rehabilitation of frontal lobe functions. In D. T. Stuss, G. Winocur, & I. H. Robertson (Eds.), *Cognitive neurorehabilitation: Evidence and appli-*

*cation* (pp. 464–486). Cambridge, England: Cambridge University Press.

- Stuss, D. T. (2006). Frontal lobes and attention: Processes and networks, fractionation and integration. *Journal of International Neuropsychological Society*, 12, 261–271.
- Niendam, T. A., Laird, A. R., Ray, K. L., Dean, Y. M., Glahn, D. C., & Carter, C. S. (2012). Metaanalytic evidence for a superordinate cognitive control network subserving diverse executive functions. *Cognitive, Affective, & Behavioral Neuroscience,* 12, 241–268.
- Norman, D., & Shallice, T. (1986). Attention to action: Willed and automatic control of behavior. In R. Davidson, R. G. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation: Advances in research and theory* (pp. 1–18). New York: Plenum Press.
- 52. Smallwood, J., Brown, K., Baird, B., & Schooler, J. W. (2012). Cooperation between the default mode network and the frontal-parietal network in the production of an internal train of thought. *Brain Research*, 1428, 60–70.
- Schneider, W., & Shiffrin, R. M. (1977). Controlled and automatic human information processing. I. Detection, search, and attention. *Psychological Review*, 84, 1–66.
- Shallice, T. (1981). Neurologic impairment of cognitive processes. *British Medical Bulletin*, 37, 187–192.
- Cicerone, K. D., & Wood, J. C. (1987). Remediation of planning disorder after closed head injury: A case study. Archives of Physical Medicine and Rehabilitation, 68, 111–115.
- Cicerone, K. D., & Giacino, J. T. (1992). Remediation of executive function deficits after traumatic brain injury. *NeuroRehabilitation*, 2, 12–22.
- Boelen, D. H., Spikman, J. M., & Fasotti, L. (2011). Rehabilitation of executive disorders after brain injury: Are interventions effective? *Journal of Neuropsychology*, 5, 73–113.
- Sohlberg, M., Avery, J., Kennedy, M. R. T., Coelho, C., Ylvisaker, M., Turkstra, L., et al. (2003). Practice guidelines for direct attention training. *Journal of Medical Speech-Language Pathology*, 11, 19–39.
- Cicerone, K. D. (2012). Facts, theories, values: Shaping the course of neurorehabilitation. The 60th John Stanley Coulter memorial lecture. *Archives of Physical Medicine and Rehabilitation*, 93, 188–191.
- 60. Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., et al. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89, 2239–2249.
- 61. Rath, J. F., Hradil, A. L., Litke, D. R., & Diller, L. (2011). Clinical applications of problem-solving research in neuropsychological rehabilitation: Addressing the subjective experience of cognitive deficits in outpatients with acquired brain injury. *Rehabilitation Psychology*, 56, 320–328.

- Hu, N., He, S., & Xu, B. (2012). Different efficiencies of attentional orienting in different wandering minds. *Consciousness and Cognition*, 21, 139–148.
- Levine, B., Stuss, D. T., Milberg, W. P., Alexander, M. P., Schwartz, M., & Macdonald, R. (1998). The effects of focal and diffuse brain damage on strategy application: Evidence from focal lesions, traumatic brain injury and normal aging. *Journal of International Neuropsychological Society*, 4, 247–264.
- Bottari, C., Gosselin, N., Guillemette, M., Lamoureux, J., & Ptito, A. (2011). Independence in managing one's finances after traumatic brain injury. *Brain Injury*, 25, 1306–1317.
- 65. Goel, V., Grafman, J., Tajik, J., Gana, S., & Danto, D. (1997). A study of the performance of patients with frontal lobe lesions in a financial planning task. *Brain*, 120(Pt. 10), 1805–1822.
- 66. Hanks, R. A., Rapport, L. J., Millis, S. R., & Deshpande, S. A. (1999). Measures of executive functioning as predictors of functional ability and social integration in a rehabilitation sample. *Archives* of *Physical Medicine and Rehabilitation*, 80, 1030–1037.
- Ponsford, J., Draper, K., & Schonberger, M. (2008). Functional outcome 10 years after traumatic brain injury: Its relationship with demographic, injury severity, and cognitive and emotional status. *Journal* of International Neuropsychological Society, 14, 233–242.
- Ross, S. R., Millis, S. R., & Rosenthal, M. (1997). Neuropsychological prediction of psychosocial outcome after traumatic brain injury. *Applied Neuropsychology*, 4, 165–170.
- Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, 114(Pt. 2), 727–741.
- Godefrey, O., & Rousseaux, M. (1997). Novel decision making in patients with prefrontal or posterior brain damage. *Neurology*, 49, 695–701.
- 71. O'Connell, R. G., & Robertson, I. H. (2011). Plasticity of high-order cognition: A review of experience-induced remediation studies for executive deficits. In S. A. Raskin (Ed.), *Neuroplasticity* and rehabilitation. New York: The Guilford Press.
- Park, N. W., & Ingles, J. L. (2001). Effectiveness of attention rehabilitation after an acquired brain injury: A meta-analysis. *Neuropsychology*, 15, 199–210.
- Sohlberg, M. M., & Mateer, C. A. (2001). Cognitive rehabilitation: An integrative neuropsychological approach. New York: The Guilford Press.
- Couillet, J., Soury, S., Lebornec, G., Asloun, S., Joseph, P. A., Mazaux, J. M., et al. (2010). Rehabilitation of divided attention after severe traumatic brain injury: A randomised trial. *Neuropsychological Rehabilitation*, 20, 321–339.
- Kim, Y. H., Yoo, W. K., Ko, M. H., Park, C. H., Kim, S. T., & Na, D. L. (2009). Plasticity of the attentional net-

work after brain injury and cognitive rehabilitation. *Neurorehabilitation and Neural Repair*, 23, 468–477.

- Lundqvist, A., Grundstrom, K., Samuelsson, K., & Ronnberg, J. (2010). Computerized training of working memory in a group of patients suffering from acquired brain injury. *Brain Injury*, 24, 1173–1183.
- Serino, A., Ciaramelli, E., Santantonio, A. D., Malagu, S., Servadei, F., & Ladavas, E. (2007). A pilot study for rehabilitation of central executive deficits after traumatic brain injury. *Brain Injury*, 21, 11–19.
- Stablum, F., Umilta, C., Mazzoldi, M., Pastore, N., & Magon, S. (2007). Rehabilitation of endogenous task shift processes in closed head injury patients. *Neuropsychological Rehabilitation*, 17, 1–33.
- Westerberg, H., Jacobaeus, H., Hirvikoski, T., Clevberger, P., Ostensson, M. L., Bartfai, A., et al. (2007). Computerized working memory training after stroke—A pilot study. *Brain Injury*, 21, 21–29.
- 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, 656–676.
- Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attention training program. *Journal of Clinical* and Experimental Neuropsychology, 19, 117–130.
- Gordon, W. A., Cantor, J., Ashman, T., & Brown, M. (2006). Treatment of post-TBI executive dysfunction: Application of theory to clinical practice. *The Journal of Head Trauma Rehabilitation*, 21, 156–167.
- Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., et al. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 86, 1565–1574.
- 84. Sohlberg, M. M., Mateer, C. A. Attention process training APT-3 a direct attention training program for persons with acquired brain injury. Retrieved from http://www.lapublishing.com/ attention-process-training-apt3/
- Cramer, S. C., Sur, M., Dobkin, B. H., O'Brien, C., Sanger, T. D., Trojanowski, J. Q., et al. (2011). Harnessing neuroplasticity for clinical applications. *Brain*, 134, 1591–1609.
- Cicerone, K. D., Mott, T., Azulay, J., & Friel, J. C. (2004). Community integration and satisfaction with functioning after intensive cognitive rehabilitation for traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 85, 943–950.
- Sohlberg, M. M., & Turkstra, L. S. (2011). Optimizing cognitive rehabilitation: Effective instructional methods. New York: The Guilford Press.
- Sohlberg, M. M., Ehlardt, L., & Kennedy, M. (2005). Instructional techniques in cognitive rehabilitation: A preliminary report. *Seminars in Speech and Language*, 26, 268–279.
- von Cramon, D. Y., Matthes-von Cramon, G., & Mai, N. (1991). Problem solving deficits in brain

injured patients. A therapeutic approach. *Neuropsychological Rehabilitation*, *1*, 45–64.

- Fox, R. M., Martella, R. C., & Marchand-Martella, N. E. (1989). The acquisition, maintenance and generalization of problem-solving skills by closed head injured adults. *Behavior Therapy*, 20, 61–76.
- Rath, J. F., Simon, D., Langenbahn, D. M., Sherr, R. L., & Diller, L. (2003). Group treatment of problemsolving deficits in outpatients with traumatic brain injury: A randomized outcome study. *Neuropsychological Rehabilitation*, 13, 461–488.
- 92. Spikman, J. M., Boelen, D. H., Lamberts, K. F., Brouwer, W. H., & Fasotti, L. (2010). Effects of a multifaceted treatment program for executive dysfunction after acquired brain injury on indications of executive functioning in daily life. *Journal of International Neuropsychological Society*, *16*, 118–129.
- 93. Chen, A. J., Novakovic-Agopian, T., Nycum, T. J., Song, S., Turner, G. R., Hills, N. K., et al. (2011). Training of goal-directed attention regulation enhances control over neural processing for individuals with brain injury. *Brain*, 134, 1541–1554.
- 94. Levine, B., Schweizer, T. A., O'Connor, C., Turner, G., Gillingham, S., Stuss, D. T., et al. (2011). Rehabilitation of executive functioning in patients with frontal lobe brain damage with goal management training. *Frontiers in Human Neuroscience*, 5, 9.
- Levine, B., Robertson, I. H., Clare, L., Carter, G., Hong, J., Wilson, B. A., et al. (2000). Rehabilitation of executive functioning: An experimental-clinical validation of goal management training. *Journal of International Neuropsychological Society*, 6, 299–312.
- 96. Novakovic-Agopian, T., Chen, A. J., Rome, S., Abrams, G., Castelli, H., Rossi, A., et al. (2011). Rehabilitation of executive functioning with training in attention regulation applied to individually defined goals: A pilot study bridging theory, assessment, and treatment. *The Journal of Head Trauma Rehabilitation*, 26, 325–338.
- Fasotti, L., Kovacs, F., Eling, P. A., & Brouwer, W. H. (2000). Time pressure management as a compensatory strategy training after closed head injury. *Neuropsychological Rehabilitation*, 10, 47–65.
- Winkens, I., Van Heugten, C. M., Wade, D. T., & Fasotti, L. (2009). Training patients in Time Pressure Management, a cognitive strategy for mental slowness. *Clinical Rehabilitation*, 23, 79–90.
- Cicerone, K. D. (2002). Remediation of "working attention" in mild traumatic brain injury. *Brain Injury*, 16, 185–195.
- Cicerone, K. D., & Azulay, J. (2007). Perceived selfefficacy and life satisfaction after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 22, 257–266.
- 101. Ylvisaker, M., & Feeney, T. (1998). Collaborative brain injury intervention: Positive everyday routines. San Diego, CA: Singular.
- 102. Dawson, D. R., Gaya, A., Hunt, A., Levine, B., Lemsky, C., & Polatajko, H. J. (2009). Using the

cognitive orientation to occupational performance (CO-OP) with adults with executive dysfunction following traumatic brain injury. *Canadian Journal of Occupational Therapy*, 76, 115–127.

- Goverover, Y., Johnston, M. V., Toglia, J., & Deluca, J. (2007). Treatment to improve self-awareness in persons with acquired brain injury. *Brain Injury*, 21, 913–923.
- 104. Cheng, S. K., & Man, D. W. (2006). Management of impaired self-awareness in persons with traumatic brain injury. *Brain Injury*, 20, 621–628.
- 105. Schlund, M. W. (1999). Self awareness: Effects of feedback and review on verbal self reports and remembering following brain injury. *Brain Injury*, 13, 375–380.
- 106. Haskins, E. C., Cicerone, K. D., Dams-O'Connor, K., Eberle, R., Langenbahn, D., Shapiro-Rosembaum, A., et al. (2012). Cognitive rehabilitation manual: Translating evidence-based recommendations into practice. Reston, VA: American Congress of Rehabilitation Medicine.
- 107. Crosson, B., Barco, P. P., Velozo, C., Olesta, M. M., Cooper, P. V., Werts, D., et al. (1989). Awareness and compensation in postacute head injury rehabilitation. *Journal of Head Trauma Rehabilitation*, 4, 46–54.
- McPherson, K. M., Kayes, N., Weatherall, M., Members of the Goals SRRG (2009). A pilot study of self-regulation informed goal setting in people with traumatic brain injury. *Clinical Rehabilitation*, 23, 296–309.
- Toglia, J., & Kirk, U. (2000). Understanding awareness deficits following brain injury. *NeuroRehabilitation*, 15, 57–70.
- 110. Cicerone, K. D. (Ed.). (2011) Process and outcome of holistic neuropsychological rehabilitation. Holistic neuropsychological rehabilitation: Changing views, Amsterdam, Netherlands.
- 111. Keith, N., & Frese, M. (2008). Effectiveness of error management training: A meta-analysis. *Journal of Applied Psychology*, 93, 59–69.
- 112. Mount, J., Pierce, S. R., Parker, J., DiEgidio, R., Woessner, R., & Spiegel, L. (2007). Trial and error

versus errorless learning of functional skills in patients with acute stroke. *NeuroRehabilitation*, 22, 123–132.

- 113. Middleton, E. L., & Schwartz, M. F. (2012). Errorless learning in cognitive rehabilitation: A critical review. *Neuropsychological Rehabilitation*, 22, 138–168.
- 114. Wood, D., Bruner, J. S., & Ross, G. (1976). The role of tutoring in problem solving. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 17, 89–100.
- 115. Toglia, J., Johnston, M. V., Goverover, Y., & Dain, B. (2010). A multicontext approach to promoting transfer of strategy use and self regulation after brain injury: An exploratory study. *Brain Injury*, 24, 664–677.
- 116. Hewitt, J., Evans, J. J., & Dritschel, B. (2006). Theory driven rehabilitation of executive functioning: Improv ing planning skills in people with traumatic brain injury through the use of an autobiographical episodic memory cueing procedure. *Neuropsychologia*, 44, 1468–1474.
- 117. Cheyne, J. A., Solman, J. F., Carriere, J. S., & Smilek, D. (2009). Anatomy of an error: A bidirectional state model of task engagement/disengagement and attention-related errors. *Cognition*, 111, 98–113.
- 118. McAvinue, L., O'Keeffe, F., McMackin, D., & Robertson, I. H. (2005). Impaired sustained attention and error awareness in traumatic brain injury: Implications for insight. *Neuropsychological Rehabilitation*, 15, 569–587.
- 119. Manly, T., Heutink, J., Davison, B., Gaynord, B., Greenfield, E., Parr, A., et al. (2004). An electronic knot in the handkerchief: "Content free cueing" and the maintenance of attentive control. *Neuropsychological Rehabilitation*, 14, 89–116.
- 120. Gracey, F., Evans, J. J., & Malley, D. (2009). Capturing process and outcome in complex rehabilitation interventions: A "Y-shaped" model. *Neuropsychological Rehabilitation*, 19, 867–890.

# Social Communication Interventions

# Margaret A. Struchen

## Abstract

Interventions that address social communication abilities are of key importance in the rehabilitation of persons with traumatic brain injury (TBI), due to the impact of social competence on social and occupational outcomes. This chapter will review various interventions that have been utilized to address social communication difficulties after TBI. The first section of the chapter outlines the typical social communication changes observed in TBI and will clarify the scope of skills encompassed by the term social communication abilities. This will be followed by a brief review of the extant literature linking social communication to social and occupational functioning. A review of the interventions that have been used and evidence of their effectiveness is then presented, followed by a case illustration to outline clinical applications of social communication interventions for persons with TBI.

### Keywords

Social skills • Communication • Interventions • Social cognition

M.A. Struchen, Ph.D. (⊠) Department of Psychology/Neuropsychology, TIRR Memorial Hermann, Houston, TX, USA

Department of Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA e-mail: Margaret.Struchen@memorialhermann.org Interventions that address social communication abilities are of key importance in the rehabilitation of persons with TBI [1, 2]. Social communication abilities are at the core of social competence and impact functional outcomes after injury, such as social integration, employment, marital relationships, and perceived family stress [3–5]. In fact, emotional, social, and behavioral impairments, including changes in social communication functioning, are more predictive of the level of participant restriction following TBI than are cognitive and physical impairments [6]. Social support is consistently found to relate to life satisfaction [7, 8] and therefore, interventions that address social communication, which may have the potential to impact social integration outcomes and increase available social support, are significant components of rehabilitation efforts that center on community integration and enhanced quality of life after TBI.

# Social Communication Abilities and Traumatic Brain Injury

While social communication impairments impact social integration and functional outcome for persons with a variety of disabilities, there is no universally accepted definition of social skill [9]. Ylvisaker [10] stated that socially skilled people are "people that are able to affect others positively and with the effect they intend, and who are capable of being affected positively by others the way the others would like to affect them." Social communication involves sending and receiving messages to and from others. Framed as an information-processing model of social competence, this includes three skill areas: receptive abilities, processing abilities, and sending abilities [11]. Social communication abilities include a variety of general competencies, in addition to specific verbal and nonverbal skills, and must be considered in relation to specific contexts and communication partners. Context includes the physical setting, the sociocultural demands of the situation, and one's relationship to the conversational partner(s) (e.g., friend, coworker, stranger, neighbor, doctor).

While there are distinguishable theoretical differences between constructs such as social skills, pragmatics, and behavioral self-regulation, there is considerable overlap in a practical sense [12]. Receptive social communication abilities can be thought to include emotion perception and theory of mind [abilities that allow us to accurately predict the thoughts and feelings of others based on affective cues (facial expressions, tone of voice, etc.), content of speech, and knowledge of context] [13]. Processing abilities include abilities to generate alternative interpretations

of social stimuli, as well as alternative potential response to such social situations. Sending skills include the various verbal and nonverbal behaviors that are executed to send messages to others.

Cognitive and behavioral changes following TBI can impact these abilities in a number of ways. Egocentricity, concreteness of thought, impulsivity, perseveration, rigidity, poor planning, reduced initiation, slowed processing speed, reduced generativity, impaired selfmonitoring, and impaired self-regulation may be observed following TBI [1, 14]. These impairments are thought to translate into social communication deficits such as: insensitivity to others, sudden topic shifts, overtalkativeness, tangentiality, overly familiar or inappropriate comments, repetition and reliance on set expressions, impoverished speech, reduced initiation of social interaction, and literal interpretation of others' statements [9, 15].

Problems with social communication skills following TBI are thought to be consequent to both cognitive and personality changes that may result from injury to brain structures, although premorbid ability, emotional reactions to disability, and environmental factors are also likely to play significant roles in shaping social outcomes after injury [16, 17]. Focal injuries in TBI, such as contusions and hematomas, occur primarily on the orbital and lateral surfaces of the frontal and temporal lobes of the brain, which are particularly vulnerable to injury due to trauma because of their proximity to the bony protuberances of the skull [18–20]. Associations between injury to these structural areas and abilities related to social communication functioning have been found by numerous investigators. Ventral frontal lobe injury has been linked to impairments in inhibition and inefficiency in learning from consequences [21, 22]. Impaired social perception has been linked to frontolimbic structures, which are vulnerable to injury in TBI [23–25]. Diffuse axonal injuries are thought to contribute to the most common cognitive impairments experienced following TBI, namely problems with slowed processing, attention and memory functioning, and executive dysfunction [26, 27]. Slowed processing speed and

attentional difficulties are thought to affect social communication abilities by contributing to reduced comprehension of information, slowed rated of speaking, long pauses within conversation, and difficulty staying on topic in group discussion. These examples, highlighting areas of typical injury following TBI, illustrate some of the reasons why social communication impairments are commonly observed in persons with moderate to severe traumatic brain injury (TBI).

# Importance of Social Communication Abilities to Functional Outcomes

Social communication abilities have been shown to play an important role in affecting the degree to which individuals with TBI participate in social and occupational roles after injury. Social competence assists in attaining acceptance of peers and family members, aids in the development and maintenance of friendships and intimate relationships, and allows individuals to adjust to the varying social demands of school, work, and community settings.

# Social Communication Abilities and Social Integration

Social isolation is an all too common consequence following injury, at least among individuals with moderate to severe TBI. Several studies have demonstrated decreasing social network size and loss of pre-injury friendships over time, with loneliness often reported as the greatest difficulty for persons with TBI [28, 29]. Social communication functioning has been specifically associated with reduced social integration in several studies. Discourse measures (analyses of language behavior such as syntax, vocabulary, conversational skills, cohesion) have been shown to be related to social integration measures [30]. Performance on social communication measures accounted for a significant amount of variance in social integration outcomes after adjusting for executive functioning measures, age, and education in a sample of persons with chronic TBI [5]. In individuals tested more acutely after TBI, social communication variables accounted for a significant amount of variance in social integration measures, after accounting for demographic and injury-related variables [31].

# Social Communication Abilities and Employment

Return to employment following TBI is often a major goal of rehabilitation efforts and is viewed as evidence of successful outcome. Social, behavioral, and emotional factors have been demonstrated to play a major role in post-injury vocational status. Brooks and colleagues identified conversational skills as a major predictor of failure to return to work following severe TBI, in addition to personality problems, behavioral disorders, and cognitive status. [3] Stambrook and colleagues found that psychosocial and emotional sequelae were significant predictors of vocational status, in addition to age and preinjury vocational status. [32] Sale and colleagues found that the most common causes of job separation in persons with TBI were "interpersonal difficulties," "social cue misperception," and "inappropriate verbalization." [33] Persons with TBI that failed to return to work were rated by informants as displaying significantly more adverse personality changes and were rated as significantly less socially skilled by independent raters [34]. Performance on social communication measures accounted for a significant amount of variance in occupational outcomes after adjusting for executive functioning measures, age, and education in a sample of persons with chronic TBI [5].

# Social Communication Abilities and Marital Relationships

Marital satisfaction following TBI can also be largely influenced by personality, social, and behavioral changes in the person with TBI. Rosenbaum and Najenson found that compared to wives of veterans with spinal cord injuries, wives of head-injured veterans reported greater negative changes in marital and family life that were attributed to personality changes of the spouses with brain injury [35]. Liss and Willer found that interpersonal disturbances and role changes negatively impacted marital relationships for persons with TBI and their spouses [36]. The presence of behavioral problems, such as social aggression, was found to be a powerful predictor of psychological distress in spouses of persons with TBI [37]. Gosling and Oddy found that over half of the female partners of men with severe TBI reported that their partner "felt like a stranger." [38]

# Social Communication Abilities and Family Burden

Perceived burden by family members has been strongly related to social, personality, emotional, and behavioral changes in persons with TBI. Early studies by Thomsen found that personality changes tended to overshadow problems in cognitive and neurophysical functioning as determinants of family burden [39, 40]. Brooks and Aughton also found that behavioral and emotional changes in the persons with TBI outranked cognitive changes in contributing to family burden, and similar results have been found across a number of studies [41]. Kreutzer and colleagues found that family members of persons with TBI reported both elevated distress and impaired family communication functioning when compared to normal controls [42]. Less socially skilled persons with TBI showed less positive affect and required more effort from their family member to maintain the problem-solving interaction, which was interpreted to suggest that extra burden is placed on family members of individuals with social skill deficits [43].

In this brief review of the literature, it is readily apparent that social communication functioning has a significant impact on social participation and disability after TBI. Given the significance of social competence, interventions designed to improve social skill functioning are of key importance in brain injury rehabilitation.

# Interventions for Social Communication Skills Following TBI

Interventions intended to improve social skills functioning have been validated for use for several clinical conditions over the years, including schizophrenia, social anxiety, and developmental disabilities [44–46]. Despite the substantial body of research conducted since the late 1970s that has reported both that social communication abilities are commonly affected and that decreases in social integration occur after TBI, the number of empirical studies that have examined the effectiveness of social communication interventions in this population is relatively small. More recently, however, social cognition, social communication, and social communication interventions have become the foci of an increasing body of laboratory and clinical research [47, 48].

In a review of the literature conducted by Struchen, a total of 19 peer-reviewed studies were identified that evaluated the effectiveness of social communication interventions for individuals with acquired brain injury (ABI) [49]. Thirteen of these studies were either case studies or case series involving a total of 19 persons with TBI. Two additional case studies involved one individual with anoxic brain injury. Six group studies were identified involving a total of 56 persons with ABI, with three such studies involving a mixed case sample. Studies identified employed a variety of treatment approaches, however, feedback, self-monitoring, modeling, behavioral rehearsal, role-play, and social reinforcement were commonly used components. Since that review, two additional randomized clinical trials (RCTs) that directly address social communication abilities [50, 51] and one feasibility trial [52] have been published. In addition, several studies have explored alternative foci for intervention, such as centering on emotion perception [53, 54] and working with the communication partner [55, 56]. The following reviews several of the social communication interventions that have been used and presents information about their effectiveness.

# Individual Interventions for Social Communication after TBI

### **Interpersonal Process Recall**

In the first published RCT of a social communication intervention for persons with ABI, Helffenstein and Wechsler [57] compared the impact of an interpersonal process recall (IPR) treatment to a nontherapeutic attention control. IPR is a training method that was originally developed for education of counselors and focused on insights into the counselor-client and teacher-learner relationship [58–60]. This approach utilizes videotape playback of various situations to stimulate recall of the various dynamics that are involved in interpersonal communication. In a typical format, the client-counselor interaction is videotaped, and then a second counselor reviews the videotape with the client to discuss client-recalled feelings or to elaborate on the meanings of various aspects of the communication [60]. The proposed advantages of the IPR method for addressing social communication abilities for persons with TBI include: immediate and direct feedback, which is particularly important when memory difficulties are present; the opportunity for the person with TBI to provide self-feedback and receive the perspectives of others; flexibility of the approach to cover various content and communication goals; opportunities for generation of alternative behaviors, modeling, rehearsal, and role-play; individualized attention; and the ability to utilize videotape of conversational exchanges in naturalistic environments as well as clinic-based conversation samples. In addition, this individual approach can be utilized in a variety of treatment settings (e.g., rural communities, general rehabilitation service settings, etc.) where a group treatment format may be less feasible.

The IPR therapeutic approach also allows for the individualized targeting of specific social communication goals, including the flexibility to focus on all three aspects of social communication: receiving, processing, and sending skills. For example, social cue perception, can be addressed within the IPR framework through a focus on observing the listener during videotape playback, followed by overt monitoring, modeling, and role-play within the treatment session. Processing skills, such as difficulty in generating communication alternatives, can be enhanced through the mutual recall and generation of alternatives within the therapy setting, which is a key aspect of the IPR approach. Finally, the IPR approach provides great opportunities for addressing expressive social communication skills, with immediate feedback available within the treatment structure and multiple opportunities for modeling and role-play.

In Helffenstein and Wechsler's study, 16 individuals with "nonprogressive" brain injury were randomized to receive either 20 h of IPR treatment or 20 h of nontherapeutic attention [57]. Treatment for the IPR group consisted of 20 sessions involving: (1) participation in a videotaped interaction, (2) structured review of the taped interaction with feedback provided by self, conversational partner, and therapist, (3) development of alternative skills, (4) modeling, and (5) rehearsal. At post-treatment assessment, those receiving IPR treatment reported significantly reduced anxiety and improved selfconcept. More importantly, the IPR group participants were rated to have significantly greater improvement in specific interpersonal skills by both treating professional staff members and independent observer raters who had been masked to treatment condition. Additionally, communication improvements were maintained at a 1-month follow-up period for a small subset of the study sample for whom data were available. Strengths of this early study included its randomized controlled design, use of independent outcome ratings, reliance on multiple measures to assess effectiveness of the intervention, and multiple methods to assess generalization of skills to outside-of-treatment settings. However, characterization of the small study sample was limited as to definition of injury etiology and severity, a clear description of the sample selection (e.g.,

consecutive series, convenience sample), and details of treatment procedures for each condition. Despite these limitations, results of this study were encouraging, particularly given the positive results for interpersonal skill improvements noted for the treated group by masked raters in nontreatment settings.

While this is the only published study exploring the use of IPR in addressing social communication after ABI, it is noted that this type of approach, or variations of this approach, are widely utilized in clinical settings. An ongoing clinical trial designed to replicate the Helffenstein & Wechsler [57] study with a larger, more clearly defined sample using a manualized approach to treatment is currently underway and may provide additional information about the utility of the IPR approach for persons with TBI [61].

## Other Individual Interventions: Case Studies

There are a few case studies presented in the literature with fairly strong methodological rigor which show the impact of individually delivered interventions for specific social communication abilities in patients with more chronic TBI (all participants were greater than 18 months postinjury). In an early study, either feedback or selfmonitoring procedures were provided to two individuals in a group setting using otheradministered or self-administered colored light cues ("red" for negative and "green" for positive) in response to communication behaviors in a multiple baseline across treatment design, with nontreatment baseline sessions conducted prior to beginning the intervention [62]. Conversational behaviors, as rated by independent observers, improved to within the range of a comparison group of noninjured individuals for both patients during implementation of both feedback and self-monitoring conditions showing an impact from the intervention; however, there was a failure to generalize to nontreatment conditions. This may have been due to the limited number of treatment sessions provided. Unfortunately, there was lack of further follow-up to assess maintenance of treatment effects after the second set of treatment sessions.

Self-monitoring was used to learn conversational skills with female peers for two adult men with severe TBI [63]. The men were trained to count the numbers of specific target behaviors (compliments, asking others, and self-disclosure) that they performed when interacting with female peers; however, no specific instructions to increase or decrease these behaviors was provided. The study was conducted in a multiple baseline across behaviors design with each conversational behavior addressed in a different stage to training. Both participants in the program showed an increase in the number of compliments and "asking other" communication behaviors that fell within the range of communication behaviors exhibited by a noninjured comparison group. Decreases in self-disclosure were also noted for these participants, however there was greater variability in performance and self-disclosing statements were still observed with greater frequency than performed for a social comparison group. Results were maintained over a 1-month follow-up period for the participant that had such data available.

Giles and colleagues showed the impact of a focused feedback intervention where the goals of treatment were to reduce verbosity and circumstantial speech in a 27-year-old man [64]. The intervention consisted of verbal instruction regarding the rationale for behavior change with an emphasis on the phrase "short answers" to cue concise responses. Half-hour sessions were provided 5 days per week for a 1-month period, with cues for "short answers" and "permission to think before responding" given at least twice per treatment session. During each session, the following tasks were practiced: (1) responses to questions for which the patient was to respond with 1-word answers, (2) responses to questions with specific content that would require brief answers, and (3) unstructured conversation. Verbal praise was uses as an immediate social reinforcement for successfully completed tasks, with "time out on the spot" used for failure to complete tasks successfully. Significant main effects were revealed using ANOVA for singlesubject design for question type and time period (baseline, treatment, post-treatment), but no

significant interactions were observed. Use of one-word responses to structured questions showed the greatest improvement and was significantly better than attempts to provide brief responses to semi-structured questions. Interestingly, performance at follow-up was significantly better than at baseline assessment, suggesting some maintenance of gains.

In a case series of four individuals with chronic TBI, Brotherton and colleagues conducted a skills training program that was individualized to target communication behaviors identified for each participant during baseline assessment, with utilization of a multiple baseline across behaviors methodology [65]. The skills training program was conducted in 1-h sessions provided twice weekly and contained the following components: role-play, increasing understanding of the rationale for changing the target behaviors, modeling the correct behaviors, behavioral rehearsal, videotape feedback on performance, and social reinforcement of correct behaviors. Results showed that two of the four participants demonstrated clear improvements and maintenance of improvements over a 1-year follow-up for motoric communication behaviors (e.g., posture, self-manipulation), and some improvements during training for verbal behaviors, although maintenance of such improvements was limited. The other two participants had variable findings, with no evidence of improvements in performance on the target behaviors at 1-year follow-up.

## **Group Interventions**

#### **Randomized Clinical Trials**

Group interventions for treating social communication impairments following TBI are a common component of many post-acute rehabilitation programs [66]. These group interventions typically present a set of important social skills (e.g., greeting another person), and train clients to perform these skills correctly, and are likely best suited for therapy clients with similar rehabilitation goals. The inherent advantages of group interventions are that there are enhanced opportunities for feedback and observation of others' communication styles [50].

In a more recent RCT study of a group intervention for social communication functioning following TBI, Dahlberg and colleagues randomized 52 adults, ranging in age from 18 to 65 who were at least 1 year post-injury, to either a group social skills intervention or to a waitlist control [50]. The intervention included twelve 90-min group sessions offered weekly during which a structured curriculum was followed, with group size limited to eight participants per group. The intervention was designed to utilize co-group leaders from different clinical backgrounds to facilitate collaboration and varying perspectives; to emphasize self-awareness and self-assessment to enhance goal-setting; to use group process to support interactions, feedback, problem-solving and social support; and to focus on generalization of skills through involvement of a friend or family member. Initial sessions addressed selfassessment and goal-setting; intermediate sessions targeted instruction of strategies for communication goals, feedback, and practice of skills; and the latter sessions emphasized generalization of skills and problem-solving. Results of this study revealed a significantly more improved performance at 12-week follow-up evaluation on seven of ten independently rated communication rating scales for those in the treatment group as compared to the no-treatment group. In addition, self-ratings of social communication skill were significantly improved as compared to controls at this time point. However, ratings of these abilities by significant others did not show significant group differences. In addition, group differences on secondary outcomes (self- or other-report measures of social integration, productivity, satisfaction with life) were not statistically significant. Inspection of change over time for the combined treatment and deferred treatment groups revealed significant improvements above baseline performance on nine of ten independently rated communication subscales, self-reported social communication skills, and on self-, other-, and group leader-rated individualized goals as measured by goal attainment scaling (GAS) methodology. At 6-month follow-up

evaluation, maintenance of gains were demonstrated with six of ten rating scales, and selfreported social communication abilities, individualized goal performance, and satisfaction with life were significantly better than baseline performance. Strengths of this study included randomized design, multiple social communication outcome measures, independence of both communication partners and communication raters for key outcome measures, analyses using both per-protocol data and intent-to-treat models, careful delineation of the participant flow and study design, and use of multiple follow-up time points. Limitations of this work included somewhat vague criteria for study eligibility regarding level of social communication impairment required for inclusion, missing data for the primary outcome measure for a large percentage of cases (25 % missing for repeated measures analyses), and lack of a nontherapeutic attention control, which makes it unclear to what extent the treatment impacted function as compared to nonspecific treatment effects. However, given the strength of the initial findings, this approach is described as a model within the American Congress of Rehabilitation Medicine Brain Injury-Interdisciplinary Special Interest Group's (ACRM BI-ISIG) manual presenting evidencedbased cognitive rehabilitation interventions [67].

McDonald and colleagues published findings of a RCT for social communication intervention involving 39 persons with severe, chronic ABI [51]. These participants were randomized to receive a social skills training program, a social activity program (nontherapeutic attention control), or to a waitlist control condition. The social skills training program included once weekly sessions for 12 weeks, which included 2 h of manualized group component to train social behaviors (such as greetings, introducing self and other, topic selection, etc.) and 1 h of training on social perception (including emotional perception and understanding of social inferences). In addition to the 3 h of group therapies, participants each received 1 h of individualized therapy with a clinical psychologist to address personally identified issues such as self-esteem, anxiety, or depression. The social activity pro-

gram included 12 weekly 4-h sessions focused on group social activities, such as cooking, crafts, and board games. Results revealed that social activity alone did not lead to improved performance on any outcome variables as compared to waitlist controls. Participants in the skills training group were noted to have improved functioning on partner-directed behaviors, specifically with relation to self-centered behavior and partner-involvement behavior as compared to the other groups. However, no significant treatment effects were observed for social perception or emotional adjustment outcomes, nor were there effects observed for self- or otherreport measures of social functioning. The authors concluded that social skills interventions produced circumscribed improvements, particularly with direct measure of social behavior. The lack of significant improvement in social perception and emotional adjustment were not entirely unexpected and may be due to the fact that participants selected for study participation were those with social communication behavioral deficits, and may or may not have had significant difficulties with social perception or emotional adjustment. Since baseline performance for some would fall within the normal range, the ability to demonstrate improvements would have been restricted.

Future studies would benefit from employing selection of participants with specific skill difficulties (i.e., social perception impairments, poor emotional adjustment) to assess more directly the ability to improve as a result of treatments designed to impact these abilities. Despite having a fairly rigorous design, several limitations are noted for this study. Attrition rates for the study led to reduced power, which was further impacted as effect sizes were smaller than anticipated. Another issue was that authors utilized reassignment to treatment condition as some subjects were unable to attend for scheduling reasons the group to which they were initially randomly assigned. Given the small numbers, the ability to use a more conservative intent-to-treat methodology would have eliminated any potential treatment effects, so this was not done.

#### **Prospective Cohort Studies**

In addition to a more rigorous randomized clinical trial methodology, several cohort studies examining the impact of social communication interventions have been published. Johnson and Newton conducted a prospective study of a group of ten individuals that participated in a group that met for 90 min each week over a 1-year period [68]. Group sessions were divided into two parts: the first half involved the entire group meeting as a whole to consider a specific issue and the latter half would consist of smaller breakout groups to allow for more detailed individual work. Sessions consisted of a review of the previous meeting, introduction of a specific topic, discussion of the main issues, practice on specific issues, role-play, and feedback from peers and therapists. Finally, generalization was encouraged by developing social opportunities that would allow for group members to work on the selected skills in realworld social settings. Following treatment, there were no significant group changes on measures of social adjustment, social performance, social anxiety, or self-esteem. However, categorical analysis revealed that while only one participant performed within the range of a normal social comparison group at pre-treatment, six individuals performed within this range at post-treatment assessment. This study had several methodological problems, including multiple statistical tests with small sample size, limited generalization attempts, and an intervention that would likely be impractical for clinical use given its year-long involvement.

Wiseman-Hakes et al. conducted a group intervention for six adolescents with ABI, four of whom were less than 8 months post-TBI, one of whom was 8 years post-TBI, and one of whom had ABI of unspecified etiology [69]. These six participants participated in an intervention of Sohlberg and colleagues "Improving Pragmatic Skills in Persons with Head Injury" [70] modified for use with a group. Four modules were taught: initiation, topic maintenance, turn-taking, and active listening, and each module consisted of an awareness phase, a practice phase, and a generalization phase. The intervention emphasized repetition, consistency, and feedback, with peers providing feedback and cueing. Significant improvements were found for ratings of pragmatic communication skills made by independent observers in nontreatment contexts for participants following treatment, and these improvements were maintained at 6-month follow-up. Given that the majority of participants were less than 8 months post-injury and no control group was utilized for the study, it is unclear to what extent changes reflect the effects of intervention versus spontaneous recovery.

Braden and colleagues conducted a cohort study with pre-post intervention and follow-up assessments as a feasibility study to explore whether a group social communication intervention that had demonstrated effectiveness in a TBI cohort without complications would be effective for a sample of persons with TBI with co-morbid neurological or psychiatric conditions [50, 52]. Participants were 30 individuals with TBI who were at least 1 year post-injury and had identified social communication impairments. In this cohort using paired t-tests, participants showed significant improvements in self- and other-ratings of social communication behaviors, in goal attainment scale achievement, and in self-reported satisfaction with life. Gains on behavioral rating measures of social communication did not reach statistical significance, nor did measures of awareness or social participation. This study suggests the potential for social communication interventions to be used in a broader population of persons with TBI and warrants further investigation.

# Interventions Focused on Receptive Communication Abilities

Until fairly recently, receptive communication skills had not been specifically addressed as primary targets of intervention for persons with TBI. However, in the past several years, there has been significantly increased interest is social cognition and TBI, with studies exploring assessment, neuroimaging, and intervention approaches related to emotion processing and theory of mind abilities [48, 53, 54]. Since accurate perception of social interactions is necessary for the successful execution of socially skilled behavior, interventions that could improve these abilities in real-world contexts would be critical to enhancing social communication abilities.

In an initial study, Bornhofen and McDonald found significant improvements for persons with TBI in several areas of emotion perception after completing an 8-week program designed to increase accuracy of judgment for facial expression, voice tone, and body language [53]. After treatment, improvements were found in the perception of basic emotions as well as the ability to understand conversational inferences conveyed by nonverbal cues. Strategies used included errorless learning and self-instructional training.

In a follow-up study, these authors attempted to compare the effectiveness of errorless learning and self-instructional training approaches to improve emotion perception for persons with TBI [54]. Participants were 18 adults with severe TBI who were at least 5 months post-injury and performed at least two standard deviations below the norms on any emotion perception measure at baseline. The study employed random assignment to allocate participants to either an errorless learning (EL) approach, a self-instructional training (SIT) approach, or a deferred treatment control condition. Training for both active treatment conditions consisted of manualized programs of weekly 2.5-h treatment sessions over a 10-week period, for a total of 25 h of therapy. Treatment was provided in small groups of two to three participants per session. Participants completed baseline assessments of cognition, emotion perception for static images, emotion perception for dynamic images, and assessment of higher order social inference making. One week after completion of treatment, the same instruments were administered in counterbalanced order and selfreport measures of psychosocial functioning were also completed. Results of the study showed that both treatment groups had modest improvements in emotion perception abilities after treatment. There was some indication that self-instructional training methods may have been slightly favorable to errorless learning, however, with small sample sizes it is difficult to

demonstrate differences. Interestingly, the authors noted changes in relatives' report of emotion perception performance to be changed in only a few cases, despite the fact that anecdotal reports of social performance change reflected significant improvements in daily functioning. The authors suggested that available instruments may have inherent insensitivity to relatively subtle and nuanced changes evidenced in participants after this type of intervention, which may impact social interactions in a significant way. This concern regarding sensitivity of measurement for outcomes for social communication interventions is a substantial challenge in this area of clinical work and research.

Radice-Neumann and colleagues conducted a randomized trial to evaluate the effectiveness of two methods of training to address emotionprocessing deficits after ABI, facial affect recognition training and stories of emotional inference training [71]. Facial affect recognition training involved feedback and vanishing cues highlighting various facial features involved with emotional expression, in addition to education regarding physical and physiological changes associated with various emotions. Stories of emotional inference training involved short stories where participants were asked to identify emotion experienced or expressed by story characters. Participants were then queried to associate these emotional expressions with similar events from their own personal lives that led to these emotions and to focus on how such emotions were experienced. Participants were 19 individuals with ABI who were at least 1 year post-injury. All but two of the participants had traumatic brain injuries. Participants completed measures of emotional inference, perception of facial affect and prosody, and affect recognition from dynamic cues. Interventions were delivered in 1-h sessions on a thrice-weekly basis in six to nine sessions. Results of this preliminary study showed that those trained on facial affect had modest improvements in emotion recognition from faces, the ability to infer emotions from context, and in their own socioemotional behavior. Those trained with stories of emotional inference showed an increased ability to report how

they would feel in given social situations. Overall, these were modest findings, but do indicate that persons with chronic TBI can improve in these skills, at least immediately after training and in similar contexts to the training situation. It is unclear to what extent these abilities might be generalized to nontreatment settings or how well changes might be maintained. However, these preliminary efforts warrant further exploration and research.

# Interventions Focused on Communication Partners

More recently, interventions that target the communication partners of persons with TBI have been developed and evaluated with a focus on addressing the context of communication to enhance success of the social interaction. Rather than focusing exclusively on the person with TBI, these interventions in a sense attempt to modify the environment in which interactions take place. Training of communication partners has been shown to have a positive impact on communication effectiveness in other populations [72, 73]. Given some of the inherent limitations in training persons with TBI, the alternative of modifying the context in which interactions occur is consistent with a broader emphasis on participation in the social network [74]. These programs recognize that people with communication problems after TBI have fundamental social competencies that can be facilitated by the communication partner [54]. Such programs also recognize that addressing both the person with TBI and the communication partners as "clients" with potential to change resultant interactions may lead to shortened lengths of stay and providing a bridge to community living that is more sustainable. The following studies either directly or indirectly attempt to shape communication interaction through interventions aimed at the communication partners of persons with TBI.

Togher and colleagues attempted one of the first studies to train communication partners as the focus of treatment for communication effectiveness in a program designed to improve the interactions of persons with TBI with police officers during service encounters [55]. In this study, 20 police officers were randomly assigned to receive either a 6-week 2-h per week training program focused on communication strategies training or standard baton and weapons training. Specific targets for communication training were based on the officers' performance during phone interactions with persons with TBI conducted at baseline. These targets included: difficulty establishing the nature of the service request, failing to stop participants with TBI from straying on to tangential topics, and difficulty closing the call. Training involved increased awareness of the officers with regard to these problems. Officers were trained to systematically consider the way they make language choices during service encounter interactions and included both didactic and role-play elements. Each session involved viewing of a videotape of a person with TBI. Officers focused on how they might interact in each case, generated specific communication strategies, and eventually engaged in practice conversations with persons with TBI. Results of this trial showed that trained police officers were able to learn communication strategies and posttraining calls were rated as more efficient and focused. Furthermore, persons with TBI engaging with trained officers were noted to improve their communication as well, with reduced episodes of unrelated speech and an increased proportion of the interaction focusing on completing the service encounter, which appeared to be related to communication options given by officers. This initial trial shows the potential benefit of focused training on communication partners to improve social communication for persons with TBI, as well as improving the key goal of increased social participation.

A more recent study by Behn et al. explored the effectiveness of a communication training program for paid caregivers of persons with TBI, with the goal of training caregivers to support increasing social interaction effectiveness for persons with TBI [56]. In this study, ten paid caregivers in a residential post-acute brain injury rehabilitation program were randomly assigned to receive either a 17-h 8-week training program or no training. The training program consisted of an adaptation of a program for communication partners of people with TBI and focused on positive communication through collaboration and elaboration of conversational strategies [10]. Results of this trial revealed that conversations of persons with TBI and their caregivers were rated as more appropriate, interesting, and rewarding for those in the treatment group as compared with controls. Additionally, trained caregivers showed an increase in their acknowledgement and awareness of the competencies of persons with TBI. It was noted, however, that improvements were restricted to structured conversations. However, at 6-month follow-up improvements were maintained for those in the treatment group. It is noted that this study was conducted in a residential long-term care facility, and it would be interesting to apply these methods to caregivers in the larger community to explore effectiveness of these strategies in home and community settings.

A number of other studies exploring interventions with communication partners of persons with TBI are ongoing and this avenue of research and clinical intervention holds considerable promise, as either a stand-alone or adjunct service to more typical social communication interventions. There have been studies examining training co-workers to assist with return-to-work for persons with TBI [75] and use of peer mentors to enhance social participation [76]. It would seem possible that such interventions, which involve situational coaching or changing the context of social interactions would be useful strategies to pursue and are consistent with current emphasis on context as a determinant of health and disability outcomes [74].

# **Case Illustration**

The following brief case illustration highlights some of the typical social communication difficulties that occur after TBI, along with description of assessment results and implementation of specific interventions.

Case N.W.: A 24-year-old man was referred for rehabilitation services after sustaining a severe TBI, which had occurred 18 months previously, with a goal of returning to work. Mr. N.W. had a history of right frontal contusion as a result of his TBI, and was observed to provide lengthy responses to questions, to dominate the conversation, to have difficulty staying on-topic in conversation, and to introduce topics that were overly personal and inappropriate to the context. In addition, N.W. tended to stand too close to others when speaking and held eye contact for periods that exceeded typical social custom. In a situational work assessment, these behaviors were described by other volunteers as contributing to some discomfort in interacting with N.W.

Results of neuropsychological testing revealed moderate impairments in attention and executive functioning, with mild to moderate impairments noted for verbal learning and memory. His performance was notable for difficulties with sustained attention, set-shifting, and multiple intrusion errors. Other than problems on verbal fluency tasks, N.W.'s performance on measures of language functioning were within normal limits. Additional measures focusing specifically on social communication, including self- and otherreport rating scales of social communication revealed self-reported difficulties with conversational flow and partner sensitivity problems, although family report of the same behaviors revealed significantly more difficulty and greater severity of impact of various communication behaviors. This suggests that impaired selfawareness likely impacted performance as well and would need to be addressed in interventions. Similar findings with regard to communication were identified using behavioral rating scales of social communication behavior utilized to rate videotaped semi-structured interactions ("getting to know you" conversation with therapist in clinic, videotape of performance on a work trial situational assessment [volunteering at a food bank with therapist's supervision]).

Goals for social communication intervention were identified based on these clinical data, on N.W.'s stated goals, and on input from N.W., his family members, and the treatment team. Given the importance of his overall goal of returning to competitive employment, a decision was made to focus initially on communication behaviors that were interfering most with his ability to function in the workplace setting. Therefore, the goals of intervention included: (1) Improving nonverbal aspects of communication through decreasing eye contact and increasing awareness of physical distance with communication partners, (2) Developing a repertoire of conversational topics that would be appropriate given the context of a work environment, and (3) Increasing focus on concise responses.

An IPR approach to treatment was used with initial sessions focused primarily on the goal of improving N.W.'s nonverbal communication skills. Interactions in naturalistic environments (talking with a supervisor at a volunteer site, interacting with fellow volunteers during a work break) were videotaped for each treatment session and then the videotape was immediately processed by the client, the job coach, and the therapist. The initial session focused on increasing awareness of both problem behaviors and responses related to these behaviors received from communication partners. In viewing the initial videotape, N.W. was able to observe that he was standing pretty close to the job coach, and noted that she was stepping backwards and seemed uncomfortable. The job coach shared with the client that his close proximity along with extended eye contact made her feel uncomfortable, and that she perceived this behavior as somewhat aggressive. N.W. was surprised at this response and stated that he did not realize he was staring at her. The therapist then asked the client to recall if he had received any feedback before about these behaviors. N.W. reported that he had had some negative reactions from women in the community, but was not really aware of the reasons for such reaction. The therapist and job coach modeled nonverbal behaviors that would likely contribute to greater communication success in the workplace setting and those that would likely be problematic focusing specifically on personal distance and eye contact. The therapist then encouraged N.W. to generate strategies that might address issues related to eye contact and personal space. N.W. suggested imagining that he should try to stand at least one arm-length away from the person to whom he was speaking. This was then the subject of role-play, in which the client was asked to exit the room and then enter to have a conversation with the job coach, focusing on maintaining an arms-length distance from her during interactions. The job coach engaged pleasantly with N.W. when he was at a comfortable distance, but was asked to make an exaggerated step backwards if her personal space was encroached upon. With regard to eye contact, it was suggested that N.W. try to look slightly away from his communication partner after every few seconds. Initially, he mentally counted slowly to five before looking away and then returning gaze. This was also rehearsed during clinical sessions. The job coach was to indicate by touching the corner of her eye if eye contact was maintained at an uncomfortable duration. N.W. was given homework after his initial session to attend to social distances with focus on his arm-length spacing with communication partners, as well as practice reducing staring through practice with regular gaze shifts at fivecount intervals. This homework was discussed with N.W. and with his family, so that support could be provided for practice in community settings. N.W. responded very well to this intervention and issues with proximity to communication partners improved dramatically after the initial session. Issues with prolonged eye contact improved gradually, and after several sessions, were rated as occurring infrequently in the work setting (once per 3-h shift, compared with an average of 5–6 times per shift at outset).

The other primary communication goals were addressed in a similar fashion with initial focus on increasing awareness of communication issues, discussion of communication behaviors that would likely lead to more success in workplace settings, generation of strategies to address the specific communication goal, modeling of skills, role-play and rehearsal, and then practicing of skills via homework assignments. Role-play and homework practice emphasized performance of behaviors in appropriate context (e.g., workplace setting) to facilitate transfer of skills to community setting. Although N.W. continued to display some problematic communication behaviors at the conclusion of the interventions (e.g., occasional staring, occasional lengthy discourse), his communication behaviors improved to the point that he was able to successfully complete a job trial with good overall feedback from his supervisor, his job coach, and co-workers. This ultimately helped facilitate his ability to attain a competitive employment position.

## **Summary and Future Directions**

Results of the studies that have examined effectiveness of interventions to address social communication difficulties after TBI suggest that there are significant impacts of these efforts, although the evidence base is limited to date. A variety of interventions and intervention approaches have shown effects on social communication performance, with some evidence indicating generalization of skills to nontreatment settings (maintenance of gain at follow-up, improved ratings of performance in nontreatment situations). However, there is much to learn in this area of clinical intervention and research.

Increased understanding of specific social communicative skills encompassing receptive, processing, and expressive abilities would provide an important foundation for the selection and matching of intervention to clinical issue. Improving our conceptualization of how underlying cognitive and behavioral constructs impact social communication abilities would also assist in developing and targeting intervention skills. Awareness of the influence that environmental factors, including skills of the communication partner, may have on social interactive competence for persons with TBI is a relatively understudied area within the field and may yield insights that would be fruitful for intervention.

As has been described in previous reviews of the social communication intervention literature in TBI, research in this area is in its early stages [11, 49]. This contrasts with the large body of literature exploring application of social skills interventions in other disability populations. There are several reasons that likely explain why a limited number of studies that have addressed this important area of neuropsychological intervention have been conducted. Studies to evaluate social communication interventions after TBI are inherently very challenging to conduct. TBI is known to involve significant heterogeneity with regard to injury severity, localization of injury, and other factors, which results in variable impacts on social communication and interpersonal skill functioning. The heterogeneity of preinjury social skill performance amongst persons with TBI also introduces great variability and makes evaluation of injury-related change difficult to assess and likelihood of response to treatment highly variable. Further work to identify specific profiles or subtypes of communication impairment would lend itself to development and implementation of more targeted interventions, which may have greater impact in modifying communication behaviors.

In order to address some of these gaps in our current understanding, an increased focus on improving our assessment approaches through validation of measurement tools to assess aspects of social communication, as well as outlining recommended approaches to the comprehensive assessment of abilities relevant to social communication competence would be very valuable. Measurement instruments and approaches that evaluate receptive, processing, and expressive aspects of social communication competence are needed, and consideration of these various aspects of communication skill should be evaluated prior to the beginning of treatment ideally. Assessment approaches might include: informal behavioral observation, use of more formal behavioral rating scales applied to communication samples, self- and other-report instruments, specific behavioral measures (facial affect recognition, tests of social inference, social problemsolving), and general neurocognitive assessment. One approach that is likely to be highly useful in measuring effectiveness of social communication interventions would be the inclusion of GAS methodologies [77]. While traditional standardized measures include a standard set of items rated on a standard level. GAS involves individually identified tasks to address specific patient goals, with levels being individually set around their current and expected levels of performance. This method consists of a priori establishment of criteria for a successful outcome, which is agreed upon by the person with TBI and the family, such that all have a realistic expectation of what is likely to be achieved and agree that the specific goals are of value. Each goal is rated on a 5-point scale, with the degree of attainment captured for each goal area, which allows for evaluation of change in target behaviors after delivery of an intervention. This approach has already been used in several social communication intervention trials to date [50, 52]. The advantages of this approach are that it avoids some of the issues inherent with standardized measures, including problems with floor and ceiling effects and lack of sensitivity to change, whereby significant changes in one or more items on a larger scale are lost in the overall summary scores [77]. While these are advantages of the GAS approach, this method does not allow for normative comparisons. Likely a combination of measurement methods would yield the best combination of sensitivity and generalizability of results.

Greater specification of the social communication issues that are the focus of intervention and improved measurement of outcomes would assist in advancing the field. Additionally, consideration of the focus of the interventions themselves is an area for continued growth and development. While typical social skills interventions are based on the notion that individuals requiring such training suffer from a lack of knowledge about social rules and customs, want to change their behavior, and can adapt skills learned in the training environment to community settings, the applicability of these assumptions to many persons with TBI is not clear. Persons with TBI are likely to have had adequate knowledge of social norms and routines, with impairments in social skills functioning reflecting a more acute change of ability consequent with injury to the

brain. Therefore, training focused on teaching social customs and norms, which is often a component of typical social skills training approaches with other populations, may be less pertinent. For some persons with TBI, where injury has affected frontal systems and involves impaired self-awareness, there may be a lack of motivation to change social communication behaviors. Finally cognitive impairments, such as impaired learning and self-regulatory abilities may interfere with the ability to transfer or apply skills to the individual's real-world environment. Consideration of these factors suggests that while specific aspects of communication skills training may be appropriate, the targets of interventions should be matched to the areas of difficulty. Furthermore, contextualized training must always be a major aspect of training to facilitate transfer of trained skills to community functioning.

Ylvisaker and colleagues outlined several clinical implications based on the available research evidence in the field of social communication interventions in TBI and the extensive body of social skills research conducted with other populations [12]. These authors identified interventions and supports as clinically supported by the current research, which are summarized here:

- Using a person-centered approach to goalsetting, such that the focus is on social success and achievement of personal aims, rather than a more punitive view where an authority figure outlines behavior that is deemed "socially appropriate" or "social inappropriate."
- Enhancing focus on educating everyday communication partners in specific social competence issues so that they can interact supportively and will not misinterpret social behavior resulting from the brain injuryrelated difficulties of individuals with TBI, such as poor initiation, impulsivity, and misreading of social cues.
- Providing extensive practice in performance of specific social communication behaviors as they relate to natural contexts, with experience of natural and logical consequences of successful performance. Use of videotaped roleplay, feedback, and self-modeling provided as

supplemental training experiences that can facilitate such practice.

- Using context-specific training for highly specific social communication behaviors that are highly valued by the person with TBI.
- Providing situational coaching with use of cueing to prevent problem social behaviors from occurring.
- Providing situational training to improve social perception by direction to nonverbal cues and context to facilitate interpretation of others' behaviors.
- Providing situational training that is targeted at improving self-monitoring of stress levels, such that individuals can either remove themselves from stressful situation or takes steps to reduce their stress levels to facilitate socially competent behaviors.
- Supplementing social skills interventions with counseling to help individuals develop a satisfying sense of self, which includes positive social interactive competence as a component.

These recommendations underscore the vital importance of context in addressing social communication behavior. In these recommendations, context is addressed through the type of behavioral targets recommended, to the settings in which interventions are provided, and to the focus of the treatment itself, which is inclusive of communication partners as well as persons with TBI. In addition, these recommendations allude to intervening not only with social skill behaviors, but also with a focus on social perceptive abilities.

To these suggestions, there are several recommendations that should be added:

- Improving specification of target social communication behaviors through comprehensive assessment of social and cognitive abilities would allow for a more targeted intervention approach.
- Identifying outcomes assessment instruments and strategies (such as GAS methodologies, structured behavioral ratings) that are most sensitive to social communication behavior changes would enhance the evidence base.
- · Increasing understanding of which individuals

benefit best from specific interventions through increased knowledge about how individual and environmental characteristics contribute to social communication behaviors after TBI would be useful in developing targeted interventions.

- Including both specific social-skills training that is contextually applied *and* direct attempts at intervening with the individual's environment (e.g., training communication partners, structuring work setting to facilitate successful social interactions) as part of the model of care.
- Studies that evaluate the effectiveness of interventions that target social communicative abilities should measure trained skills at posttreatment intervals, but should also include generalization probes, including assessment of maintenance of gains and social skills performance in community settings.

The current evidence base for interventions focused on social communication after TBI supports use of such treatments in clinical practice for this population. However, there continues to be a strong need to increase our understanding of social communication abilities and how best to link specific interventions to specific communication profiles. Although inquiry into social communication interventions after TBI is still in its infancy, there has been considerable growth in research in this area, including clinical trials of social skills training, investigations of social cognition, investigations of the impact of environmental factors on social interactive skill, and exploring interventions aimed at communication partners to facilitate communicative competence. Given the critical importance of social competence to successful community participation for persons with TBI, it is encouraging that this area of clinical practice is gaining attention. It is hoped that this increased focus on broad aspects of research related to social communication abilities will lead to clearer practice guidelines in the future.

Acknowledgement Preparation of this chapter was partially supported by the U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020.

## References

- Hartley, L. L. (1995). Cognitive-communicative abilities following brain injury. San Diego, CA: Singular Publishing Group.
- Marsh, N. V. (1999). Social skill deficits following traumatic brain injury: Assessment and treatment. In S. McDonald, L. Togher, & C. Code (Eds.), *Communication disorders following traumatic brain injury* (pp. 175–210). East Sussex, England: Psychology Press Ltd.
- Brooks, D. N., McKinlay, W., Symington, C., Beattie, A., & Campsie, L. (1987). Return to work within the first seven years of severe head injury. *Brain Injury*, 1, 5–19.
- Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: A literature review and recommendations. *Brain Injury*, 9(1), 81–92.
- 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(2), 185–198.
- Mooney, G. F. (1988). Relative contributions of neurophysical, cognitive, and personality changes to disability after brain injury. *Cognitive Rehabilitation*, 6(5), 14–20.
- Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of Personality Assessment*, 49, 71–75.
- Fuhrer, M., Rintala, D. H., Hart, K. A., Clearman, R., & Young, M. E. (1992). Relationship of life satisfaction to impairment, disability, and handicap among persons with spinal cord injury living in the community. *Archives of Physical Medicine and Rehabilitation*, 73(6), 52–557.
- McDonald, S. (1992). Communication disorders following closed head injury: New approaches to assessment and treatment. *Brain Injury*, 6, 283–292.
- Ylvisaker, M. (1998). Traumatic brain injury rehabilitation: Children and adolescents. Boston: Butterworth-Heinemann.
- McFall, R. M. (1982). A review and reformulation of the concept of social skills. *Behavioral Assessment*, 4, 1–33.
- 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.
- Martin-Rodriguez, J. F., & Leon-Carrion, J. (2010). Theory of mind deficits in patients with acquire brain injury: A quantitative review. *Neuropsychologia*, 48(5), 1181–1191.
- Prigatano, G. P. (1999). Principles of neuropsychological rehabilitation. New York: Oxford University Press.

- Flanagan, S., McDonald, S., & Togher, L. (1995). Evaluating social skills following traumatic brain injury: The BRISS as a clinical tool. *Brain Injury*, 9(4), 321–338.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., Starkstein, S. E., Forrester, A. W., & Geisler, F. (1993). Depression following traumatic brain injury: A 1 year longitudinal study. *Journal of Affective Disorders*, 27, 233–243.
- Gomez-Hernandez, R., Max, J. E., Kosier, T., Paradiso, S., & Robinson, R. G. (1997). Social impairment and depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 78, 1321–1326.
- Holbourn, A. H. S. (1943). Mechanics of head injuries. *Lancet*, 1, 438–441.
- Ommaya, A. K., & Corrao, P. (1971). Pathologic biomechanics of central nervous system injury in head impact and whiplash injury. In K. M. Bringhaus (Ed.), *Accident pathology* (pp. 16–181). Washington, DC: U.S. Government Printing Office.
- Adams, J. H., Graham, D. I., Scott, G., Parker, L. S., & Doyle, D. (1980). Brain damage in fatal nonmissile head injury. *Journal of Clinical Pathology*, 33, 1132–1145.
- Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex*, 10, 284–294.
- Schlund, M. W. (2002). Effects of acquired brain injury on adaptive choice and the role of reduced sensitivity to contingencies. *Brain Injury*, 16, 527–535.
- Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, 2, 1032–1037.
- Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to response autonomically to social stimuli. *Behavioural Brain Research*, *41*, 81–94.
- Tranel, D., Bechara, A., & Denburg, N. L. (2002). Asymmetric functional roles of right and left ventromedial prefrontal cortices in social conduct, decisionmaking, and emotional processing. *Cortex, 38*, 589–612.
- Ommaya, A. K., & Gennarelli, T. A. (1974). Cerebral concussion and traumatic unconsciousness: Correlation of experimental and clinical observations on blunt head injuries. *Brain*, 97, 633–654.
- Meyers, C. A., Levin, H. S., Eisenberg, H. M., & Guinto, F. C. (1983). Early versus late lateral ventricle enlargement following closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 46, 1092–1097.
- Kozloff, R. (1987). Networks of social support and the outcome from severe head injury. *The Journal of Head Trauma Rehabilitation*, 2, 14–23.
- Temkin, N. R., Corrigan, J. D., Dikmen, S. S., & Machamer, J. (2009). Social functioning after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 24(6), 460–467.

- Snow, P., Douglas, J., & Ponsford, J. (1998). Conversational discourse abilities following severe traumatic brain injury: A follow-up study. *Brain Injury*, 12(11), 911–935.
- 31. Struchen, M. A., Pappadis, M. R., Sander, A. M., Burrows, C. S., & Myszka, K. A. (2011). Examining the contribution of social communication abilities and affective/behavioral functioning to social integration outcomes for adults with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 26(1), 40–42.
- 32. Stambrook, M., Moore, A. D., Peters, L. C., Devianene, C., & Hawryluk, G. A. (1990). Effects of mild, moderate and severe closed head injury on longterm vocational status. *Brain Injury*, 4(2), 183–190.
- 33. Sale, P., West, M., Sherron, P., & Wehman, P. (1991). Exploratory analysis of job separations from supported employment for persons with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 6(3), 1–11.
- 34. Godfrey, H. P. D., Partridge, F. M., Knight, R. G., & Bishara, S. (1993). Course of insight disorder and emotional dysfunction following closed head injury. *Journal of Clinical and Experimental Neuropsychology*, 15, 503–515.
- 35. Rosenbaum, M., & Najenson, T. (1976). Changes in life patterns and symptoms of low mood as reported by wives of severely brain-injured soldiers. *Journal of Consulting and Clinical Psychology*, 44, 881–888.
- Liss, M., & Willer, B. (1990). Traumatic brain injury and marital relationships: A literature review. *International Journal of Rehabilitation Research*, 12(4), 309–320.
- Linn, R. T., Allen, K., & Willer, B. S. (1994). Affective symptoms in the chronic stage of traumatic brain injury: A study of married couples. *Brain Injury*, 8(2), 135–147.
- Gosling, J., & Oddy, M. (1999). Rearranged marriages: Marital relationships after head injury. *Brain Injury*, 13(10), 785–796.
- Thomsen, I. V. (1974). The patient with severe head injury and his family. *Scandinavian Journal of Rehabilitation Medicine*, 6, 180–183.
- Thomsen, I. V. (1984). Late outcome of very severe blunt head trauma: A 10-15 year second follow-up. *Journal of Neurology, Neurosurgery, and Psychiatry*, 4, 260–268.
- Brooks, D. N., & Aughton, M. E. (1979). Psychological consequences of blunt head trauma. *International Rehabilitation Medicine*, 1, 160–165.
- Kreutzer, J. S., Gervasio, A. H., & Camplair, P. S. (1994). Patient correlates of caregivers' distress and family functioning after traumatic brain injury. *Brain Injury*, 8(3), 211–230.
- 43. Godfrey, H. P. D., Knight, R. G., & Bishara, S. N. (1991). The relationship between social skill and family problem-solving following very severe closed head injury. *Brain Injury*, 5(2), 207–211.
- 44. Liberman, R. P., Eckman, T. A., & Marder, S. R. (2001). Training in social problem solving among

persons with schizophrenia. *Psychiatric Services*, 52, 31–33.

- 45. Hambrick, J. P., Weeks, J. W., Harb, G. C., & Heimberg, R. G. (2003). Cognitive-behavioral therapy for social anxiety disorder: Supporting evidence and future directions. *CNS Spectrums*, 8, 373–381.
- 46. Kennedy, C. H. (2001). Social interaction interventions for youth with severe disabilities should emphasize independence. *Mental Retardation and Developmental Disabilities Research Reviews*, 7, 122–127.
- McDonald, S., & Flanagan, S. (2004). Social perception deficits after traumatic brain injury: Interaction between emotion recognition, mentalizing ability, and social communication. *Neuropsychology*, 18, 572–579.
- Milders, M., Ieswaart, M., Crawford, J. R., & Currie, D. (2006). Impairments in theory of mind shortly after traumatic brain injury and at 1-year follow-up. *Neuropsychology*, 20, 400–408.
- Struchen, M. A. (2005). Social communication interventions for persons with traumatic brain injury. In W. M. High Jr., A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation of traumatic brain injury* (pp. 88–117). New York: Oxford University Press.
- 50. Dahlberg, C. A., Cusick, C. P., Hawley, L. A., Newman, J. A., Morey, C. E., Harrison-Felix, C. L., et al. (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, 1561–1573.
- 51. McDonald, S., Tate, R., Togher, L., Bornhofen, C., Long, E., Gertler, P., et al. (2008). Social skills treatment for people with severe, chronic acquired brain injuries: A multicenter trial. *Archives of Physical Medicine and Rehabilitation*, 89, 1648–1659.
- 52. Braden, C., Hawley, L., Newman, J., Morey, C., Gerber, D., & Harrison-Felix, C. (2010). Social communication skills group treatment: A feasibility study for persons with traumatic brain injury and comorbid conditions. *Brain Injury*, 24(11), 1298–1310.
- Bornhofen, C., & McDonald, S. (2008). Treating deficits in emotion processing following traumatic brain injury. *Neuropsychological Rehabilitation*, 18(1), 22–44.
- Bornhofen, C., & McDonald, S. (2008). Comparing strategies for treating emotion perception deficits in traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 23(2), 103–115.
- Togher, L., McDonald, S., Code, C., & Grant, S. (2004). Training communication partners of people with traumatic brain injury: A randomized controlled trial. *Aphasiology*, *18*(4), 313–335.
- Behn, N., Togher, L., Power, E., & Heard, R. (2012). Evaluating communication training for paid carers of people with traumatic brain injury. *Brain Injury*, 26(13–14), 1702–1715.
- Helffenstein, D. A., & Wechsler, F. S. (1982). The use of interpersonal process recall (IPR) in the remediation of interpersonal and communication skill deficits

in the newly brain-injured. *Clinical Neuropsychology*, 4(3), 139–143.

- Kagan, N. (1975). Influencing human interaction: Eleven years with IPR. *Canadian Counselor*, 9, 74–97.
- Kagan, N., Krathwohl, D. R., & Miller, R. (1963). Stimulated recall in therapy using videotape: A case study. *Journal of Counseling Psychology*, 10, 237–243.
- Kagan, N. (1969). Interpersonal process recall. Journal of Nervous and Mental Disease, 148(4), 365–374.
- 61. Struchen, M. A., Clark, A. N., Davis, L. C., Mazzei, D. M., Bogaards, J. A., Sander, A. M., et al. Use of Interpersonal Process Recall training to address social communication abilities after traumatic brain injury (unpublished manuscript).
- Gajar, A., Schloss, P. J., Schloss, C. N., & Thompson, C. K. (1984). Effects of feedback and self-monitoring on head trauma youths' conversation skills. *Journal of Applied Behavior Analysis*, 17, 353–358.
- 63. Schloss, P. J., Thompson, C. K., Gajar, A. H., & Schloss, C. K. (1985). Influence of self-monitoring on heterosexual conversational behaviors of head trauma youth. *Applied Research in Mental Retardation*, 6, 269–282.
- 64. Giles, G. M., Fussey, I., & Burgess, P. (1988). The behavioral treatment of verbal interaction skills following severe head injury: A single case study. *Brain Injury*, 2, 75–79.
- 65. Brotherton, F. A., Thomas, L. L., Wisotzek, I. E., & Milan, M. A. (1988). Social skills training in the rehabilitation of patients with traumatic closed head injury. *Archives of Physical Medicine and Rehabilitation*, 69, 827–832.
- 66. Boake, C. (1991). Social skills training following head injury. In J. S. Kreutzer & P. H. Wehman (Eds.), *Cognitive rehabilitation for persons with traumatic brain injury*. Baltimore: Paul H. Brooks.
- 67. Haskins, E. C., Cicerone, K., Dams-O'Connor, K., Eberle, R., Langenbahn, D., Shapiro-Rosenbaum, A., et al. (2012). *Cognitive rehabilitation manual: Translating evidence-based recommendations into practice*. Reston, VA: American Congress of Rehabilitation Medicine.

- Johnson, D. A., & Newton, A. (1987). Social adjustment and interaction after severe head injury: II. Rationale and basis for intervention. *British Journal of Clinical Psychology*, 26, 289–298.
- Wiseman-Hakes, C., Stewart, M. L., Wasserman, R., & Schuller, R. (1998). Peer group training of pragmatic skills in adolescents with traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 13*, 23–38.
- Sohlberg, M. M., Perlewitz, P. G., Johansen, A., Schultz, J., Johnson, L., & Hartry, A. (1992). *Improving pragmatic skills in persons with head injury*. Tucson, AZ: Communication Skill Builders.
- Radice-Neumann, D., Zupan, B., Tomita, M., & Willer, B. (2009). Training emotional processing in persons with brain injury. *The Journal of Head Trauma Rehabilitation*, 24(5), 313–323.
- 72. Kagan, A., Black, S., Duchan, J., Mackie, N., & Square, P. (2001). Training volunteers as conversation partners using "Supported Conversation for Adults with Aphasia": A controlled trial. *Journal of Speech, Language, and Hearing Research,* 44, 624–638.
- Ripich, D., Ziol, E., Fritsch, T., & Durand, E. (1999). Training Alzheimer's disease caregivers for successful communication. *Clinical Gerontologist*, 21, 37–53.
- 74. World Health Organization. (2001). International classification of functioning, disability, and health. Report. Geneva, Switzerland: World Health Organization.
- 75. O'Reilly, M. F., Lacnioni, 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.
- 76. Struchen, M. A., Davis, L. C., Bogaards, J. A., Hudler-Hull, T., Clark, A. N., Mazzei, D. M., et al. (2011). Making connections after brain injury: Development and evaluation of a social peermentoring program for persons with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 26(1), 4–19.
- Turner-Stokes, L. (2009). Goal attainment scaling (GAS): A practical guide. *Clinical Rehabilitation*, 23(4), 362–370.

# **Impaired Self-Awareness**

# Mark Sherer and Jennifer Fleming

## Abstract

Persons with neurologic disorders often show impaired ability to accurately perceive the effects of their disorders on their physical, cognitive, and behavioral abilities. For conditions that have a sudden onset such as stroke or traumatic brain injury (TBI) the degree of impairment of this ability is greatest early after onset so that, for example, soon after right hemisphere stroke, the person with stroke may not perceive motor impairment on the left side of the body, but gradually becomes more aware of this impairment as he/she recovers. For progressive conditions such as the various dementias, the degree of impairment of self-awareness worsens as the condition progresses so that a person with Alzheimer's disease may be aware of subtle memory impairment in early stages of the disease, but unaware of very severe memory impairment once the disease has progressed. In either case, the perception of impairment is least when the actual impairment is greatest and greatest when the actual impairment is least.

Impaired self-appraisal of functioning is referred to as anosognosia in conditions such as the neglect syndrome after right hemisphere stroke or Anton's syndrome (unawareness of cortical blindness) after bilateral posterior cerebral artery strokes where the lack of awareness may be complete. In TBI, this condition is simply called impaired self-awareness (ISA) as persons with injury usually have some awareness of their deficits once they recover from the confused state (post-traumatic amnesia).

J. Fleming, Ph.D.

M. Sherer, Ph.D., ABPP, FACRM (⊠) TIRR Memorial Hermann, Houston, TX, USA

University of Texas Medical School at Houston, Baylor College of Medicine, Houston, TX, USA e-mail: Mark.Sherer@memorialhermann.org

University of Queensland School of Health and Rehabilitation Sciences and Princess Alexandra Hospital, Brisbane, QLD, Australia

For persons with TBI, severity of ISA is greatest in early recovery. By definition, patients in coma or the vegetative state have no self-awareness. Though not directly assessed, minimally conscious patients are assumed to have extremely limited self-awareness. Once patients recover to the confused state, they remain with very severe impairment of self-awareness. Often after resolution of confusion, patients may deny that they have sustained TBI at all and deny any residual effects of the injury. Even when acknowledging injury, we have seen patients insist that their cognitive abilities after severe TBI are much better than they were prior to sustaining TBI. Patients who are unable to walk safely may attempt to remove restraints to leave their beds or wheelchairs to go to the toilet or simply to attempt to leave the room. Patients may refuse therapies because they do not believe that they have impairments that need to be treated. In the post-acute period of recovery, patients may attempt to drive in spite of motor and sensory deficits or pursue employment or independence goals that are at odds with their current functional limitations. To ensure patient safety, compliance with needed therapies, and the best possible outcomes for patients, neuropsychologists and others treating persons with TBI must assess and, when needed, provide treatment for deficits in self-awareness.

This chapter will: (1) review the nature of ISA after TBI and describe its impact on rehabilitation therapies and patient outcome, (2) describe approaches to assessment of ISA, (3) review the literature on interventions to improve self-awareness in persons with TBI, and (4) provide practical guidance illustrated with clinical cases for intervening with patients with ISA.

Keywords

Self-awareness • Metacognition • Traumatic brain injury • Assessment • Rehabilitation • Intervention • Psychology • Adjustment

## Impaired Self-Awareness After Traumatic Brain Injury

### Incidence and Course of Recovery

All persons with moderate or severe traumatic brain injury (TBI) have impaired self-awareness (ISA) early after injury while confused. The confused state which is characterized by disorientation, cognitive impairment, restlessness, fluctuation in cognitive and other neurobehavioral functions, and other deficits [1] is not compatible with the ability to form accurate self-perceptions. Even after resolution of confusion, a number of studies have shown that patients in early recovery from TBI rate themselves as having less impairment than do treating clinicians or family members/significant others indicating some degree of ISA [2–4]. In a mixed sample of patients with TBI and aneurysm rupture, 97 % rated themselves as less impaired than they were rated by treating clinicians [5]. Remarkably, over 25 % of persons in early recovery from moderate and severe TBI actually rate themselves as having better cognitive and other neurobehavioral skills than they did prior to their injuries, clearly indicating substantial ISA [4].

Severity of ISA varies by the area assessed. Studies consistently show greater ISA for cognitive and behavioral functioning as compared to physical functioning [3, 6]. Overall degree of ISA is associated with injury severity so that persons with more severe injuries have greater ISA [4].

As for other neurobehavioral deficits, ISA shows a recovering course [2, 7]. In a study of 123 persons with moderate and severe TBI who were assessed an average of 45 days post-injury and again at 1 year post-injury, Hart and colleagues [8] found significant improvement over time for overall self-awareness as well as awareness for cognitive and behavior/affective functioning. Awareness for motor/sensory functioning did not improve over this time interval, but unawareness in this area was minimal at baseline assessment. In spite of the improvement over time, persons with injury still rated themselves as less impaired at 1 year post-injury than did caregivers for overall functioning, cognitive functioning, and behavioral affective functioning. Patients selected for treatment in post-acute community integration programs may be especially likely to have significant ISA as persons with relatively mild cognitive impairment but who are having poor outcomes due to ISA may be attractive candidates for such programs. In a study of 66 persons with TBI treated in a post-acute community integration program, Sherer and colleagues [3] found that 97 % had some degree of impairment of self-awareness.

## **Neural Substrate**

Investigation of self-reflection in non-injured persons has indicated that the anterior medial prefrontal cortex and the posterior cingulate have increased activation during tasks requiring selfawareness [9]. In an fMRI study comparing typically developing adolescents to adolescents with TBI, Newsome and colleagues [10] found that injured adolescents showed greater activation of more posterior brain areas such as the cuneus, lingual gyrus, and parahyppocampal gyrus when performing a task requiring judgments about the self.

While injured adolescents did not show greater activation of the anterior medial prefrontal cortex, they did show increased activation of posterior cingulate white matter. The authors interpreted these findings as indicating that disruption of prefrontal connectivity resulted in greater dependence on posterior regions for this task. This interpretation is consistent with the notion that self-awareness depends on a widespread neural network requiring integration of multiple brain areas. Diffuse axonal injury (DAI) is a key aspect of the neuropathology of TBI [11]. Widespread DAI results in partial disconnection of key cortical regions that normally function in an integrated manner to support complex neurobehavioral functions. Thus, it is to be expected that many persons with injury will show ISA even if they do not have focal contusions to areas such as the medial prefrontal and posterior cingulated cortices that are primarily responsible for self-awareness. Preliminary evidence [12] indicates that DAI is a key finding in injured persons with ISA.

# Association with Cognitive Impairment and Other Neurobehavioral Deficits

As noted above, severity of ISA is associated with overall TBI injury severity and thus ISA tends to be associated with other neurobehavioral deficits. While findings are inconsistent, there is some evidence that persons with greater overall cognitive impairment have greater ISA after TBI [13]. Based on the importance of frontal brain regions for self-awareness, there has been interest in the association between ISA and executive function deficits. Findings have also been somewhat inconsistent in this arena, but investigations have generally found a positive association between degree of executive function deficit and degree of ISA [14].

There is growing evidence of a particular association of ISA with deficits in social cognition after TBI. Social cognition is the ability to understand the behavior of others and react appropriately in social situations [15]. As with ISA, impairments of social cognition have been associated with medial prefrontal lesions, cingulate lesions, and white matter lesions [16]. Also, as with ISA, there is some evidence that social cognition abilities are associated with execution functions [17]. Based on these findings, an association between ISA and social cognition is expected. Indeed, self-awareness of some attributes such as social skills and attractiveness only has meaning in a social context. An investigation of ISA and social cognition found that persons with greater impairment of social cognition showed greater impairment of self-awareness [18]. This finding suggests that impaired ability to judge the emotions and thoughts of others may deprive persons with injury of important feedback needed to form accurate judgments of their abilities and their social impact on others. Family members and friends often label the changes in social interaction style due to ISA and impaired social cognition as personality change.

## Impact on Rehabilitation and Functional Outcome

Much of the interest in ISA after TBI is driven by the impact that ISA has on the rehabilitation process and long-term rehabilitation outcomes. For persons in early recovery from TBI, the key impact of ISA is decreased compliance with treatment and failure to observe safety precautions [19]. In addition, patients with more severe ISA require a greater intensity of service, and, thus, greater cost to achieve outcomes similar to patients with less severe ISA [20].

ISA has been associated with increased distress for caregivers of persons with TBI [21]. Since many persons with moderate and severe TBI will have an increased need for caregiver support for an extended period, if not permanently, after injury, preservation of caregiver mental health is a key issue. Caregiver burnout poses a threat to quality of life and community integration for persons with TBI [22].

Of greatest concern, patients with high levels of ISA have been shown to have poor functional outcomes. Sherer and colleagues [4] showed that at discharge from inpatient rehabilitation at a median of 42 days post-injury, patients with high ISA had only half the odds of having a favorable outcome as patients with more accurate selfawareness. This relationship obtained even after adjustment for age, years of education, injury severity, and functional status at admission for rehabilitation. Additional analysis of these data [23] indicated that while virtually every patient showed some degree of ISA, low levels of ISA were not associated with increased risk of poor outcome. With regard to long-term community integration outcomes after TBI, Sherer and colleagues [24] found that, at about 2.5 years postinjury, patients with high ISA had only one quarter the odds of being employed as those with more accurate self-awareness. These results obtained even after adjustment for injury severity, time since injury, degree of cognitive impairment, and pre-injury employment status. It should be noted that participants in this study were clients in a post-acute brain injury rehabilitation program and, due to admission criteria for such programs, the degree to which this finding would generalize to the larger population of persons with TBI is not known.

### Measurement of ISA

### **General Approaches**

By pure definition, self-awareness is a difficult cognitive function to measure in the individual patient due to its subjective nature. Measurement involves evaluating the extent to which an individual is able to objectively recognize limitations arising from TBI while at the same time appreciating their subjective significance [25]. Rather than being assessed directly, the level of impairment of self-awareness must be inferred from either the patient's self-report of his/her abilities and limitations, or from observation of some aspect of his/her behavior [26].

A distinction can be made between methods best suited to assessing different components of self-awareness. Metacognitive/intellectual awareness lends itself to assessment by knowledgebased methods such as questionnaires or interviews, and on-line awareness (emergent/anticipatory awareness) is more appropriately assessed by performance-based methods [27]. On-line awareness (i.e., the ability to monitor and modify behavior during actual performance) is best assessed by observation of task performance, for example counting the number of self-corrected errors. A number of authors have reviewed the topic of assessment of self-awareness in neurological rehabilitation [26–29] and have broadly identified three main approaches to assessment: These are the discrepancy method, clinical ratings, and observation of behavior on functional activities. Each of these approaches to the measurement of ISA is described below.

## **Discrepancy Method**

Metacognitive awareness or self-knowledge is most commonly measured by comparing the patient's self-report of his or her perceived functional abilities with the report or opinion of another, presumably more objective, source of information. This informant may be a significant other, such as a close relative or friend, or may be a therapist or staff member. Self-ratings of performance may also be compared with actual performance or test results. The most common method is to calculate the discrepancy between the patient's self-ratings and the informant's ratings on a questionnaire to give an indication of the direction and magnitude of self-awareness impairment. For example, when the informant score is subtracted from the patient score, a positive discrepancy score indicates that the patient overestimates his or her level of ability compared to the informant, or has an impairment of selfawareness. A zero or near zero discrepancy score indicates no impairment of self-awareness, and a negative score indicates the patient underestimates his or her level of ability compared to informants.

The first self-awareness questionnaire of this type to be developed for use with the TBI population was the Patient Competency Rating Scale (PCRS) [30]. The PCRS is a 30-item questionnaire which uses a 5-point Likert scale to rate the ease with which the patient is able to perform behavioral tasks including activities of daily living (e.g., how much of a problem do I have in preparing my own meals?), cognitive skills (e.g., how much of a problem do I have in remembering names of people I see often?), interpersonal skills (e.g., how much of a problem do I have in recognizing when something I say or do has upset someone else?), and emotional status (e.g., how much of a problem do I have in keeping from being depressed). The PRCS is available in both patient and significant other versions and self-awareness scores are usually calculated using the discrepancy method described above. Other less sensitive methods of scoring include comparing the average perceived competency score across all items (range = 1-5) for patients and informants (e.g., [31]) or classifying patients into three groups based on whether the highest number of items have patient self-ratings greater to, equal to, or less than the informant ratings [32]. Another approach has been to calculate the mean PCRS difference score on individual items [33]. The PCRS has a demonstrated test-retest reliability for the patient (r=0.97) and informant versions (r=0.92) [6] and inter-rater reliability for staff ratings (average r=0.92) [31]. Borgaro and Prigatano [34] modified the PRCS to a 19-item scale for use in acute neurorehabilitation and demonstrated that the PCRS-NR had three psychometrically sound factors relating to emotional, interpersonal and cognitive functioning.

A range of other questionnaires have been developed to assess self-awareness in the TBI population using the discrepancy method. Examples of these include the Awareness Questionnaire [35], the Functional Self-Assessment Scale [36], the Head Injury Behaviour Scale [7], and the Dysexecutive Questionnaire (DEX) from the Behavioral Assessment of the Dysexecutive Syndrome (BADS) [37]. Other self-report questionnaires which have not been specifically designed for measuring awareness have been adapted for this purpose by creating a significant other version of the questionnaire and using the discrepancy method to compare it with self-ratings. For example, this method has been used with the Mayo-Portland Adaptability Index and

1		2	3	4	5
muc	h se	a little worse	about the same	a little better	much better
	1.	How good is your	ability to live indepe	endently now as c	ompared to
	2.	How good is your ability to manage your money now as compared to			
	3.	How well do you get along with people now as compared to before your injury? (behavioral/affective) How well can you do on tests that measure thinking and memory skills			
	4.				
	5.	How well can you do the things you want to do in life now as compared to before your injury? (behavioral/offsetive)			
	6.	How well are you able to see now as compared to before your injury?			
	7.	How well can you (motor/sensory)	hear now as compar	red to before your	· injury?
	8.	How well can you move your arms and legs now as compared to before your injury? (motor/sensory)			
	9.	How good is your coordination now as compared to before your injury?			
	10.	How good are you	at keeping up with	the time and date	and where you
	11.	How well can you (cognitive)	concentrate now as o	compared to befo	re your injury?
	12.	How well can you before your injury?	express your though (cognitive)	ts to others now a	as compared to
	13.	How good is your your injury? (cogni	memory for recent e tive)	vents now as com	pared to before
	14.	How good are you injury? (behavioral/	at planning things r	now as compared	to before your
	15.	How well organized (cognitive)	d are you now as co	mpared to before	your injury?
	16.	How well can you before your injury?	keep your feelings in (behavioral/affective	n control now as o e)	compared to
	17.	How well adjusted injury? (behavioral	emotionally are you affective)	now as compared	d to before your
Modified	l with	permission from She	erer M. Bergloff P. H	Boake C. High W	. Levin E. The

<sup>1</sup>Modified with permission from Sherer M, Bergloff P, Boake C, High W, Levin E. The Awareness Questionnaire: Factor structure and internal consistency. Brain Injury 1998;12:63-68.

**Fig.1** Awareness questionnaire. Modified with permission from Sherer M, Bergloff P, Boake C, High W, Levin E. The Awareness Questionnaire: Factor structure and internal consistency. Brain Injury 1998;12:63–68

the Sickness Impact Profile in self-awareness studies [38, 39], and to determine self-awareness for specific cognitive functions such as memory performance [40, 41].

The Awareness Questionnaire (AQ) developed by Sherer and colleagues [35] has emerged as one of the mostly widely used self-awareness questionnaires and is provided in Fig. 1 as an example of the discrepancy approach. The AQ has three versions (patient, clinician and family member) each consisting of 17 items which rate the patient's functioning following TBI compared to pre-injury on a 5-point scale where 1=much worse and 5=much better. Total scores range from 17 to 85 with scores of 51 indicating the patient's functioning is similar to pre-injury. Three subscales with strong internal consistency have been demonstrated using factor analysis—motor and sensory (four items), cognitive (seven items), and behavioral and affective (six items) [35]. Discrepancy scores are generated by subtracting clinician or family member ratings from patient self-ratings and can range from -68 to +68. The AQ has been used in numerous studies of self-awareness after TBI demonstrating its validity, for example relationship to long-term employment outcome [24]. In one study, the AQ showed only moderate correlations with the PCRS but both measures performed comparably in predicting employability at discharge [4]. Based on these results, preliminary cut-off scores were proposed with AQ discrepancy scores of <20 indicating mild or no impairment of self-awareness, 20–29 indicating moderate impairment, and >29 severe impairment of self-awareness [4].

Research using the discrepancy method to evaluate impairment of self-awareness has generally demonstrated that the majority of patients underestimate their impairments or limitations compared to ratings by family or staff [5]. However the accuracy with which patients' self-ratings reflect their actual perceived brain injury-related limitations may be questioned as it can be influenced by a number of factors [42]. These include the contribution of psychological factors such as denial of disability, the desire for a favorable presentation of self, the degree of willingness to engage in selfdisclosure, and caution about how the information may be used in clinical decision making. For example, a client with TBI may be concerned that disclosing details of difficulties may delay discharge or clearance to return to work or driving [43].

A second difficulty with assessing impairment of self-awareness is establishing an objective measure of functional competency or limitations against which to compare the patient's self-report [42]. The reports of family members may be biased as a result of denial or unawareness of the extent of disability in the early stages following TBI, especially while the patient is still in hospital, and at later stages post-injury the emotional stress, strain and fatigue associated with caring for a person with TBI may lead family members to overestimate the extent of disability [39]. While an obvious solution may be the reliance on clinicians' ratings as a more objective source of information, these too may be limited by a lack of knowledge of the patient's premorbid personality and abilities, as well as limited exposure to the patient's performance in real-life environments [42].

Some studies have overcome the difficulties with the accuracy and objectivity of informants' reports by using comparison with neuropsychological test performance. For example, Allen and Ruff [44] used a questionnaire to evaluate the self-awareness of patients with TBI and controls in the areas of sensorimotor function, attention, mathematics, language and reasoning, learning and memory, and reasoning. Self-ratings were compared with performance on neuropsychological testing to determine the level of selfawareness. Similarly, in a metamemory study, Livengood and colleagues [40] used comparison of performance on memory assessments with patient predictions of performance to measure the level of self-awareness. Self-ratings can also be compared to performance on functional tasks. For example, in acquired brain injury rehabilitation, the Assessment of Awareness of Disability (AAD) can be used following performance of activities of daily living to compare patient's selfratings of motor and process skills with therapist's ratings [45].

A final issue raised with respect to using the discrepancy method for measuring self-awareness is the magnitude of the difference score required as a cut-off for identifying impairment of self-awareness [39]. As highlighted with the PRCS above, several different approaches can be applied to determine a discrepancy score, and the approach is likely to influence the number of patients who are identified as having an impairment of self-awareness [39]. The magnitude of discrepancy scores is also restricted if the informant rates the patient as being fully competent, thereby allowing little room for discrepancies in ratings to be achieved [4, 46].

## **Clinician Rating**

The clinician rating method of assessing the level of self-awareness in patients with TBI relies upon clinical judgment to determine the extent of impairment of self-awareness using some form of rating scale. In essence this approach is not significantly different from the discrepancy approach, except that instead of informant ratings or test scores, the clinician uses his or her own Table 1 Self-awareness of deficits interview<sup>a</sup>

1. Self-awareness of deficits
Are you any different now compared to what you were like before your accident? In what way? Do you feel that anything about you, or your abilities has changed
Do people who know you well notice that anything is different about you since the accident? What might they notice?
What do you see as your problems, if any, resulting from your injury? What is the main thing you need to work on/would like to get better?
Prompts:
Physical abilities (e.g., movement of arms and legs, balance, vision, endurance)?
Memory/confusion?
Concentration?
Problem solving, decision making, organizing and planning things?
Controlling behavior?
Communication?
Getting along with other people?
Has your personality changed?
Are there any other problems that I haven't mentioned?
2. Self-awareness of functional implications of deficits
Does your brain injury have any affect on your everyday life? In what way?
Prompts:
Ability to live independently?
Managing finances?
Looking after family/manage home?
Driving?
Work/Study?
Leisure/Social Life?
Are there any other areas of life that you feel have changed/may change?
3. Ability to set realistic goals
What do you hope to achieve in the next 6 months? Do you have any goals? What are they?
In 6 months time, what do you think you will be doing? Where do you think you'll be?
Do you think your head injury will still be having any affect on your life in 6 months time?
If yes: How?
If no: Are you sure?
Scoring. Total SADI scores range from 0 to 9 with higher
scores indicating greater impairment of self-awareness.

Scores for each of the three subscales range from:

- 1 = mild self-awareness impairment
- 2=moderate self-awareness impairment

3=severe self-awareness impairment

<sup>a</sup>Modified with permission from Fleming JM, Strong J, Ashton R. Self-awareness of deficits in adults with traumatic brain injury: how best to measure? Brain Injury. 1996;10:1–15 knowledge of the patient's performance for comparison with self-reports. This method is therefore reliant upon the clinician's judgment and his or her ability not to be influenced by personal characteristics of the patient such as their likeability, attractiveness, and communication skills [29].

Clinician ratings of self-awareness are generally based on the patient's responses to a structured interviewed, and there are several interview-based assessments including the Self-Awareness of Deficits Interview (SADI) [42], the Self-Regulation Skills Interview (SRSI) [47], and the Awareness Interview [13]. The SADI is provided in Table 1 as an illustration of the use of a clinician-rated structured interview for measuring metacognitive knowledge or intellectual awareness. The SADI measures the level of selfawareness on three subscales: (1) self-awareness of impairment, (2) self-awareness of functional implications, and (3) ability to set realistic goals [42]. The questions on the SADI build upon previous interview formats used in psychiatry [48] and social cognition research [49]. The patient's responses are transcribed verbatim by the interviewer during the interviewer, or alternatively, interviews may be audiotaped. The responses are rated on a 4-point scale similar in format to the scale used by Bisiach et al. [50] for rating anosognosia for hemianopia, but adapted to cover the range of impairments possible following TBI. On each dimension, a score of 0 indicates no disorder of self-awareness and a score of 3 indicates a severe disorder of self-awareness, giving a total possible range of scores from 0 to 9. Detailed scoring guidelines are provided.

In designing the rating scale a number of points were taken into consideration. First, patients with TBI may display "borderline" awareness in which they acknowledge certain impairments (notably physical limitations), and ignore others (such as cognitive and personality changes), or they can describe problems that others have noticed but they are not convinced themselves that they exist [32, 51]. Second, understanding the functional implications of impairments may be limited by a lack of opportunity to try various tasks in the acute post-injury phase. Third, realistic goal setting is seen to reflect the degree of self-awareness [52], with the

adjustment of pre-injury goals seen as an important step in the development of self-awareness after TBI [51]. Finally, in scoring an individual's responses on the rating scale, the interviewer needs some background knowledge of the patient's level of function. Therefore, a relative's and/or clinician checklist can be used to gather collateral information to assist with assigning SADI scores. A full version of the SADI and the checklist are available from the authors.

An initial inter-rater reliability study indicated acceptable agreement between raters for total scores with an intraclass correlation coefficient (ICC) of 0.82 [42]. A second inter-rater reliability study where two raters were both present during the actual interviews yielded a higher ICC of 0.85 [53]. Test-retest reliability over a 2- to 4-week period was high (ICC=0.92) [54]. The SADI has been significantly correlated with measures of frontal lobe functioning and injury severity [55], and with the AQ and measures of work status in individuals with acquired brain injury [56].

In contrast to the SADI which measures metacognitive awareness, the SRSI [47] was designed to measure on-line awareness skills in relation to a main area of difficulty identified by the patient. The SRSI has six items (emergent awareness, anticipatory awareness, readiness to change, strategy generation, degree of strategy use, and strategy use) which are scored by the interviewer using a 10-point rating scale. The items are grouped into three indices derived by factor analysis which include Awareness, Readiness to Change, and Strategy Behaviour Index [47]. The SRSI has test-retest (0.81-0.92) and inter-rater (0.69–0.91) reliability [47] and was significantly correlated with SADI scores and work status in adults with acquired brain injury [56].

The Awareness Interview [13] involves the use of both a clinician-rated interview and the discrepancy method by comparing neuropsychological test scores to patient responses in order to quantify self-awareness. Interview questions address the five specific areas of motor, intellectual, orientation, memory, speech or language, and visual perceptual impairment. Interview responses are scored on a 3-point scale to reflect the perceived amount of impairment on each function. These scores are then compared to ratings by a neuropsychologist on a comparable scale based on the results of neuropsychological testing of the same functions. Deviation scores are generated by comparing the two sets of scores to give scores ranging from 0= no discrepancy to 2=maximum discrepancy. Two additional questions (regarding awareness of the reason for hospitalization and awareness of general test performance and ability to resume normal activities) are scored using deviation scores which are then totaled with the other six deviations scores to give an Awareness Index (range 0-16). The Awareness Index therefore represents a more objective way of using clinician-rated interviews to measure self-awareness, but is related to only a limited spectrum of impairments seen following TBI and does not include items relating to executive dysfunction, and interpersonal, behavioral and emotional changes for which people with TBI often lack self-awareness.

Clinician ratings are also used to give scores on the ISA and Denial of Disability (DD) Clinicians' Rating Scale [57]. This scale was designed to differentiate between ISA of neurological origin and DD of psychological origin in the individual patient with TBI. Both the ISA and DD scale consist of ten items which are rated as "yes" or "no" by the clinician with items rated "yes" then scored on a 0-10 scale of severity giving a maximum possible total score of 100 on each scale. Items on the ISA scale relate to a lack of spontaneous reports of difficulties, little affective reaction to feedback, "cognitive perplexity" in response to feedback or difficulties, other higher level cognitive problems such as impaired initiation, self-monitoring and planning. In contrast items on the DD include some minimal admission of difficulties, a negative affective reaction to feedback, use of arguments or excuses to explain behavior, a lack of severe impairment in initiation, planning, and self-monitoring on testing, and possible catastrophic reactions when faced with failure. Inter-rater reliability on the scales was generally high except when the clinician rated the degree of impaired self-awareness in the ISA group [57].

# **Observation of Behavior**

The observation of task performance or behavior provides a third method of assessment of selfawareness, which particularly targets on-line awareness. Observational methods do not usually involve patient self-reports but focus on the patient's task selection and avoidance, error detection, and error correction during task performance [26]. In one of the first studies of online awareness after TBI, Hart and colleagues [58] engaged participants in a naturalistic multi-level action task that involved making toast, wrapping a gift, and packing a lunchbox. Performances were videotaped and then analyzed to record instances of error correction (i.e., attempts to redress an error) and error detection (e.g., verbalizations, exclamations, facial expressions, and manual gestures signifying the participant's awareness that an error had occurred). This study showed that aspects of on-line awareness could be reliably and objectively measured without reliance on self-report.

Ownsworth and colleagues [59] used a similar behavioral approach to measuring on-line awareness with a TBI participant during meal preparation and work activities. This included measures of error frequency (i.e., recording errors that compromised safety, outcome or time efficiency) and error behavior. Error behavior was systematically observed using a "pause, prompt, praise" technique which involved the therapist initially allowing a "pause" following an error to allow for self-correction, then a non-specific prompt, followed by a specific prompt if the error was not corrected. Errors were categorized as selfcorrected, corrected with non-specific prompt, or corrected with specific prompt. This approach was adapted in a subsequent study [60] to classify errors as self-corrected errors (i.e., corrected after a 5-10 s pause) or therapist-corrected errors (i.e., corrected after a prompt) and checks (i.e., requests for advice or verification). In both studies, inter-rater reliability for frequency and classification of errors was established.

In summary, the standardized assessment of on-line awareness remains an under-developed area. It can be concluded that self-awareness can be measured in a number of ways which tap into different aspects of self-awareness. Therefore for any individual patient, it is advisable to use more than one approach to establish an understanding of his or her level of self-awareness across domains.

# **Empirical Studies of Interventions**

### **General Approaches**

The past two decades have seen an increasing emphasis on the development of interventions specifically targeting impairment of self-awareness after TBI, or incorporating self-awareness training into other cognitive rehabilitation approaches. A small but growing body of empirical studies provides evidence that self-awareness interventions can be effective in enhancing rehabilitation outcomes. Consequently, a review of the latest evidence concluded that metacognitive strategy training (i.e., targeting self-monitoring and selfregulation) should be a practice standard in the cognitive rehabilitation of people with executive dysfunction following TBI [61]. The treatment of ISA after TBI has been reviewed previously by several authors [26, 51, 62-64] and interventions described include feedback, education, behavior therapy, psychotherapy, milieu-oriented programs, game formats, group programs, and real world experiences. This section reviews the research relating to self-awareness interventions including the use of various approaches to providing feedback, predicted performance, occupation-based experiences, and the use of other techniques such as psychotherapy, education, and group programs.

### Feedback Approaches

The provision of feedback to patients is a fundamental component of rehabilitation and includes feedback on test results, functional performance, and strengths and limitations. Arguably the primary rationale for providing feedback is to improve a patient's self-awareness thereby enabling him or her to identify areas for improvement, or the need for strategies to improve performance. Timely, specific and consistent feedback is emphasized as being an important component of all awareness interventions [51, 65]. A systematic review of intervention studies which used a feedback component to improve self-awareness identified 12 studies of varied methodological quality including single case experimental designs [66]. Three studies were randomized controlled trials involving a total of 62 people with brain injury of mixed etiology [67-69]. A meta-analysis found a moderate effect size for the pooled estimate of improvement in self-awareness after completing a feedback intervention (Hedges adjusted g=0.64, 95 % confidence interval 0.11–1.16). Furthermore, feedback interventions had large effect sizes for improving functional task performance and patient satisfaction with therapy [66]. Most feedback interventions use a combination of different forms of feedback including selfpredictions and self-evaluations of performance, verbal feedback from a therapist, and in some cases, videotaped feedback. Peer feedback is also an important ingredient of group interventions.

Direct *therapist feedback* is a common approach to facilitating both intellectual and online awareness in patients with TBI [29, 51, 65]. Verbal feedback on performance is thought to be more readily accepted by the patient if provided by a trusted therapist in the context of a strong therapeutic alliance [70]. A "sandwich technique" in which negative feedback is preceded, and followed, by positive feedback is recommended [63, 71]. Klonoff and colleagues [70] described well-timed therapist feedback as an integral component of their cognitive retraining program which led to successful work placement in a case study of a patient with TBI.

While some authors have claimed that direct therapist feedback may be too confrontational and force patients to defend their confabulatory beliefs [72], research has demonstrated that, on the contrary, feedback of self-awareness assessment data led to a decrease in subjective reports of grief in participants with brain injury [73]. Another repeated measures study found that feedback from a consultant neurologist on the findings of brain scans and possible neurobehavioral outcome led to significant improvements in self-awareness as measured by the AQ and SADI in 17 patients with brain injury [74]. Interestingly, in this study, direct feedback was also associated with a decrease in self-reported symptoms of anxiety and depression.

Videotaped feedback has been recommended by several authors as an effective method for improving self-awareness of functional performance including awareness of behavioral and communication problems [62, 64, 75, 76]. Videotaped feedback has been used as an element of several self-awareness interventions with people with TBI (e.g., [59, 77]) and demonstrated to be more effective than verbal feedback and experiential feedback in a randomized controlled trial with 54 participants with TBI [78]. Schmidt et al. [78] used feedback on performance in a meal preparation task on four occasions over a 2-week period. The group that received a combination of video and verbal feedback had significantly greater gains in on-line awareness as measured by an error count and intellectual awareness measured by AQ discrepancy score. Interestingly, there were no changes in the level of emotional distress associated with any of the feedback interventions. McGraw-Hunter et al. [79] also described the use of video self-modeling to teach cooking skills to four individuals with TBI. In this study, the participants were videoed performing the cooking task with step by step direction from the researcher and any errors were edited from the video. In the experimental phase, the participants viewed the video prior to performing the cooking task with a graduated system of prompting, praise and corrective feedback. The approach was effective in achieving skill acquisition for three of the four participants within four training sessions, however the effect on selfawareness was not examined.

### **Predicted Performance**

The technique of predicted performance involves asking patients to *self-predict* their performance prior to completing a task (e.g., the amount of difficulty expected, how much assistance will be needed, how long it will take, or the need for strategies). Following task performance, patients complete a *self-evaluation* in which they rate their performance of the task, which can then be compared with the predicted performance as well as with therapist feedback on the performance [68]. While predicted performance is most commonly used in conjunction with the performance of functional activities, it has also been used successfully to improve awareness of performance on memory tasks in single case studies [80, 81] and for verbal recall and arithmetic tasks in a single group session [82].

### **Occupation-Based Interventions**

Experiential feedback or occupation-based interventions involve participation in real-life activities that allow the person with TBI to discover his or her own errors [71]. The selection of meaningful occupations to improve self-awareness contains elements of "supported risk taking" or "planned failure" and needs to be well-structured and supported by the therapist to minimize any emotional distress [51, 65, 83]. Even without specific intervention targeting self-awareness, individuals with TBI have reported that their selfawareness developed as a result of comparing their current ability to perform familiar occupations with their pre-injury status [83]. An occupation-based approach acknowledges that self-awareness training involves "rebuilding a sense of self" [76, p. 181]; while engaging in structured occupational experiences, the person with TBI uses self-monitoring techniques to discover strengths and weaknesses, and to develop strategies and new ways of doing things, thereby promoting self-efficacy.

Toglia [76] described a dynamic interactional approach to improving self-awareness using engagement in meaningful occupations. In the pre-activity phase, this approach involves the use of techniques such as self-prediction, guided anticipation of challenges, and strategy generation. During the activity, techniques include "stop and check" periods, self-questioning (e.g., Am I keeping track of everything?) and therapist feedback to reinforce strategy use. The postactivity phase can include various forms of selfassessment such as video feedback, self-ratings, guided questioning, and comparison of outcomes with a template or model. Journaling or structured logs may also be used to promote selfreflection on the activity, as well as broader identification of cognitive failures and strategies in daily life. Role reversal is another technique developed by Toglia for promoting self-awareness using functional activities. In role reversal, the therapist performs the activity and deliberately makes errors, while the patient observes the performance and identifies errors and suggests strategies, an approach which may be less cognitively demanding and less threatening than identifying errors during one's own performance.

Several studies have used single case study designs to illustrate the effectiveness of occupationbased approaches using many of the above techniques to improve self-awareness after TBI [59, 77, 84, 85]. Usually occupation-based interventions are used in conjunction with other techniques such as feedback to promote self-awareness. The three randomized controlled trials in the Schmidt et al. [66] systematic review described above each involved occupation-based interventions.

In the first of these, Cheng and Man [67] used a combination of education, experiential feedback, self-prediction and goal setting to significantly improve intellectual awareness as measured by the SADI in 11 intervention participants compared to a control group. In the second study, Goverover et al. [68] used a combination of occupation-based intervention and predicted performance with ten participants with TBI when performing instrumental activities of daily living over six therapy sessions. Compared to a control group, the intervention group improved significantly in task performance and self-regulation skills. However, no significant differences were found for task-specific or general self-awareness.

The third study [69] was a randomized controlled trial with three treatment arms, one of which involved individual occupation-based support. Occupational activities were selected on the basis of participants' goals and performed in their home or community with a focus on self-monitoring and self-correction of errors, and use of self-regulation strategies. After eight weekly intervention sessions participants in this group showed significant improvements in goal attainment, however the level of self-awareness was not explicitly evaluated in the original study. In the subsequent meta-analysis [66], it was found that compared to a wait-list control group, the standardized mean difference of discrepancy scores on the PCRS was not significant for this study by itself (Hedges adjusted g = 0.48, 95 % confidence interval of -0.41 to 1.38).

In another study, Ownsworth et al. [60] used a single case study design to demonstrate improvements in self-regulation skills in two participants with brain injury during meal preparation activities. The metacognitive skills training approach involved a "pause, prompt, praise" approach and incorporated sessions of role reversal, videotaped feedback, and post-task discussion. Specifically, during task performance, the therapist waited (pause) if the participant started to make an error to allow self-correction of the error and provided non-specific direction (prompt) if the error continued. The therapist then affirmed the participant for correct performance (praise). Compared to baseline, both participants showed a significant increase in self-corrected errors, decrease in therapist corrected errors, and decrease in number of times the participant checked to ensure accuracy of task performance. In contrast, another participant who engaged in an extended baseline of behavioral practice without metacognitive skills training showed no significant changes in error correction, and an increase in the number of checks suggesting an increased reliance on therapist support. Interestingly, the participants receiving metacognitive skills training rated themselves lower on the PCRS following the intervention suggesting more accurate self-awareness, whereas the behavioral practice participant perceived greater selfcompetency following the intervention.

Ownsworth et al. [86] also described the use of a similar metacognitive contextual intervention component as part of a larger program for two individuals with TBI and one with stroke to facilitate achieving paid work after long-term unemployment. Techniques used included selfprediction, self-monitoring, and self-evaluation of performance of functional tasks in the participants' homes and workplaces. The program also included group education and support activities, family involvement, sessions with disability support counselors, and a work trial with employer education and support, so it not possible to determine which component or components of the intervention were effective. However, all three participants had maintained paid employment 6 months later.

### **Other Approaches**

Other approaches to facilitating self-awareness following TBI include group therapy, education, adjustment counseling, and psychotherapy. Many of the studies pioneering these techniques and others described above have occurred in the context of comprehensive neuropsychological community integration programs [29, 87-89]. These holistic milieu-oriented programs employ approaches which combine cognitive retraining with psychotherapeutic interventions to address both neuropsychological and emotional or adjustment issues, and to enhance community integration outcomes. While the development of self-awareness has been a major focus of the rehabilitation programs, outcomes generally have been reported in terms of better emotional adjustment and higher levels of productivity, rather than improvements in self-awareness per se. A study by Malec and Moessner [88] however, found that 62 graduates of a comprehensive day treatment program had diminished impairment of selfawareness compared to pre-intervention. They also found that improvements in self-awareness and distress levels were associated with positive behavioral changes but not vocational outcomes. Studies such as these illustrate the difficulties associated with determining the effectiveness of specific self-awareness intervention techniques, as in practice they are usually used in combination, as well as the difficulty determining the extent that gains in self-awareness impact upon community integration outcomes.
The use of *group programs* is an important component of the above comprehensive treatment programs, as it allows for valuable peer feedback, role modeling and support during group discussions [29]. There have, however, been few studies which have specifically examined the use of group interventions to facilitate self-awareness. Ownsworth, McFarland and Young [90] conducted a 16-week group support and psychoeducation program with 21 participants with long-term acquired brain injury. The participants showed significant improvements in levels of on-line self-awareness and strategy use, as well as improved psychosocial function, and gains were maintained at a 6-month follow-up.

*Psychotherapy* is another integral component of many neuropsychological rehabilitation programs that aim to develop self-awareness after TBI. The aim of psychotherapy is to assist patients to explore feelings of loss and anger [83], and to establish realistic goals and reestablish meaning in life [89]. Both individual and group psychotherapy can been used following TBI [29]. For patients who display denial or minimization of information that is too painful to acknowledge, psychotherapy has been recommended to strengthen their emotional readiness to cope with the rehabilitation process [83]. The successful use of a psychotherapeutic approach following TBI has been demonstrated in a case study [91] and as part of a comprehensive day program [29].

Psychotherapy, along with other counseling approaches to facilitate adjustment and acceptance of disability, emphasize the importance of a strong therapeutic alliance. Establishing trust and providing a safe and accepting environment are at the foundations of successful awareness interventions and enhance engagement in rehabilitation [29, 51, 64]. This process may also include a focus on goal setting, in particular discussing and acknowledging the patient's personal goals and incorporating them into meaningful therapy goals [52]. Motivational interviewing has been presented as a goal setting approach which may be incorporated into holistic rehabilitation programs to enhance the therapeutic relationship and facilitate self-awareness and acceptance of disability [92].

Education is another fundamental component of many rehabilitation programs which aim to enhance self-awareness. Educational approaches may include written materials, individual information sessions, groups, and game formats, and content may cover brain function, brain impairment, the effects of TBI, and strategy use. Often family members are included in education sessions [51]. It is thought that, by maintaining an informational format in group education sessions, individuals with TBI can discuss typical brain injury problems in a less threatening way, and hear from others who may be prepared to discuss their personal experiences and cognitive difficulties, thereby promoting self-acceptance [29]. The research is unclear, however, as to whether education alone leads to improvements in selfawareness. For example, a randomized controlled trial of a single 30-min educational session for improving knowledge and awareness in children with TBI did not find any significant improvement [93]. Educational board games have also been developed to enhance knowledge and awareness in patients with brain injury [94, 95]. These were designed to provide opportunity for patients to learn about the cognitive and behavioral sequelae of brain injury through repetition of information in a non-threatening way; however, the approach was demonstrated to be effective only for improving general knowledge and not accuracy of self-appraisal [95]. When combined with facilitated discussion before the game, the approach led to improved self-awareness of personal strengths and limitations in three participants with severe TBI [94].

In summary, this section has described a number of intervention approaches to improving selfawareness following TBI and overviewed the empirical studies that have investigated their effectiveness. It is evident that most selfawareness interventions are comprised of more than one technique and are delivered as programs which aim to improve not only self-awareness, but psychosocial functioning more generally. Further research is required to establish which techniques, or combinations or techniques, are most effective, and with what type of patients. Indeed for some individuals with severe cognitive impairment, attempts to improve self-awareness may not succeed, and interventions should focus more on compensation for the impairment in self-awareness (e.g., in order to prevent the individual from engaging in unsafe or risky behaviors) [51] or use behavioral interventions which do not require self-awareness to improve function [72]. For other individuals, interventions may lead to improved self-awareness but this may not necessarily translate into functional gains (e.g. due to increased emotional distress), and further research is needed to determine what type of intervention is best suited to particular awareness sub-types.

# Recommendations for Clinical Practice and Case Examples

#### The Big Picture

As the review above indicates, there are multiple possible approaches to address ISA with the goal of improving treatment compliance and patient outcome. Unfortunately, none of these approaches has extensive empirical support. The current state of knowledge does not support specific clinical guidelines for treatment of ISA. Rather, the clinician must rely on experience and clinical judgment to develop an individualized approach to meet the needs of the patient based on the patient's clinical status, phase of recovery, social support network, and other considerations. All approaches to treatment will rely on feedback to the person with injury, to some degree, and all approaches will be enhanced if the treating clinician(s) can establish an effective therapeutic alliance with the patient.

Therapeutic, or working, alliance is a construct commonly described in the psychotherapy literature. It is generally defined as having three components: the bond between the clinician and the client, agreement on the means of therapy, and agreement on the goals of therapy [96]. This bond may be based on the therapist's ability to convey empathy, the client's confidence in the therapist's expertise, the therapist's genuine commitment to facilitating the client's best interests, and/or other aspects of the relationship. However, this bond is not based on friendship or personal affection. Clinicians with limited training and/or experience in counseling may fail to appreciate this difference. In persons with brain injury, a stronger alliance between the patient and therapist has been shown to result in more accurate self-awareness for the patient [97]. An effective therapeutic alliance can be regarded as a necessary though not sufficient condition for improvement in self-awareness in rehabilitation of patients with TBI.

One key issue that can drive the treatment approach selected is the primary goal of the awareness intervention. If the primary goal is treatment compliance and acceptance of obtainable long-term goals, the approach will be different than if the goal is re-establishment of a positive sense of self after catastrophic injury.

# Enhancing Treatment Compliance in a Patient with ISA

In a different context (compliance with wearing a splint), Kuipers and colleagues [98] surveyed rehabilitation clients to determine factors that the clients believed affected their compliance with treatment. These factors were (1) client and therapist collaboration in treatment, (2) client trust in therapist expertise, (3) client understanding of the purpose of treatment, (4) fit of the therapeutic approach with client goals and lifestyle, (5) support from family and friends, and (6) any discomfort, including feelings of stigma, caused by being in treatment. With a little modification, these factors are applicable to interventions to improve self-awareness. Interventions pursued with these issues in mind are likely to maximize therapeutic alliance between the therapist(s) and client. We will describe a clinical case of a person with profound ISA after TBI that illustrates how therapy guided by these principles can facilitate compliance with treatment and lead to a favorable outcome.

Tom was a 25-year-old male who sustained TBI at age 15 in a motor vehicle collision. Records indicated an initial Glasgow Coma Scale score of 7 indicating severe injury. CT imagery indicated a left temporal bone fracture with hemorrhagic contusion of the left temporal lobe, widespread subarachnoid hemorrhage, diffuse brain swelling, and a number of small contusions primarily in the frontal lobes. At initial evaluation, Tom was found to be oriented. His speech was mildly dysarthric and he spoke at a rapid rate causing some problems with intelligibility. Testing showed impaired comprehension for complex language tasks, moderate impairment of verbal memory, decreased dexterity with the right upper extremity, and poor problem solving. Tom's social presentation was awkward. Comments were occasionally off topic. He answered questions impulsively and frequently interrupted the interviewer.

Four years after his injury, Tom eventually graduated from high school with tremendous support from his widowed mother. He was an only child. Tom stated that his goal was to find employment as an insurance agent. Tom had no history of any paid employment. He had completed previous rehabilitation programs of various types including training to be an upholsterer, but was never placed in a job due to his insistence on working as an insurance agent and the collective judgment of all others involved, including his mother, that there was no chance that he could successfully do this type of work.

Tom was admitted to a comprehensive, postacute brain injury rehabilitation program and received 3.5 h of therapy a day, 4 days a week. Therapy goals were to improve speech intelligibility and social skills, implement use of compensatory strategies to improve memory, and develop a plan for placement in paid work. Tom denied having any difficulties at all with intelligibility, social interactions, or memory and indicated that he already had a plan to work as an insurance agent. The team avoided confronting Tom on any of these issues. To address communication issues, we videotaped Tom interacting with other program clients and reviewed the videotapes with him in individual sessions. Since Tom declined to make any notes or use any memory strategies that he would need to initiate, we developed cue sheets to prompt him through mul-

tistep tasks. These cue sheets were small enough to easily fit in Tom's pocket. To work on developing a plan for job placement, we tried Tom out in various simulated jobs we could create in the associated rehabilitation hospital. Tom assured us that it was pointless to work on any job activity that was not related to becoming an insurance agent, but with coaxing we were able to get him to participate in activities, particularly if these activities gave him the chance to interact with therapists at the hospital. On a few occasions, he made inappropriate remarks to female therapists and we firmly redirected him. He denied doing anything wrong, but because he was motivated to continue this opportunity for interaction, we were able to get him to modify his behavior.

Therapists were always focused on being supportive of Tom. We complimented him any time he spoke more slowly or used one of the cuing sheets. We looked for any opportunity to praise his performances on simulated jobs and generally did not comment on errors. Rather, we simply had him redo the task until it was correctly done. Tom was naturally deferent to male authority figures so the program director always had Tom address him as doctor when they spoke. Even though Tom denied any need for treatment, therapists explained the purpose behind each task on which we worked. While Tom continued to focus on work as an insurance agent, we repeatedly emphasized that we were working to help him achieve his goal of obtaining a job. Early on, we fully explained the rationale behind our approach to Tom's mother. She was very supportive of our approach with Tom and she had great influence on Tom as she was the only person that Tom interacted with regularly prior to admission to our program. Finally, we normalized all tasks to the greatest extent possible. Staff made a point of speaking more slowly around Tom to show that a slower rate was easier to understand. Staff used cuing lists in front of Tom to help them remember tasks that they were doing. We described all simulated work tasks as preparations to help Tom achieve his eventual goal of working.

After several weeks of working with Tom, we identified work tasks that he could complete and the amount of cuing needed to guide him through these tasks. We also determined that while Tom very much enjoyed interacting with others, such interactions frequently got him off task. By negotiating with the manager of a local restaurant, we were able to place Tom in a job doing custodial work after hours at the restaurant about 10 h a week. We provided onsite job coaching for the first 4 weeks of employment. Tom initially resisted this placement, but we emphasized that this would be his first paid job and that it might eventually lead to another job that was more similar to his eventual goal. Tom's mother played a key role in convincing Tom to give the job a chance. While Tom always saw the job as transitional, he remained at the placement. He found the paychecks rewarding and began to go on outings without his mother to the movie theater and the local mall, activities that he had never engaged in prior to treatment.

By focusing on compliance as our goal rather than insisting that Tom have the same view of his situation as we did, we were able to make great progress in our work with Tom. We believed that if Tom could become convinced that we were on his side and trying to do the best we could to help him that he would be more likely to comply with our requests. At discharge assessment, we could not demonstrate that Tom's speech intelligibility, social skills, and/or memory were improved. However, he was consistently using the simple cue cards we created for his work duties.

# Facilitating Establishment of a Positive Self-Concept in a Patient with ISA

Formation of an accurate self-perception is a complex cognitive task requiring integration of multiple cognitive systems [99]. Self-awareness requires, among other skills, accurate observations of one's behaviors and their outcomes, integration of individual behavioral performances into a coherent self-concept, introspection and reconsideration of self perceptions over time and across settings, and assimilation of direct and indirect feedback from social interactions. Achieving more accurate self-awareness is only a preliminary step toward re-establishing a positive sense of self after a catastrophic injury. Pioneers in community re-integration programming for persons with TBI such as Ben-Yishay et al. [100] and Prigatano [101] have viewed psychotherapy as a key aspect of therapy intended to facilitate improved functioning and eventual return to work. Following TBI, injured persons may have a profound sense that something is not right. In the presence of ISA, this vague sense of something being wrong is not connected to an accurate appraisal of the cognitive and behavioral deficits caused by the injury. Rather, the injured person is confused and frustrated [101]. Caregivers and family members will often try to help the injured person by pointing out deficits due to the injury as an explanation for the feeling that things are not right. Often this feedback will simply result in more frustration leading to strain in relationships with the primary social support network which in turn leads to greater feelings of despair and abandonment.

For persons with mild TBI who are distressed after injury, psychotherapy may be the only therapy needed. However, in our experience, the most successful approach to addressing a lost sense of self in patients with significant cognitive and/or behavioral deficits combines therapy to address the deficits, feedback to improve self-awareness, and psychotherapy to address the injury to the self. Prigatano's model [101] for psychotherapy after TBI focuses on the importance of work (productive activities), love (passion, intimacy, and commitment to others), and play (a sense of the inner playful self). Description of all the intricacies of providing psychotherapy to persons with cognitive and social communication deficits is beyond the scope of this chapter, but we will describe a clinical case to illustrate how cognitive rehabilitation, self-awareness, and psychotherapy interventions can be integrated to facilitate a favorable outcome.

Joan was a 35-year-old divorced female who sustained TBI in a motor vehicle collision. Her Glasgow Coma Scale on admission to the Emergency Department was 10 indicating a moderate injury. Duration of post-traumatic confusion (amnesia) was 8 days. She received 2 weeks of inpatient rehabilitation that addressed cognitive and behavioral deficits as well as her fractured right forearm. Goals included improving functional skills such as dressing, cooking, etc. while she remained with a cast on her right arm. By 3 months post-injury, the cast was removed and she returned to full-time work as a sales clerk in a department store. She immediately noticed difficulty with fatigue and asked to be cut back to 20 h a week from her usual schedule of over 40 h a week.

Cognitive testing revealed mild impairment of verbal memory and mild cognitive slowing on motor and verbal tasks. Performance on motor tasks was influenced by residual weakness in her right hand and arm that was thought to be related to her fracture rather than to her brain injury. Interview with Joan's sister revealed that she thought that Joan was irritable and distractible since her injury. She described Joan as having a total change in personality. This resulted in conflict when they were together and the sister was spending less time with Joan as a result. Joan was initially reluctant to allow program staff to speak to her work supervisor, but eventually gave permission. The supervisor indicated that Joan had been a top performer prior to her injury frequently receiving bonus checks based on her high sales totals. Initially on return to work, Joan had difficulty recalling procedures for processing coupons and exchanges. Her supervisor was not surprised by this as procedures had changed shortly before Joan had been injured and Joan had been away from work for 3 months. However, Joan became very frustrated when processing coupons and exchanges and she rejected help from others. The supervisor indicated that even though Joan had been short with fellow employees causing some hurt feelings, he still regarded her as one of his best sales clerks.

Since Joan was working 20 h a week, her therapy program was scheduled around her work schedule. She received 3 h of therapy a week working on memory compensation, 2 h a week working on strengthening and increasing flexibility in her right hand and arm, and 1 h of psychotherapy.

The cognitive therapist obtained materials from Joan's job site that described steps in processing purchases in which coupons were used, processing exchanges, and various other work tasks. Joan resisted efforts to review these materials in therapy sessions as she indicated that she knew them perfectly well and that her problems at work had been due to fatigue or computer malfunction. In a process that took three or four sessions, the therapist was able to convince Joan to write down the procedures and then compare her description of the procedures to information provided by her supervisor. Joan was only willing to do this if the therapist did not look at her description, but allowed Joan to make the comparison independently. While Joan silently looked over her work and the work materials, the therapist noticed that Joan added some notes in the margins of her page. The therapist commented that she frequently used cue sheets to help her recall the steps in complex processes. She challenged Joan to see how clever she could be in developing a cue sheet that would allow even the therapist who was inexperienced in these procedures to complete them. Reluctantly, Joan accepted the challenge. Joan's initial efforts involved a good deal of text description and the therapist worked with Joan to create briefer cuing strategies that emphasized key words and acronyms.

Concurrent with Joan's work with the cognitive therapist, Joan was doing strengthening and stretching activities for her right hand and arm with an occupational therapist. As her rapport with Joan increased, the therapist engaged Joan in a conversation about the benefits of regular exercise for reducing fatigue and improving cognitive abilities. Joan had been an avid cyclist before her injury, but had not engaged in any regular exercise since her injury. She agreed with the therapist to set a goal of walking an hour day, 4 days a week. The therapist was confident that Joan could easily reach this goal based on the intensity of her past cycling program.

The psychotherapist initially focused on building a therapeutic alliance with Joan. He encouraged Joan to talk about her feelings about the accident and how she felt she had been changed by the accident. He reflected back to her the feelings of despair and confusion. After a couple of sessions he asked Joan to describe herself as she had been before her injury. Key attributes that Joan identified for herself were that she was a high energy person and a tireless worker. She became tearful when describing the overwhelming fatigue that she had felt at times since her injury. Knowing that Joan had started a walking program, the therapist commented on how remarkable it was that she could walk for a full hour in spite of her fatigue. Joan responded that it was easy, that anyone would walk for an hour. She went on to add that she planned to increase her walks to 11/2 h starting the next week. A transforming moment in therapy occurred with the therapist asked Joan what she liked to do for fun. She had to give this a good deal of thought before she answered. She mentioned cycling and shopping with her sister and admitted that she had done neither of these things since her injury. With the therapist's encouragement she committed to ask her sister to go shopping with her the next weekend.

Within 6 weeks of treatment, Joan had perfected her cuing sheets for work and regained normal strength in her right hand. She had reconnected with her sister who had been her primary source of social support since her divorce. The treatment team negotiated a gradual return to fulltime work with Joan's supervisor with Joan increasing her work hours by 5 h a week based on feedback from her supervisor that her work was satisfactory. Four weeks later Joan was working fulltime and felt that she was making progress in adjusting to the "new" Joan she found herself to be after her injury.

Joan's treatment addressed the triad of work, love (her relationship her sister), and play (taking the initiative to engage in activities that had been fun for her prior to her injury). Since Joan's cognitive impairments were relatively mild, she was quickly able to improve her skills in developing self-cuing strategies and she showed good benefit from these strategies. Walking partially replaced cycling as a form of exercise as her physician had restricted her from return to cycling. Joan's energy level seemed to improve as she increased the duration of her walks. Rather than reassuring Joan that she was not that severely injured and that she would likely continue to improve, the psychotherapist accepted the level of distress that Joan said she was experiencing and looked for opportunities to encourage her to engage in activities that would likely decrease her distress. The coordinated approach taken by the treatment team maximized the social influence that the team could have in modifying Joan's behavior.

#### Summary

Research with persons with TBI shows that impairment of accurate self-awareness is common early after injury and in the post-acute period. Nonetheless, the level of self-awareness improves over time. Preliminary findings suggest that the degree of impairment of self-awareness must reach a critical level before it adversely affects outcome. For an important subgroup of persons with TBI, ISA is a key deficit that affects compliance with treatment and outcome. Given the impact of ISA, it is important that therapists working with persons with ISA are familiar with approaches to assessment and intervention. While there are a number of proven approaches to assessment, treatment approaches require additional investigation though it seems clear that any successful treatment will involve provision of feedback and that client acceptance of feedback and compliance with treatment is facilitated by a strong therapeutic alliance between the client and treatment team. When designing interventions to address ISA, it is important to have a unified approach involving the entire treatment team and to tailor the approach to address compliance or self-identity depending on the needs of the client.

Acknowledgement Preparation of this chapter was partially supported by the U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020.

# References

- Sherer, M., Nakase-Thompson, R., Yablon, S. A., & Gontkovsky, S. T. (2005). Multidimensional assessment of acute confusion after TBI. Archives of Physical Medicine and Rehabilitation, 86, 896–904.
- Fleming, J., & Strong, J. (1999). A longitudinal study of self-awareness: Functional deficits underestimated by persons with brain injury. *Occupational Therapy Journal of Research*, 19, 3–17.
- Sherer, M., Boake, C., Levin, E., Silver, B. V., Ringholz, G., & High, W., Jr. (1998). Characteristics of impaired awareness after traumatic brain injury. *Journal of International Neuropsychological Society*, 4, 380–387.
- Sherer, M., Hart, T., Nick, T. G., Whyte, J., Nakase Thompson, R., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84, 168–176.
- Newman, A. C., Garmoe, W., Beatty, P., & Ziccardi, M. (2000). Self-awareness of traumatically brain injured patients in the acute inpatient rehabilitation setting. *Brain Injury*, 14(4), 333–344.
- Prigatano, G. P., Altman, I. M., & O'Brien, K. P. (1990). Behavioral limitations that traumatic-braininjured patients tend to underestimate. *Clinical Neuropsychology*, 4, 163–176.
- Godfrey, H. P., Partridge, F. M., Knight, R. G., & Bishara, S. (1993). Course of insight disorder and emotional dysfunction following closed head injury: A controlled cross-sectional follow-up study. *Journal* of Clinical and Experimental Neuropsychology, 15, 503–515.
- Hart, T., Seignourel, P. J., & Sherer, M. (2009). A longitudinal study of awareness after moderate-tosevere traumatic brain injury. *Neuropsychological Rehabilitation*, 19, 161–176.
- Johnson, S. C., Baxter, L. C., Wilder, L. S., Pipe, J. G., Heiserman, J. E., & Prigatano, G. P. (2002). Neural correlates of self-reflection. *Brain*, 125, 1808–1814.
- Newsome, M. R., Scheibel, R. S., Hanten, G., Chu, Z., Steinberg, J. L., Hunter, J. V., et al. (2010). Brain activation while thinking about the self from another person's perspective after traumatic brain injury in adolescents. *Neuropsychology*, 24, 139–147.
- Gennarelli, T. A., & Graham, D. I. (2005). Neuropathology. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.), *Textbook of traumatic brain injury* (pp. 27–50). Washington, DC: American Psychiatric Publishing.
- Sherer, M., Hart, T., Whyte, J., Nick, T. G., & Yablon, S. A. (2005). Neuroanatomic basis of impaired selfawareness after traumatic brain injury: Findings from early computed tomography. *The Journal of Head Trauma Rehabilitation*, 20(4), 287–300.
- Anderson, S. W., & Tranel, D. (1989). Awareness of disease states following cerebral infarction, demen-

tia, and head trauma: Standardized assessment. *Clinical Neuropsychology*, *3*(4), 327–339.

- 14. Bivona, U., Ciurli, P., Barba, C., Onder, G., Azicnuda, E., Silvestro, D., et al. (2008). Executive function and metacognitive self-awareness after severe traumatic brain injury. *Journal of International Neuropsychological Society*, 14(5), 862–868.
- Spikman, J. M., Timmerman, M. E., Milders, M. V., Veenstra, W. S., & van der Naalt, J. (2012). Social cognition impairments in relation to general cognitive deficits, injury severity, and prefrontal lesions in traumatic brain injury patients. *Journal of Neurotrauma*, 29, 101–111.
- Levin, H. S., Wilde, E. A., Hanten, G., Li, X., Chu, Z. D., Vásquez, A. C., et al. (2011). Mental state attributions and diffusion tensor imaging after traumatic brain injury in children. *Developmental Neuropsychology*, 36(3), 273–287.
- Martin, I., & McDonald, S. (2003). Weak coherence, no theory of mind, or executive dysfunction? Solving the puzzle of pragmatic language disorders. *Brain Language*, 85, 451–466.
- Bach, L. J., & David, A. S. (2006). Self-awareness after acquired and traumatic brain injury. *Neuropsychological Rehabilitation*, 16, 397–414.
- Lam, C. S., McMahon, B. T., Priddy, D. A., & Gehred-Schultz, A. (1988). Deficit awareness and treatment performance among traumatic head injury adults. *Brain Injury*, 2(3), 235–242.
- Malec, J. F., & Degiorgio, L. (2002). Characteristics of successful and unsuccessful completers of 3 postacute brain injury rehabilitation pathways. *Archives of Physical Medicine and Rehabilitation*, 83, 1759–1764.
- 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. *The Journal of Head Trauma Rehabilitation*, 17(2), 155–174.
- Sander, A. M., Caroselli, J. S., High, W. M., Jr., Becker, C., Neese, L., & Scheibel, R. (2002). Relationship of family functioning to progress in a post-acute rehabilitation program following traumatic brain injury. *Brain Injury*, 16(8), 649–657.
- Sherer, M., Hart, T., & Nick, T. G. (2003). Measurement of impaired self-awareness after traumatic brain injury: A comparison of the patient competency rating scale and the awareness questionnaire. *Brain Injury*, 17, 25–37.
- 24. Sherer, M., Bergloff, P., Levin, E., High, W. M., Oden, K. E., & Nick, T. G. (1998). Impaired awareness and employment outcome after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, *13*, 52–61.
- 25. Prigatano, G. P., & Schacter, D. L. (1991). Introduction. In G. P. Prigatano & D. L. Schacter (Eds.), Awareness of deficit after brain injury: Clinical and theoretical issues (pp. 3–15). New York: Oxford University Press.
- Katz, N., & Maeir, A. (2011). Higher level cognitive functions enabling participation: Awareness and

executive functions. In N. Katz (Ed.), *Cognition, occupation and participation across the life span* (3rd ed., pp. 13–40). Bethesda, MD: American Occupational Therapy Association Press.

- Simmond, M., & Fleming, J. (2003). Occupational therapy assessment of self-awareness following traumatic brain injury: A literature review. *British Journal of Occupational Therapy*, 66, 447–453.
- Morton, N., & Barker, L. (2010). The contribution of injury severity, executive and implicit functions to awareness deficits after traumatic brain injury. *Journal of International Neuropsychological Society*, 16, 1089–1098.
- Sherer, M., Oden, K., Bergloff, P., Levin, E., & High, W. M. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *Neurorehabilitation*, 10, 25–37.
- 30. Prigatano, G. P., Fordyce, D. J., Zeiner, H. K., Roueche, J., Pepping, M., & Wood, B. (1986). *Neuropsychological rehabilitation after brain injury*. Baltimore: John Hopkins University Press.
- Fordyce, D. J., & Roueche, J. R. (1986). Changes in perspectives of disability among patients, staff and relatives during rehabilitation of brain injury. *Rehabilitation Psychology*, 31, 17–229.
- Prigatano, G. P., & Altman, I. M. (1990). Impaired awareness of behavioral limitations after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 71, 1058–1064.
- Wallace, C. A., & Bogner, J. (2000). Awareness of deficits: Emotional implications for persons with brain injury and their significant others. *Brain Injury*, 14, 422–428.
- Borgaro, S. R., & Prigatano, G. P. (2003). Modification of the Patient Competency Rating Scale for use on an acute neurorehabilitation unit: The PCRS-NR. *Brain Injury*, *17*, 847–853.
- 35. Sherer, M., Bergloff, P., Boake, C., High, W. M., & Levin, E. (1998). The Awareness Questionnaire: Factor analysis structure and internal consistency. *Brain Injury*, 12, 63–68.
- 36. Garmoe, W., Newman, A., & O'Connell, M. (2005). Comparison of brain injury and orthopedic inpatients using the Functional Self-Assessment Scale (FSAS). *The Journal of Head Trauma Rehabilitation*, 20, 348–358.
- Wilson, B. A., Alderman, N., Burgess, P. W., Emslie, H., & Evans, J. J. (1996). *Behavioural assessment of the dysexecutive syndrome*. Bury St. Edmunds, England: Thames Valley Test Company.
- Malec, J. F., Buffington, A. L. H., Moessner, A. M., & Degiorgiom, L. (2000). A medical/vocational case coordination system for persons with brain injury: An evaluation of employment outcomes. *Archives of Physical Medicine and Rehabilitation*, 81, 1007–1015.
- 39. Pagulayan, K. F., Temkin, M. R., Machamer, J. E., & Dikmen, S. S. (2007). Measurement and magnitude of awareness difficulties after traumatic brain injury: A longitudinal study. *Journal of International Neuropsychological Society*, 13, 561–570.

- Livengood, M., Anderson, J. W., & Schmitter-Edgecombe, M. (2010). Assessment of memory selfawareness following traumatic brain injury. *Brain Injury*, 24, 598–608.
- Roche, N. L., Fleming, J. M., & Shum, D. H. K. (2002). Self-awareness of prospective memory failure in adults with traumatic brain injury. *Brain Injury*, 16, 931–945.
- Fleming, J. M., Strong, J., & Ashton, R. (1996). Selfawareness of deficits in adults with traumatic brain injury. How best to measure? *Brain Injury*, 10, 1–15.
- Ownsworth, T., Clare, L., & Morris, R. (2006). An integrated biopsychosocial approach to understanding awareness deficits in Alzheimer's disease and brain injury. *Neuropsychological Rehabilitation*, 16, 415–438.
- Allen, C. C., & Ruff, R. M. (1990). Self rating versus neuropsychosocial performance of moderate versus severe head-injured patients. *Brain Injury*, 4, 7–17.
- Anderson, R. A., Doble, S. E., Merritt, B. E., & Kottorp, A. (2010). Assessment of awareness of disability measures among persons with acquired brain injury. *Canadian Journal of Occupational Therapy*, 77, 22–29.
- 46. Hart, T., Whyte, J., Polansky, M., Millis, S., Hammond, F. M., Sherer, M., et al. (2003). Concordance of patient and family report of neurobehavioral symptoms at 1 year after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84, 204–213.
- Ownsworth, T. L., McFarland, K., & Young, R. M. (2000). Development and standardization of the selfregulation skills interview (SRSI): A new clinical assessment tool for acquired brain injury. *Clinical Neuropsychologist*, 14, 76–92.
- Markova, I. S., & Berrios, G. E. (1992). The assessment of insight in clinical psychiatry: A new scale. *Acta Psychiatrica Scandinavica*, 86, 159–164.
- Levine, M. J., Van Horn, K. R., & Curtis, A. B. (1993). Developmental models of social cognition in assessing psychosocial adjustments in head injury. *Brain Injury*, 7, 153–167.
- Bisiach, E., Vallar, G., Perani, D., Papagno, C., & Berti, A. (1986). Unawareness of disease following lesions of the right hemisphere. Anosognosia for hemianopia. *Neuropsychologia*, 24, 471–482.
- Barco, P. P., Crosson, B., Bolesta, M. M., Werts, D., & Stout, R. (1991). Training awareness and compensation in postacute head injury rehabilitation. In J. S. Kreutzer & P. H. Wehman (Eds.), *Cognitive rehabilitation for persons with traumatic brain injury. A functional approach* (pp. 129–146). Baltimore: Paul H. Brookes.
- Bergquist, T. F., & Jacket, M. P. (1993). Programme methodology: Awareness and goal setting with the traumatically brain injured. *Brain Injury*, 7, 275–282.
- 53. Fleming, J. M., Strong, J., & Ashton, R. (1998). Cluster analysis of self-awareness levels in adults with traumatic brain injury and relationship to outcome. *The Journal of Head Trauma Rehabilitation*, 13(5), 39–51.

- Simmond, M., & Fleming, J. (2003). Reliability of the self-awareness of deficits interview for adults with traumatic brain injury. *Brain Injury*, 17, 325–337.
- Bogod, N. M., Mateer, C. A., & MacDonald, S. W. S. (2003). Self-awareness after traumatic brain injury: A comparison of measures and their relationship to executive functions. *Journal of International Neuropsychological Society*, 9, 450–458.
- Wise, K., Ownsworth, T., & Fleming, J. (2005). Convergent validity of self-awareness measures and their association with employment outcomes in adults following acquired brain injury. *Brain Injury*, 19, 765–775.
- Prigatano, G. P., & Klonoff, P. S. (1998). A clinician's rating scale for evaluating impaired selfawareness and denial of disability after brain injury. *Clinical Neuropsychologist*, 12, 56–67.
- Hart, T., Giovanetti, T., Montgomery, M. W., & Schwartz, M. F. (1998). Awareness of errors in naturalistic action after traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 13, 16–26.
- Ownsworth, T. L., Fleming, J., Desbois, J., Strong, J., & Kuipers, P. (2006). A metacognitive contextual intervention to enhance error awareness and functional outcome following traumatic brain injury: A single case experimental design. *Journal of International Neuropsychological Society*, *12*, 54–63.
- Ownsworth, T., Quinn, H., Fleming, J., Kendall, M., & Shum, D. (2010). Error self-regulation following traumatic brain injury: A single case study evaluation of metacognitive skills training and behavioural practice. *Neuropsychological Rehabilitation*, 20, 51–80.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., et al. (2011). Evidence-base cognitive rehabilitation: Updated review of literature from 2003-2008. Archives of Physical Medicine and Rehabilitation, 92, 519–530.
- 62. Sherer, M. (2005). Rehabilitation of awareness. In W. M. High Jr., A. M. Sander, M. A. Struchen, & K. A. Hart (Eds.), *Rehabilitation interventions following traumatic brain injury: State of the science* (pp. 31–46). New York: Oxford University Press.
- Fleming, J., & Ownsworth, T. (2006). A review of awareness interventions in brain injury rehabilitation. *Neuropsychological Rehabilitation*, 16, 474–500.
- Lucas, S. E., & Fleming, J. M. (2005). Interventions for improving self-awareness following acquired brain injury. *Australian Occupational Therapy Journal*, 52, 160–170.
- 65. DeHope, E., & Finegan, J. (1999). The selfdetermination model: An approach to develop awareness for survivors of traumatic brain injury. *NeuroRehabilitation*, 13, 3–12.
- Schmidt, J., Lannin, N., Fleming, J., & Ownsworth, T. (2011). Feedback interventions for impaired selfawareness following brain injury: A systematic review. *Journal of Rehabilitation Medicine*, 43, 673–680.
- Cheng, S. K. W., & Man, D. W. K. (2006). Management of impaired self-awareness in persons with traumatic brain injury. *Brain Injury*, 20, 621–628.

- Goverover, Y., Johnston, M. V., Toglia, J., & DeLuca, J. (2007). Treatment to improve self-awareness in persons with acquired brain injury. *Brain Injury*, 21, 913–923.
- 69. Ownsworth, 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, 81–88.
- Klonoff, P. S., O'Brien, K. P., Prigatano, G. P., Chiapello, D. A., & Cunningham, M. (1989). Cognitive retraining after traumatic brain injury and its role in facilitating awareness. *The Journal of Head Trauma Rehabilitation*, 4, 37–45.
- Toglia, J., & Kirk, U. (2000). Understanding awareness deficits following brain injury. *NeuroRehabilitation*, 15, 57–70.
- Bieman-Copland, S., & Dywan, J. (2000). Achieving rehabilitative gains in anosognosia after TBI. *Brain Cognition*, 1, 1–18.
- Coetzer, B. R., & Corney, M. J. R. (2001). Grief and self-awareness following brain injury and effect of feedback as an intervention. *Journal of Cognitive Rehabilitation*, 19, 8–14.
- Roberts, C. B., Fafal, R., & Coetzer, B. R. (2006). Feedback of brain-imaging findings: Effect on impaired awareness and mood in acquired brain injury. *Brain Injury*, 20, 485–497.
- Alexy, W. D., Foster, M., & Baker, A. (1983). Audiovisual feedback: An exercise in self-awareness for the head injured patient. *Cognitive Rehabilitation*, *1*, 8–10.
- 76. Toglia, J. P. (2011). The dynamic interactional model of cognition in cognitive rehabilitation. In N. Katz (Ed.), *Cognition, occupation and participation* across the life span (3rd ed., pp. 161–201). Bethesda, MD: American Occupational Therapy Association Press.
- Fleming, J. M., Lucas, S. E., & Lightbody, S. (2006). Using occupation to facilitate self-awareness in people who have acquired brain injury. *Canadian Journal of Occupational Therapy*, *12*, 54–63.
- Schmidt, J., Fleming, J., Ownsworth, T., & Lannin, N. (2013). Videotape feedback on functional task performance improves self-awareness after traumatic brain injury: A randomised controlled trial. *Journal of Neurorehabilitation and Neural Repair*, 27, 316–324.
- McGraw-Hunter, M., Faw, G. D., & Davis, P. K. (2006). The use of video self-modelling and feedback to teach cooking skills to individuals with traumatic brain injury: A pilot study. *Brain Injury*, 20, 1061–1068.
- Rebmann, M. J., & Hannon, R. (1995). Treatment of unawareness of memory deficits in adults with acquired brain injury: Three case studies. *Rehabilitation Psychology*, 40, 279–287.

- Schlund, M. W. (1999). Self-awareness: Effects of feedback and review on verbal self reports and remembering following brain injury. *Brain Injury*, *13*, 375–380.
- Youngjohn, J. R., & Altman, I. M. (1989). A performance-based group approach to the treatment of anosognosia and denial. *Rehabilitation Psychology*, 34, 217–222.
- Langer, K. G., & Padrone, F. J. (1992). Psychotherapeutic treatment of awareness in acute rehabilitation of traumatic brain injury. *Neuropsychological Rehabilitation*, 2, 59–70.
- Dirette, D. K., Plaisier, B. R., & Jones, S. J. (2008). Patterns and antecedents of the development of selfawareness following traumatic brain injury: The importance of occupation. *British Journal of Occupational Therapy*, *71*, 44–51.
- Landa-Gonzalez, B. (2001). Multicontextual occupational therapy intervention: A case study of traumatic brain injury. *Occupational Therapy International*, 8, 49–62.
- Ownsworth, T. (2010). A metacognitive contextual approach for facilitating return to work following acquired brain injury: Three descriptive case studies. *Work*, 36, 381–388.
- Ben-Yishay, Y., Silver, S. M., Piasetsky, E., & Rattok, J. (1987). Relationship between employability and vocational outcome after intensive holistic cognitive rehabilitation. *The Journal of Head Trauma Rehabilitation*, 2, 35–48.
- Malec, J. F., & Moessner, A. M. (2000). Selfawareness, distress, and postacute rehabilitation outcome. *Rehabilitation Psychology*, 45, 227–241.
- 89. Prigatano, G. P. (1986). Psychotherapy after brain injury. In G. P. Prigatano, D. J. Fordyce, H. K. Zeiner, J. R. Roueche, M. Pepping, & B. C. Wood (Eds.), *Neuropsychological rehabilitation after brain injury* (pp. 67–95). Baltimore: John Hopkins University Press.
- Ownsworth, T. L., McFarland, K., & Young, R. M. (2000). Self-awareness and psychosocial functioning following acquired brain injury: An evaluation of a group support programme. *Neuropsychological Rehabilitation*, 10, 465–484.
- Ownsworth, T. (2000). The impact of defensive denial upon adjustment following traumatic brain injury. *Neuropsychoanalysis*, 7, 83–94.

- 92. Medley, A. R., & Powell, T. (2010). Motivational interviewing to promote self-awareness and engagement in rehabilitation following acquired brain injury: A conceptual review. *Neuropsychological Rehabilitation*, 20, 481–508.
- Beardmore, S., Tate, R., & Liddle, B. (1999). Does information and feedback improve children's knowledge ad awareness of deficits after traumatic brain injury? *Neuropsychological Rehabilitation*, 9, 45–62.
- 94. Chittum, W. R., Johnson, K., Chittum, J. M., Guercio, J. M., & McMorrow, M. J. (1996). Road to awareness: An individualised training package for increasing knowledge and comprehension of personal deficits in persons with acquired brain injury. *Brain Injury*, 10, 763–776.
- 95. Zhou, J., Chittum, R., Johnson, K., Poppen, R., Guercio, J., & McMorrow, M. J. (1996). The utilization of a game format to increase knowledge of residuals among people with acquired brain injury. *The Journal of Head Trauma Rehabilitation*, 11, 51–61.
- Bordin, E. S. (1979). The generalizability of the psychoanalytic concept of the working alliance. *Psychotherapy Theory Research and Practice*, 16, 252–260.
- Schonberger, M., Humle, F., & Teasdale, T. W. (2006). The development of the therapeutic working alliance, patients' awareness and their compliance during the process of brain injury rehabilitation. *Brain Injury*, 20(4), 445–454.
- Kuipers, K., Rassafiani, M., Ashburner, J., Griffin, J., Worley, L., Moes, L., et al. (2009). Do clients with acquired brain injury use the splints prescribed by occupational therapists? A descriptive study. *NeuroRehabilitation*, 24, 365–375.
- 99. Ochsner, K. N., Knierim, K., Ludlow, D. H., Hanelin, J., Ramachandran, T., Glover, G., et al. (2004). Reflecting upon feelings: An fMRI study of neural systems supporting the attribution of emotion to self and other. *Journal of Cognitive Neuroscience*, 16(10), 1746–1772.
- 100. Ben-Yishay, Y., Rattock, J., Lakin, P., Piasetsky, E., Ross, B., Silver, S. L., et al. (1985). Neuropsychologic rehabilitation: Quest for a holistic approach. *Seminars in Neurology*, 5(3), 252–259.
- Prigatano, G. P. (1999). Principles of neuropsychological rehabilitation. New York: Oxford University Press.

# Emotional Distress Following Traumatic Brain Injury

Allison N. Clark

# Abstract

A traumatic brain injury (TBI) is a significant life event that can have serious and long-lasting impacts on neurobehavioral and psychosocial functioning, and may cause disruptions in major life areas. High levels of emotional distress are frequently experienced by persons who have sustained a TBI, and are associated with poorer functional outcomes following injury. Effective assessment and treatment of emotional distress and the "wounded soul" (J Head Trauma Rehabil 1991, 6(4):1–10) in persons with TBI are well-recognized as important components of rehabilitation following TBI. This chapter will focus on the most common types of emotional distress experienced by persons who have sustained a TBI: depression and anxiety. Factors associated with the experience of emotional distress following injury, assessment considerations, and interventions for emotional distress will be discussed. Finally, two case studies that high-light issues that may impact the psychological treatment of emotional distress in persons with TBI will be presented.

#### Keywords

Traumatic brain injury • Emotional distress • Depression • Anxiety

# Depression

Depression is the most common mood disorder experienced by persons who have sustained a traumatic brain injury (TBI). A recent systematic

A.N. Clark, Ph.D. (⊠) Baylor College of Medicine, Houston, TX, USA

Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA e-mail: Allison.Clark@memorialhermann.org review found that the prevalence of depression in persons with TBI, diagnosed with structured clinical interviews, was approximately 30 % across varying time-points post-injury [1]. In a prospective cohort study of patients hospitalized for TBI, 53.1 % of the sample met criteria for a diagnosis of depression at some point during the first year following injury [2]. A recent longitudinal study of persons with TBI who received inpatient rehabilitation found that three-quarters of those who were depressed at 1 year post-injury experienced clinically significant depressive symptoms at 2 years post-injury [3]. Reported rates vary widely, likely reflecting differences across study samples and in how depression is assessed (e.g., 11.9–64 % within the first year of injury [2, 4–6]), yet the reported prevalence rates of depression in persons with TBI are often markedly higher than the 7 % rate reported for the general population [7]. These findings underscore the large number of persons affected by depression following TBI.

Persons with depression following TBI experience poorer functional outcomes compared to persons without depression following injury, even after controlling for the severity of injury [5]. Post-TBI depression has been associated with decreased life satisfaction and perceived quality of life [2, 8, 9], disruptions in social and family functioning [10–12], and vocational difficulties [11, 13, 14]. A recent study by Hart et al. [5] described a monotonic dose-response relationship between severity of depression and measures of disability, societal participation, and satisfaction with life. Increasing severity of depression was associated with increasing functional deficits in this sample. Rosenthal, Christensen and Ross [15] described depression in persons with TBI as "a barrier to the achievement of optimal rehabilitation goals in the successful reintegration of the patient into the home, family, community, and work environment."

# Factors Associated with Depression Post-TBI

Several factors are associated with an increased risk of depression following injury, including younger age [2, 5], lower levels of education [2, 16], a premorbid history of psychiatric disorder [2, 17], premorbid substance abuse [2, 5, 16], depression at the time of injury [2], and poorer pre-injury social [18] and vocational functioning [16]. Findings regarding sex are mixed, with some studies showing that women are at greater risk for depression following injury [5] while others have found higher rates of depression among men [16].

There is consistent evidence that the severity of the injury, which is often defined according to Glasgow Coma Scale scores or duration of posttraumatic amnesia, is not related to depression following injury [2, 5, 13, 16, 19]. Yet other injury-related factors may contribute to depression following TBI. Major depression following TBI has been associated with reductions in hippocampal [20] and left prefrontal gray matter volumes, especially in ventrolateral and dorsolateral regions [17], as well as hypometabolism of the lateral and dorsal frontal cortex and cingulate gyrus, and increased activation in ventral limbic and paralimbic structures [21]. Disruptions to major neurotransmitter systems, including serotinergic, glutaminergic, cholinergic, and dopaminergic systems, and neuroendocrine abnormalities have also been hypothesized to contribute to depression following TBI [21]. A particular lesion location is not considered necessary or sufficient for the development of depression following TBI [21], but biological factors may play a greater role in early onset compared to late-onset post-TBI depression [18, 22]. The development and experience of depression following TBI is likely influenced by biological, psychological, and social factors [21].

The relationship between depression and functional outcomes is complicated, since depression may contribute to poorer functioning via reduced motivation or compliance with rehabilitation or may reflect the emotional response to changes in functioning and participation following injury. Studies that investigated the temporal relationship between depression and functional status suggest that the experience of functional limitations precedes the development of later depression [23, 24]. Pagulayan et al. [23] examined health-related quality of life and depression at 1, 6, and 12 months post-injury in 135 adolescents and adults with complicated mild to severe TBI, and found that early report of health-related impairments was associated with depression at 1 year post-injury. In contrast, there was no significant relationship between early depression and health-related impairments at 1 year post-injury. Schönberger et al. [24] investigated the temporal relationship between depression and functional status in 122 adults with mild to severe TBI, and found that poor functional status at 6 months post-injury predicted depression at 12 months post-injury. In a consecutive sample of 96 patients with mild to severe TBI, lack of improvement in perceived functioning following hospital discharge was associated with depression at 3 months post-discharge, controlling for age and depressive symptoms at the time of discharge [25]. In a related study, the persistence of disability and the development of disability following initial recovery were associated with depression and decreased feelings of self-worth in a sample of 334 persons 5-7 years following mild, moderate, or severe TBI [19]. These findings suggest that the experience of functional limitations and disability appears to play an important role in depression following TBI.

#### **Depressive Symptoms**

The assessment and diagnosis of depression following TBI can be challenging because some of the symptoms of depression, such as poor concentration, decreased energy, and sleep disturbances, may be attributed to the TBI. Concerns regarding inflated scores on self-report measures and inaccurate diagnosis are understandable given these overlapping symptoms. Yet depressive symptoms experienced by persons with TBI are generally consistent with depressive symptoms reported by the general population [13, 26,27]. Cook et al. [26] investigated responses to the Patient Health Questionnaire 9 (PHQ-9) in a sample of primary care patients versus a sample of patients with TBI to determine if PHQ-9 scores were inflated among patients with TBI and to examine the validity of PHQ-9 items, especially those that assess transdiagnostic symptoms, to assess depression in persons with TBI. They found that all PHQ-9 items were loaded on a single depression factor in both the primary care and TBI samples. There was no differential item functioning that could be attributed to TBI, and no systematic inflation of scores in persons with TBI. They concluded that these findings fail to support the belief that some symptoms of depression are not valid indicators of depression among persons with TBI.

However, depressive symptoms may manifest differently among persons with TBI. Seel, Macciocchi, and Kreutzer [28] have described how empirically based symptom manifestations in persons with TBI may present in relation to corresponding DSM-IV criteria for a major depressive episode. For example, the symptom of depressed mood may appear as irritability, frustration, anger, or aggression. Lack of confidence, discomfort around others, and social withdrawal may indicate feelings of worthlessness. Regarding the symptom of diminished thinking ability, selfreported attention and memory problems are often greater than objective findings [28]. Symptoms that have been found to differentiate depressed from non-depressed persons with TBI include rumination, self-criticism, distress, guilt, depressed mood, lack of energy, feelings of worthlessness, and suicidal ideation [27, 29]. Although discussion of aggressive behaviors following TBI is beyond the scope of this chapter, it is important to note that among persons with aggression following TBI, depression is often present [17, 30].

#### **Depression and Cognition**

There is some evidence that depression impacts cognition, especially executive functioning, following TBI [17, 31, 32]. Schiehser et al. [32] investigated neuropsychological functioning, depression, and symptom-complaints in a sample of 71 noncombatant military personnel with mild to moderate TBI. They found that depression, but not injury severity, was associated with decreased scores on measures of executive functioning and memory. In a study by Jorge et al. [17], 91 patients with mild, moderate, and severe traumatic brain injury underwent comprehensive psychiatric and neuropsychological evaluations at 3 months post-injury. Patients with and without major depression did not differ on measures of injury severity. However, patients with major depression performed worse on two of three measures of executive functioning when compared to patients without major depression; no differences were observed on measures of language and memory. In a sample of 74 patients with mild to moderate TBI approximately 6 months postinjury, patients with major depression performed worse on measures of working memory, processing speed, and verbal memory, and showed greater perseveration on the Wisconsin Card Sorting Task [33]. In contrast, in a separate sample of 100 patients with moderate to severe TBI, there was no relationship between depression and neuropsychological functioning at 6 months post-injury [34]. In a related study, Fann et al. [35] reported improvements in psychomotor speed, cognitive efficiency, verbal memory, and flexible thinking following an 8-week treatment trial of sertraline for depression in a sample of 15 persons with mild TBI.

# Anxiety

The literature on anxiety following TBI, though smaller compared to that on post-TBI depression, indicates that anxiety is commonly experienced in persons with TBI [36-41]. The reported rates for anxiety disorders, like those for depression, vary due to methodological and sample differences but are generally higher than the rates reported for the general population [7]. Generalized anxiety disorder (GAD) and posttraumatic stress disorder (PTSD) are the most frequently diagnosed anxiety disorders in persons who have sustained a TBI. Gould et al. [40] investigated the development of psychiatric disorders using a structured clinical interview in a prospective sample of 102 persons with predominantly moderate to severe TBI, and found that 44 % met criteria for an anxiety disorder during the first 12 months following injury. The most common anxiety disorders diagnosed were anxiety disorder not otherwise specified (35 %) and PTSD (12.7 %). Whelan-Goodinson used a computerized structured clinical interview to study psychiatric disorders in a sample of 100 persons with medically documented mild to severe TBI who were between 6 months and 51/2 years postinjury and found that 38 % experienced at least one anxiety disorder [42]. The most common disorders were GAD (17 %) and PTSD [14 %]. Ashman et al. [36] investigated rates of Axis I disorders in 188 persons with self-identified TBI between 3 months and 4 years post-injury. The rate of PTSD was 38 % among those at 1 year post-injury, 18 % for those at 2 years post-injury, and 33–34 % among those 3–4 years post-injury. The combined rate of other anxiety disorders was 30 % among those at 1 year post-injury, 27 % for those at 2 years post-injury, 21 % at 3 years postinjury, and 26 % at 4 years post-injury. Anxiety, like depression, is also associated with poorer functional outcomes, including occupational activities and interpersonal relationships [43].

There is a high level of comorbidity between mood and anxiety disorders in persons who have sustained a TBI. Prevalence rates for the presence of a comorbid anxiety disorder among persons with depression following TBI range from 41 to 76.7 % [2, 17, 44]. Anxiety is more common in depressed persons with TBI than in nondepressed persons with TBI [2]. The most consistent factor associated with anxiety following TBI is the preinjury history of anxiety disorder [36, 40, 45]. However, some studies have also reported that older age [45], unemployment [45], and being female [36] were associated with anxiety following TBI. The current evidence suggests that injury severity is not associated with anxiety following injury [40, 45], which is consistent with the findings regarding depression and injury severity. Memory for the traumatic event has been associated with the development of PTSD in a prospective study of 120 persons with mild TBI [46]. Fourteen percent of the overall sample met criteria for PTSD at 6 months post-injury, and persons with memory of the traumatic event were at higher risk for developing PTSD compared to persons with no memory for the event. Memory of the traumatic event, acute posttraumatic symptoms, acute symptoms of depression and anxiety, and history of psychiatric disorder were associated with increased risk of PTSD at 6 months post-injury in this sample.

Much of the recent literature on PTSD and TBI has focused on mild TBI in military samples. The relationship between PTSD and mild TBI is complicated due to the overlap of symptoms and their association with postconcussive symptoms. A discussion of postconcussion syndrome is beyond the scope of this chapter. Yet it is important to note that reports of postconcussion symptoms and post-traumatic stress are highly interrelated in military samples. For example, Hoge et al. [47] reported that soldiers with injuryrelated loss of consciousness (LOC) had higher rates of physical and post-concussive symptoms compared to soldiers with other injuries as a result of injury; however, these relationships, with the exception of headache, were not significant after adjusting for PTSD and depression. Other studies using military samples have also found that postconcussive symptom reporting following mild TBI is related to PTSD [48].

# Suicide

A number of population-based and clinical studies have documented an increased risk of death from suicide among persons who have sustained a TBI [49–51]. Persons with TBI are at three to four times higher risk for committing suicide compared to the general population [52]. Elevated rates of suicide attempts [53] and suicidal ideation [54] have also been reported among persons with TBI. Persons who sustain a TBI as a result of a suicide attempt had more pre-injury psychiatric and psychosocial problems, including substance abuse, previous suicide attempt, and previous psychiatric hospitalization compared to persons with unintentional injuries [55].

Increased risk of suicide in persons with TBI has been associated with several factors including concurrent psychiatric disorders, substance abuse, and self-inflicted mechanism of injury [49]. Persons with ICD codes indicative of more severe injuries (cerebral contusion, intracerebral hemorrhage) showed an increased risk of suicide compared to persons with ICD codes suggestive of less severe injuries (concussion, cranial fractures) [49]. The findings regarding sex are mixed, with some studies reporting no sex differences with respect to suicide attempt or suicidal ideation [52, 54], while others have reported an increased risk of death by

suicide among females [49]. Persons with TBI with a post-injury history of emotional or psychiatric disorders, substance dependence, or both are more likely to attempt suicide compared to those with no significant psychiatric or substance abuse history [56]. Some neurobehavioral consequences of TBI, such as aggression, depression, and impulsivity, and concomitant disturbances in family and occupational functioning, are also general suicide risk factors and may contribute to increased risk following injury [57].

Tsaousides et al. [54] investigated suicidal ideation (SI), defined as thoughts of dying or suicide with or without plan, in a sample of 356 adults who had experienced TBI. Approximately 28 % of this sample endorsed SI at least once during a 5-year follow-up period. SI was associated with pre-injury substance abuse, post-injury anxiety disorders, and concurrent depressive and anxiety disorders, but was not related to demographic characteristics, injury severity, time postinjury, or pre-injury psychiatric history. SI was also associated with lower perceptions of wellbeing, quality of life, and perceived social support. The latter is consistent with research with veterans that suggests that social support may serve as a protective buffer against SI [58].

Attention to warning signs as well as knowledge of risk factors is important in the assessment of suicide risk in persons with TBI. Simpson and Tate [56, 59] conducted interviews with 43 persons with TBI who had attempted suicide to identify antecedent circumstances to the attempt. Results of these interviews revealed that warning signs and circumstances that preceded the attempt included feelings of depression and hopelessness (e.g., "sick of being the way that I was"; "wish my life had ended at the accident"), relationship conflicts and breakdowns ("my wife decided to separate and took the children"), instrumental difficulties, social isolation, an intolerable accumulation of stress, and nonspecific references regarding perception of injury (e.g., "nothing can be done to treat the brain injury"). They also found that 48.3 % of those who made an attempt post-injury made at least one other suicide attempt, and that repeated events were often done over a circumscribed time period. Simpson and Tate [56] argue that persons with TBI who have attempted suicide should be closely monitored for at least 1 year following their first attempt.

There is no evidence for a critical post-injury time period for increased risk of suicide [54, 60]. Rather, this risk appears to persist over time [54, 60]. Given the persistence of elevated risk, Wasserman et al. [61] propose that clinicians utilize a low threshold for screening for suicide among persons with TBI, especially for those with mood disorders and substance abuse, and include inquiries regarding suicidal ideation, behavior, and intent in clinical interviews [28].

There is no significant evidence regarding the validity of screening instruments specifically for suicide in persons with TBI, or for the effectiveness of specific treatments for suicidality in persons with TBI. Therefore, adherence to current general practice guidelines is recommended [57, 60]. Simpson and Tate [60] have described how the Institute of Medicine's three-tiered model of suicide prevention strategies (universal, selected, indicated) can be applied to the TBI population. These prevention strategies are presented in Table 1.

# Assessment Considerations

The structured clinical interview remains the gold standard for diagnosing depressive and anxiety disorders in persons with TBI. However, self-report measures are often used to screen for emotional distress and to monitor symptoms over time. Self-report measures are frequently used clinically and in empirical investigations of emotional distress. There are several measures with demonstrated validity as screening measures for depression and anxiety following TBI [28, 62]. These measures include: the Beck Depression Inventory-II [63], the Center for Epidemiological Studies-Depression Scale [64], the Patient Health Questionnaire-9 [65], Neurobehavioral Functioning Inventory-Depression Scale [66], the Depression Anxiety Stress Scales [67, 68], and the Hospital Anxiety and Depression Scale [69].

Self-report measures are best used for ruling out the presence of an emotional disorder [28].

 Table 1
 Suicide prevention in persons with TBI

Level of intervention			Clinical management		
Universal		•	Promote positive mental health		
•	All persons with TBI	•	Assess hopelessness and SI proactively		
		•	Recognize that persons may be at risk at various times post-injury		
		•	Promote long-term supports		
		•	Monitor males and females equally		
Selected		•	Provide treatment for emotional disorders and substance abuse		
•	Persons with TBI at risk for suicide	•	Monitor persons with comorbid conditions and persons who sustained TBI as result of suicide attempt		
Indicated		•	Reduce lethality of environment		
•	Persons with TBI for whom suicide is an identified	•	Provide treatment for emotional disorders and substance abuse		
	issue	•	For persons with past suicide attempt, be aware that persons may use more than one method		
		•	Conduct full clinical interview, including structured risk assessment		
		•	Monitor for at least 12 months following a suicide attempt		
		•	Provide emergency contact card with crisis numbers		

Adapted from Simpson and Tate

Cook et al. [26] suggest that clinicians utilize an "inclusive" approach to the diagnosis of depression following TBI, where all symptoms are counted toward the diagnosis regardless of possible cause (e.g., TBI or depression), and that clinicians not minimize reported cognitive or somatic symptoms when diagnosing depression in this population. Clinicians should use structured clinical interviews following a positive screen to confirm a suspected diagnosis in persons with TBI. The structured clinical interview is also indicated for the differential diagnosis of patients with TBI who present with complex symptoms and with symptoms that can be attributed to multiple disorders [28]. In cases where clinicians have concerns regarding the impact of impaired awareness on the validity of a patient's responses, use of specific and concrete questions during the clinical interview is recommended [28].

#### Interventions

Patients with depression and/or anxiety may receive pharmacological and/or psychological treatment for their emotional distress. Unfortunately, the literature suggests that there are large numbers of persons with clinically significant depression and anxiety who are not receiving treatment [2, 42]. There is insufficient evidence regarding the efficacy of a specific class of medications for the treatment of depression or anxiety following TBI [70, 71]. There is not sufficient evidence to support practice recommendations regarding specific psychological treatments for depression or anxiety [70, 71] of persons with TBI. However, there is a growing body of literature investigating the use of psychological interventions validated in the general population, especially cognitive-behavioral therapy, to treat emotional distress in persons with TBI.

Cognitive-behavioral approaches to the treatment of emotional distress have demonstrated effectiveness in many populations, and appear ideally suited for the TBI population because they offer inherent structure and focus. This structure and content can be adapted for use with persons with cognitive deficits [72]. In fact, the majority of psychological interventions for emotional distress following TBI described in the extant literature are cognitive-behavioral interventions that have been modified for use with persons with cognitive deficits following injury [73–76] or include cognitive-behavioral components [77]. Results of these studies suggest that the use of adapted cognitive and behavioral interventions may improve emotional functioning following TBI. However, there are several limitations to these findings including small sample sizes, lack of control group, mixed etiology of injury, and use of convenience samples that may not be experiencing significant levels of emotional distress. Some studies excluded persons with more 
 Table 2
 Adaptations to cognitive-behavioral interventions

•	Provision of supplementary written materials	•	Built-in repetition and review of key concepts
•	Focus on concrete goals	•	Providing "extra time" for sessions
•	Provision of session summary notes	•	Providing within- session breaks
•	Limiting the amount of text on worksheets	•	Use of larger font size
•	Limiting size of group		Using visual aids and checklists
•	Providing multiple choice options on worksheets	•	Reducing emphasis on self-directed, higher level reasoning skills

severe cognitive deficits, resulting in improved internal validity, but limited generalizability of findings and prevention of further examination of the impact of cognitive deficits on response to the intervention.

Persons who sustain a TBI, especially a moderate or severe TBI, often experience cognitive deficits that could have a negative impact on their ability to benefit from standard psychological treatments. For example, poor attention and memory could impact learning and recall of session content. Decreased initiation could impact perceived compliance with treatment. Problemsolving deficits could impact completion of homework assignments. Thus, psychological interventions for the treatment of depression and anxiety following TBI should take such deficits into account and be adapted to fit the needs of the individual client. Incorporating cognitive rehabilitation techniques into psychotherapeutic interventions may be important for maximizing response to psychological treatments for emotional distress following brain injury [78].

Reported adaptations to the structure and content of cognitive-behavioral interventions are listed in Table 2. These and other adaptations have also been applied in a recent pilot study of a mindfulness-based stress reduction program for reducing depression in persons with mild TBI and post-concussion syndrome [79]. Findings to date suggest that persons with cognitive deficits can participate in and benefit from these adapted interventions [73, 74, 76]. It is also important to note that psychological treatment of emotional distress is a key component of comprehensive cognitive rehabilitation programs, and there is evidence that participation in these programs is associated with improved outcomes, including community integration, life satisfaction, emotional functioning, and self-efficacy [80, 81]. However, the impact of program components that are specific to the treatment

of emotional distress is unknown at this time.

The importance of developing and maintaining a meaningful life following TBI is often an important part of psychotherapy following TBI [82]. Ruff [83] describes some of the unique issues faced by psychotherapy clients with TBI due to the nature of their injury-related deficits and their effects on multiple life areas, including social relationships, vocational functioning, and financial status. Ruff suggests specific topic areas to explore when helping clients to re-establish meaning following TBI. These include identifying expectations for the anticipated future prior to injury, understanding how the TBI has altered the client's life and introduced functional limitations, grieving the loss of the anticipated future, and developing a realistic and meaningful future that involves living in accordance with one's core values. Given the relationship of functional limitations to depression, addressing functional limitations may be of considerable importance for improving and maintaining emotional well-being. Pagulayan et al. [23] suggest that addressing functional limitations may be an important part of treatment for depression in persons with TBI. Finally, consideration of individual preferences regarding treatment types is also important since comfort with and acceptance of treatment type may impact participation and adherence. Fann et al. [84] conducted a telephone survey of 145 persons with mild to severe TBI to explore preference regarding different treatments for depression. Physical exercise and counseling were preferred over other types of treatment including antidepressants, self-help materials, and group therapy or support groups.

There is a strong need for more research in psychotherapeutic interventions for emotional distress following TBI. Specifically, future studies should include larger samples, utilize measures validated for the diagnosis of emotional distress in the TBI population, include an appropriate control condition as well as measures of treatment fidelity, and include long-term follow-up assessments. Investigation of cognitive and psychosocial factors that may impact response to treatment is also warranted so that patients may be "matched" with the treatment most appropriate for them. Examination of interventions validated in other populations, such as behavioral activation approaches, may also be beneficial. Development and evaluation of interventions that target the needs of persons with dual substance abuse and mood disorder diagnoses following TBI is important given the unique needs of this population. Finally, efforts aimed at preventing depressive and anxiety disorders and maintaining emotional well-being over time can have significant and positive impacts on persons with TBI.

## **Case Studies**

The following case studies present different psychological approaches to treating emotional distress in persons who have experienced a TBI, and discuss factors that can inform treatment planning. Case 1 is an example of the treatment of emotional distress in a traditional outpatient setting. Case 2 is an example of the treatment of emotional distress within an interdisciplinary rehabilitation setting, and illustrates how other behavioral interventions can have an impact on emotional functioning following TBI. These examples also highlight how cognitive rehabilitation techniques can be incorporated into psychological interventions for emotional distress.

#### Case 1

A 39-year-old woman who experienced a moderate TBI at the age of 22 presented for neuropsychological evaluation with complaints of attention problems and anxiety. There was no other significant medical history. The patient graduated from high school and was employed full-time in a call center. She was the single mother of an 11-year-old boy. During the clinical interview, the patient described feeling overwhelmed at work following a recent promotion and uncertain of her ability to handle new job responsibilities. Her son is enrolled in gifted classes at his middle school, and she stated she was "not smart enough since my injury" to help her son with his increasingly difficult homework or to communicate with his teachers. She reported feeling "sick to my stomach" at a recent back-to-school night as the teachers reviewed upcoming student projects. Neuropsychological evaluation revealed mild impairments in information processing speed and variable performance on measures of attention, verbal learning, and memory. Performance on other cognitive measures was within normal limits. Responses to selfreport measures of emotional functioning revealed moderate to severe anxiety and mild depressive symptoms. The patient was referred for psychological treatment for anxiety and depression.

The results of the neuropsychological evaluation suggested that this patient would be a good candidate for a psychological intervention that included components of CBT, and her treatment plan was developed in accordance with this model. The patient expressed good understanding of this therapeutic approach and was able to identify thoughts with minimal prompting. She was an active and engaged participant during treatment sessions; however, she demonstrated very poor completion of assigned homework. Discussion of this homework issue revealed that the patient misplaced her homework on some occasions and, at other times, did not remember it until she was on the way to her appointment. Thus, this failure to complete the homework reflected a memory problem rather than noncompliance with the treatment plan. The therapist applied a structured problem-solving approach to the discussion of potential compensatory strategies she could use to help remember her homework, including the use of a memory station, memory notebook, smartphone, and checklists. The patient expressed a strong preference for use of her smartphone applications to help remember homework, and to have electronic versions of the homework that she could access easily. The patient, in collaboration with the therapist, developed a compensatory strategy centered around her smartphone to help her remember her homework. This strategy became quite successful following some initial refinement of the components. The patient's role in developing this strategy and successfully managing this memory problem was also used as evidence to counter her belief that she was "not smart enough" since her injury.

#### Case 2

A 19-year-old male who experienced a severe TBI at the age of 7 was referred to a post-acute brain injury rehabilitation program for vocational services. Neuropsychological evaluation showed impairments in multiple domains including attention, learning, memory, executive functions, and processing speed, as well as clinically significant depressive symptoms. Clinical interview revealed that the patient had some acquaintances, but no close friends; he socialized exclusively with family. Notable social communication deficits were observed including poor eye contact, the telling of moderately offensive jokes, frequent interruptions characterized by off-topic comments. The examiner noted that the client frequently acted younger than his age. His stated goals were to get a job, to obtain his driver's license, and to have a girlfriend.

In contrast to Case 1, the results of the neuropsychological evaluation described in Case 2 documented several cognitive impairments that would likely impact his ability to benefit from a CBT-based approach, especially impairments in executive functions. Thus, a more behavioral approach to the treatment of his depressive symptoms appeared warranted. The client expressed understanding of, and agreement with, his treatment plan. Further assessment revealed that the client enjoyed outdoor activities such as playing sports and walking with his dog. The client and therapist developed a schedule of regular meaningful activities and investigated communitybased recreational sports programs which led to his registration in a local judo class. Initially, he had some difficulty following his activity schedule. He reported that he could not remember what he was supposed to do first. Family also reported that he would confuse the order of the steps or get distracted by another activity and fail to return to the task. The therapist helped the client to develop checklists that contributed to improved completion of the tasks. Also, family decided to start paying client for completing household chores, including simple yard work and laundry, to reward successful task completion. Participation in meaningful activities and task completion improved. This was associated with improvements in depressive symptoms. The patient also participated in a social skills intervention group to address his social communication problems. Strengthening social skills can contribute to improved relationships with others, and thus increase perceived social support, which also plays an important role in the experience of emotional distress.

Acknowledgments Preparation of this chapter was partially supported by the U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133G070222, H133A070043, H133B090023, and H133A120020.

## References

- Guillamondegui, O. D., Montgomery, S. A., Phibbs, F. T., McPheeters, M. L., Alexander, P. T., Jerome, R. N., et al. (2011). Traumatic brain injury and depression. Comparative effectiveness review no. 25. (Prepared by the Vanderbilt Evidence-based Practice Center under Contract No. 290-2007-10065-I.) AHRQ publication no. 11-EHC017-EF. Rockville, MD: Agency for Healthcare Research and Quality. Retrieved from www.effectivehealthcare.ahrq.gov/ reports.final.cfm
- Bombardier, C. H., Fann, J. R., Temkin, N. R., Esselman, P. C., Barber, J., & Dikmen, S. S. (2010). Rates of major depressive disorder and clinical outcomes following traumatic brain injury. *JAMA*, *303*(19), 1938–1945.
- Hart, T., Hoffman, J. M., Pretz, C., Kennedy, R., Clark, A. N., & Brenner, L. A. (2012). A longitudinal study of major and minor depression following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 93(8), 1343–1349.
- Rao, V., Rosenberg, P., Bertrand, M., Salehinia, S., Spiro, J., Vaishnavi, S., et al. (2009). Aggression after traumatic brain injury: Prevalence and correlates.

Journal of Neuropsychiatry and Clinical Neurosciences, 21(4), 420–429.

- Hart, T., Brenner, L., Clark, A. N., Bogner, J. A., Novack, T. A., Chervoneva, I., et al. (2011). Major and minor depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 92(8), 1211–1219.
- Bay, E., Hagerty, B. M., & Williams, R. A. (2007). Depressive symptomatology after mild-to-moderate traumatic brain injury: A comparison of three measures. *Archives of Psychiatric Nursing*, 21(1), 2–11.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617–627.
- Hibbard, M. R., Ashman, T. A., Spielman, L. A., Chun, D., Charatz, H. J., & Melvin, S. (2004). Relationship between depression and psychosocial functioning after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 85(4 Suppl 2), S43–S53.
- Underhill, A. T., Lobello, S. G., Stroud, T. P., Terry, K. S., Devivo, M. J., & Fine, P. R. (2003). Depression and life satisfaction in patients with traumatic brain injury: A longitudinal study. *Brain Injury*, 17(11), 973–982.
- Jorge, R. E., Robinson, R. G., Starkstein, S. E., & Arndt, S. V. (1994). Influence of major depression on 1-year outcome in patients with traumatic brain injury. *Journal of Neurosurgery*, *81*(5), 726–733.
- Gomez-Hernandez, R., Max, J. E., Kosier, T., Paradiso, S., & Robinson, R. G. (1997). Social impairment and depression after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 78(12), 1321–1326.
- Groom, K. N., Shaw, T. G., O'Connor, M. E., Howard, N. I., & Pickens, A. (1998). Neurobehavioral symptoms and family functioning in traumatically braininjured adults. *Archives of Clinical Neuropsychology*, *13*(8), 695–711.
- Seel, R. T., Kreutzer, J. S., Rosenthal, M., Hammond, F. M., Corrigan, J. D., & Black, K. (2003). Depression after traumatic brain injury: A National Institute on Disability and Rehabilitation Research Model Systems multicenter investigation. *Archives of Physical Medicine and Rehabilitation*, 84(2), 177–184.
- Franulic, A., Carbonell, C. G., Pinto, P., & Sepulveda, I. (2004). Psychosocial adjustment and employment outcome 2, 5 and 10 years after TBI. *Brain Injury*, *18*(2), 119–129.
- Rosenthal, M., Christensen, B. K., & Ross, T. P. (1998). Depression following traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79(1), 90–103.
- Dikmen, S. S., Bombardier, C. H., Machamer, J. E., Fann, J. R., & Temkin, N. R. (2004). Natural history of depression in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 85(9), 1457–1464.

- Jorge, R. E., Robinson, R. G., Moser, D., Tateno, A., Crespo-Facorro, B., & Arndt, S. (2004). Major depression following traumatic brain injury. *Archives* of General Psychiatry, 61(1), 42–50.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., Starkstein, S. E., Forrester, A. W., & Geisler, F. (1993). Depression following traumatic brain injury: A 1 year longitudinal study. *Journal of Affective Disorders*, 27(4), 233–243.
- Whitnall, L., McMillan, T. M., Murray, G. D., & Teasdale, G. M. (2006). Disability in young people and adults after head injury: 5–7 year follow up of a prospective cohort study. *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 77(5), 640–645.
- Jorge, R. E., Acion, L., Starkstein, S. E., & Magnotta, V. (2007). Hippocampal volume and mood disorders after traumatic brain injury. *Biological Psychiatry*, 62(4), 332–338.
- Jorge, R. E., & Starkstein, S. E. (2005). Pathophysiologic aspects of major depression following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 20(6), 475–487.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., Forrester, A. W., Geisler, F., & Starkstein, S. E. (1993). Comparison between acute- and delayed-onset depression following traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 5(1), 43–49.
- 23. Pagulayan, K. F., Hoffman, J. M., Temkin, N. R., Machamer, J. E., & Dikmen, S. S. (2008). Functional limitations and depression after traumatic brain injury: Examination of the temporal relationship. *Archives of Physical Medicine and Rehabilitation*, 89(10), 1887–1892.
- 24. Schönberger, M., Ponsford, J., Gould, K. R., & Johnston, L. (2011). The temporal relationship between depression, anxiety, and functional status after traumatic brain injury: A cross-lagged analysis. *Journal of International Neuropsychological Society*, 17(5), 781–787.
- Ownsworth, T., Fleming, J., Haines, T., Cornwell, P., Kendall, M., Nalder, E., et al. (2011). Development of depressive symptoms during early community reintegration after traumatic brain injury. *Journal of International Neuropsychological Society*, 17(1), 112–119.
- 26. Cook, K. F., Bombardier, C. H., Bamer, A. M., Choi, S. W., Kroenke, K., & Fann, J. R. (2011). Do somatic and cognitive symptoms of traumatic brain injury confound depression screening? *Archives of Physical Medicine and Rehabilitation*, 92(5), 818–823.
- Kreutzer, J. S., Seel, R. T., & Gourley, E. (2001). The prevalence and symptom rates of depression after traumatic brain injury: A comprehensive examination. *Brain Injury*, 15(7), 563–576.
- Seel, R. T., Macciocchi, S., & Kreutzer, J. S. (2010). Clinical considerations for the diagnosis of major depression after moderate to severe TBI. *The Journal* of *Head Trauma Rehabilitation*, 25(2), 99–112.
- 29. Aloia, M. S., Long, C. J., & Allen, J. B. (1995). Depression among the head-injured and non-head-

injured: A discriminant analysis. *Brain Injury*, 9(6), 575–583.

- Baguley, I. J., Cooper, J., & Felmingham, K. (2006). Aggressive behavior following traumatic brain injury: How common is common? *The Journal of Head Trauma Rehabilitation*, 21(1), 45–56.
- Chamelian, L., & Feinstein, A. (2006). The effect of major depression on subjective and objective cognitive deficits in mild to moderate traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 18(1), 33–38.
- 32. Schiehser, D. M., Delis, D. C., Filoteo, J. V., Delano-Wood, L., Han, S. D., Jak, A. J., et al. (2011). Are self-reported symptoms of executive dysfunction associated with objective executive function performance following mild to moderate traumatic brain injury? *Journal of Clinical and Experimental Neuropsychology*, 33(6), 704–714.
- 33. Rapoport, M. J., McCullagh, S., Shammi, P., & Feinstein, A. (2005). Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 17(1), 61–65.
- 34. Satz, P., Forney, D. L., Zaucha, K., Asarnow, R. R., Light, R., McCleary, C., et al. (1998). Depression, cognition, and functional correlates of recovery outcome after traumatic brain injury. *Brain Injury*, 12(7), 537–553.
- Fann, J. R., Uomoto, J. M., & Katon, W. J. (2001). Cognitive improvement with treatment of depression following mild traumatic brain injury. *Psychosomatics*, 42, 48–54.
- 36. Ashman, T. A., Spielman, L. A., Hibbard, M. R., Silver, J. M., Chandna, T., & Gordon, W. A. (2004). Psychiatric challenges in the first 6 years after traumatic brain injury: Cross-sequential analyses of Axis I disorders. Archives of Physical Medicine and Rehabilitation, 85(4 Suppl 2), S36–S42.
- Hibbard, M. R., Uysal, S., Kepler, K., Bogdany, J., & Silver, J. (1998). Axis I psychopathology in individuals with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 13(4), 24–39.
- Hiott, D. W., & Labbate, L. (2002). Anxiety disorders associated with traumatic brain injuries. *NeuroRehabilitation*, 17(4), 345–355.
- Fann, J. R., Katon, W. J., Uomoto, J. M., & Esselman, P. C. (1995). Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *The American Journal of Psychiatry*, 152(10), 1493–1499.
- 40. Gould, K. R., Ponsford, J. L., Johnston, L., & Schönberger, M. (2011). The nature, frequency and course of psychiatric disorders in the first year after traumatic brain injury: A prospective study. *Psychological Medicine*, 41, 2099–2109.
- Bryant, R. A., O'Donnell, M. L., Creamer, M., McFarlane, A. C., Clark, C. R., & Silove, D. (2010). The psychiatric sequelae of traumatic injury. *The American Journal of Psychiatry*, *167*(3), 312–320.
- 42. Whelan-Goodinson, R., Ponsford, J., Johnston, L., & Grant, F. (2009). Psychiatric disorders following trau-

matic brain injury: Their nature and frequency. *The Journal of Head Trauma Rehabilitation*, 24(5), 324–332.

- Whelan-Goodinson, R., Ponsford, J., & Schönberger, M. (2008). Association between psychiatric state and outcome following traumatic brain injury. *Journal of Rehabilitation Medicine*, 40(10), 850–857.
- 44. Jorge, R. E., Robinson, R. G., Starkstein, S. E., & Arndt, S. V. (1993). Depression and anxiety following traumatic brain injury. *Journal of Neuropsychiatry* and Clinical Neurosciences, 5(4), 369–374.
- 45. Whelan-Goodinson, R., Ponsford, J. L., Schönberger, M., & Johnston, L. (2010). Predictors of psychiatric disorders following traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 25(5), 320–329.
- 46. Gil, S., Caspi, Y., Ben-Ari, I. Z., Koren, D., & Klein, E. (2005). Does memory of a traumatic event increase the risk for posttraumatic stress disorder in patients with traumatic brain injury? A prospective study. *The American Journal of Psychiatry*, 162(5), 963–969.
- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. Soldiers returning from Iraq. *New England Journal of Medicine*, 358(5), 453–463.
- Lippa, S. M., Pastorek, N. J., Benge, J. F., & Thornton, G. M. (2010). Postconcussive symptoms after blast and nonblast-related mild traumatic brain injuries in Afghanistan and Iraq war veterans. *Journal of International Neuropsychological Society*, 16(5), 856–866.
- Teasdale, T. W., & Engberg, A. W. (2001). Suicide after traumatic brain injury: A population study. *Journal of Neurology, Neurosurgery, and Psychiatry*, 71(4), 436–440.
- Ventura, T., Harrison-Felix, C., Carlson, N., Diguiseppi, C., Gabella, B., Brown, A., et al. (2010). Mortality after discharge from acute care hospitalization with traumatic brain injury: A population-based study. Archives of Physical Medicine and Rehabilitation, 91(1), 20–29.
- Brenner, L. A., Ignacio, R. V., & Blow, F. C. (2011). Suicide and traumatic brain injury among individuals seeking Veterans Health Administration services. *The Journal of Head Trauma Rehabilitation*, 26(4), 257–264.
- Simpson, G., & Tate, R. (2002). Suicidality after traumatic brain injury: Demographic, injury and clinical correlates. *Psychological Medicine*, 32(4), 687–697.
- 53. 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.
- 54. Tsaousides, T., Cantor, J. B., & Gordon, W. A. (2011). Suicidal ideation following traumatic brain injury: Prevalence rates and correlates in adults living in the community. *The Journal of Head Trauma Rehabilitation*, 26(4), 265–275.

- 55. Brenner, L. A., Carlson, N. E., Harrison-Felix, C., Ashman, T., Hammond, F. M., & Hirschberg, R. E. (2009). Self-inflicted traumatic brain injury: Characteristics and outcomes. *Brain Injury*, 23 (13–14), 991–998.
- Simpson, G., & Tate, R. (2005). Clinical features of suicide attempts after traumatic brain injury. *Journal* of Nervous and Mental Disease, 193(10), 680–685.
- 57. Dennis, J. P., Ghahramanlou-Holloway, M., Cox, D. W., & Brown, G. K. (2011). A guide for the assessment and treatment of suicidal patients with traumatic brain injuries. *The Journal of Head Trauma Rehabilitation*, 26(4), 244–256.
- Brenner, L. A., Homaifar, B. Y., Adler, L. E., Wolfman, J. H., & Kemp, J. (2009). Suicidality and veterans with a history of traumatic brain injury: Precipitants events, protective factors, and prevention strategies. *Rehabilitation Psychology*, 54(4), 390–397.
- Simpson, G. K., & Tate, R. L. (2007). Preventing suicide after traumatic brain injury: Implications for general practice. *Medical Journal of Australia*, 187(4), 229–232.
- Simpson, G. K., & Tate, R. (2007). Suicidality in people surviving a traumatic brain injury: Prevalence, risk factors and implications for clinical management. *Brain Injury*, 21(13–14), 1335–1351.
- 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.
- Dahm, J., Wong, D., & Ponsford, J. (2013). Validity of the Depression Anxiety Stress Scales in assessing depression and anxiety following traumatic brain injury. *Journal of Affective Disorders*, 151(1), 392–396.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation.
- Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1(3), 385–401.
- Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606–613.
- 66. Kreutzer, J., Marwitz, J., Seel, R., & Serio, C. (1996). Validation of a neurobehavioral functioning inventory for adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 77, 116–124.
- Lovibond, S. H., & Lovibond, P. F. (1995). Manual for the Depression Anxiety Stress Scales (2nd ed.). Sydney: Psychology Foundation.
- Henry, J. D., & Crawford, J. R. (2005). The 21-item version of the Depression Anxiety Stress Scales (DASS–21): Normative data and psychometric evaluation in a large non-clinical sample. *British Journal of Clinical Psychology*, 44, 227–239.

- Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. *Acta Psychiatrica Scandinavica*, 67(6), 361–370.
- 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.
- Soo, C., & Tate, R. (2007). Psychological treatment for anxiety in people with traumatic brain injury. *Cochrane Database Syst Rev*, (3), CD005239
- Khan-Bourne, N., & Brown, R. G. (2003). Cognitive behaviour therapy for the treatment of depression in individuals with brain injury. *Neuropsychological Rehabilitation*, 13(1–2), 89–107.
- Hsieh, M. Y., Ponsford, J., Wong, D., Schönberger, M., McKay, A., & Haines, K. (2012). A cognitive behaviour therapy (CBT) programme for anxiety following moderate-severe traumatic brain injury (TBI): Two case studies. *Brain Injury*, 26(2), 126–138.
- 74. Simpson, G. K., Tate, R. L., Whiting, D. L., & Cotter, R. E. (2011). Suicide prevention after traumatic brain injury: A randomized controlled trial of a program for the psychological treatment of hopelessness. *The Journal of Head Trauma Rehabilitation*, 26(4), 290–300.
- Anson, K., & Ponsford, J. (2006). Evaluation of a coping skills group following traumatic brain injury. *Brain Injury*, 20(2), 167–178.
- Bradbury, C. L., Christensen, B. K., Lau, M. A., Ruttan, L. A., Arundine, A. L., & Green, R. E. (2008). The efficacy of cognitive behavior therapy in the treatment of emotional distress after acquired brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(12 Suppl), S61–S68.
- 77. Armengol, C. G. (1999). A multimodal support group with Hispanic traumatic brain injury survivors.

*The Journal of Head Trauma Rehabilitation, 14*(3), 233–246.

- Mateer, C. A., Sira, C. S., & O'Connell, M. E. (2005). Putting Humpty Dumpty together again: The importance of integrating cognitive and emotional interventions. *The Journal of Head Trauma Rehabilitation*, 20(1), 62–75.
- Azulay, J., Smart, C. M., Mott, T., & Cicerone, K. D. (2013). A pilot study examining the effect of mindfulness-based stress reduction on symptoms of chronic mild traumatic brain injury/postconcussive syndrome. *The Journal of Head Trauma Rehabilitation*, 28(4), 323–331.
- Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., et al. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89(12), 2239–2249.
- Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., et al. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine* and Rehabilitation, 86(8), 1565–1574.
- Prigatano, G. P. (1991). Disordered mind, wounded soul: The emerging role of psychotherapy in rehabilitation after brain injury. *The Journal of Head Trauma Rehabilitation*, 6(4), 1–10.
- Ruff, R. (2013). Selecting the appropriate psychotherapies for individuals with traumatic brain injury: What works and what does not? *NeuroRehabilitation*, 32(4), 771–779.
- 84. 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. *The Journal of Head Trauma Rehabilitation*, 24(4), 272–278.

# Treating and Collaborating With Family Caregivers in the Rehabilitation of Persons with Traumatic Brain Injury

Angelle M. Sander

# Abstract

Family members are an important source of support for persons with traumatic brain injury (TBI) during the recovery process and are potential partners in the rehabilitation process. Unfortunately, family members often experience substantial emotional distress and disruption of family functioning. These difficulties can impact the rehabilitation process and the recovery of the person with injury. Understanding the problems faced by family members and potential ways to assist them in helping the person with injury to achieve maximum functioning is important for neuropsychologists working in the area of TBI. The current chapter begins with an overview of the types of difficulties faced by family members in the acute and chronic phases of TBI. Next, an overview will be provided of the ways in which neuropsychologists can work with family members in different treatment settings including acute care, inpatient rehabilitation, post-acute rehabilitation, and private practice. For each setting, case examples are provided to describe the role of the family in treatment and how the neuropsychologist can meet family members' needs and involve them as partners in the rehabilitation process. The chapter ends with additional tips for working with family caregivers after TBI, along with a sample of educational resources that can be distributed to family members.

#### Keywords

Caregivers • Family members • Working with family members • Rehabilitation with caregivers

A.M. Sander, Ph.D. (⊠) Physical Medicine and Rehabilitation, Baylor College of Medicine and Harris Health System, Houston, TX, USA

Brain Injury Research Center, TIRR Memorial Hermann, Houston, TX, USA e-mail: Angelle.Sander@memorialhermann.org

# Overview of Impact of TBI on Family Caregivers

Over three decades of empirical work leaves no doubt that traumatic brain injury (TBI) results in significant distress for family caregivers. Emotional distress, including depression and anxiety, has been documented during the first year following injury and as long as 7 years after injury [1–10]. While injury-related impairments in the person with TBI often improve over time, caregivers have been shown to experience increased distress over time [11, 12]. This distress is manifested in a variety of ways, including increased seeking of mental health services and use of alcohol and sedative drugs [13]. Approximately one-third of primary caregivers of persons with TBI report clinical levels of pre-injury emotional distress, which may make them especially vulnerable to poor adjustment following injury [14]. Indeed, caregivers with a pre-injury history of treatment for psychological difficulties have been shown to report greater emotional distress at 1 year following injury [15].

Research has also documented the negative impact of TBI on family relationships. Caregivers have reported disruption of normal family functioning following TBI, including decreased communication, blurring of family roles, and decreased sharing of warmth and affection [4]. These changes may be partially related to cognitive and behavioral impairments in the person with TBI, but can also be a result of injury-related changes in family schedules, finances, and lifestyles. For example, financial stress may occur when the person with injury is not able to resume working. This can result in other family members working extra jobs, which leads to reduced family time. This situation can also lead to feelings of resentment for some family members and guilt for others, which can further strain relationships, resulting in a disruption of normal family functioning.

The marital relationship has been shown to be particularly vulnerable to negative consequences based on TBI, with spouses of persons with TBI reporting substantial levels of distress [16-18]. This distress is related to altered dynamics of the relationship (e.g., spouse in a dependent role), as well as role strain resulting from the uninjured spouse resuming a larger number of financial and household responsibilities [19]. Emotional and behavioral changes in the person with injury have been noted as a primary factor contributing to spousal distress [20, 21], and many spouses report feeling as if they are married to a stranger [19]. Reduced physical and emotional intimacy [22], as well as more negative perceptions and interactions within couples' relationships [23], have been documented. These changes can lead to reduced marital satisfaction [19, 23], reduced relationship quality [12], and eventually to separation or divorce [24, 25].

Due to the fact that TBI occurs disproportionately in younger people, it is not uncommon for persons with TBI to be single at the time of injury. In these cases, a parent may be the primary caregiver. There is some evidence that parents adjust better to the role of caring for a person with TBI as compared to spouses [16–18]; however, this finding has not been consistent across studies and parents have been noted to have emotional distress following injury [3, 5, 6]. Parents are often elderly and/or retired and may have limited resources to care for an adult child with injury. Furthermore, caring for an adult child with TBI may result in disruption of existing roles and boundaries as the adults were likely functioning independently of parents before injury and may now be in an unanticipated role of dependency. Conflicts between independence and dependence can be particularly stressful in these situations as the parents of an adult with TBI may have to restrict activities of their child due to cognitive or behavioral impairments.

While there is no research on the impact of parental TBI on children in the family, it is difficult to imagine that the children are unaffected. Children may experience substantial emotional distress including fear for their own safety and that of their parents, depression and anxiety regarding changes in their parent with injury, anger or resentment regarding decreased attention from one or both parents, and uncertainty regarding their futures. Children in this situation may become depressed or may act out behaviorally. This can result in increased stress on the family system.

# Importance of Addressing Family Caregiver Needs in the Context of Providing Neuropsychological Services Following TBI

For persons recovering from TBI, family members or caregivers can play a crucial role in following through with recommended strategies and services and can contribute to positive outcomes for the person with TBI. Researchers have documented that caregiver distress and unhealthy family functioning are related to worse participation outcomes for persons with complicated mild to moderate injury [26] and that poor progress in post-acute rehabilitation is predicted by unhealthy family functioning [27] and caregiver emotional distress [28]. This is likely because family caregivers experiencing distress are less able to provide a supportive environment to facilitate the ability of the person with TBI to compensate for injury-related impairments and to assist with resuming activities in the home and community. Neuropsychologists who work in rehabilitation settings treating persons with TBI can facilitate positive outcomes by engaging family caregivers in treatment. This is particularly the case when teaching strategies to compensate for cognitive impairments such as those in attention, memory, and executive functioning (e.g., planning, problemsolving, self-regulation). The length of stay in rehabilitation programs is typically brief and family members can assist with generalizing the strategies to everyday activities in the home and community. Even neuropsychologists who do not work in a rehabilitation setting will have reason to engage family members. While some neuropsychologists still work in settings where testing is used solely for diagnostic purposes, this is becoming much more rare. It is typical for referral sources to request recommendations for improving function in the face of cognitive impairments. Given that most persons with TBI have impairments that may make it difficult to recall and independently follow through with recommendations, providing feedback to family members is an important method of providing comprehensive care. Family members who are physically and mentally healthy are more likely to follow through with recommendations.

## Family Caregiver Needs

Kreutzer and colleagues completed a series of studies documenting the needs of family caregivers at various time points following TBI [29–31]. Their findings provided evidence that the priority rated as most important by family members was the need to receive medical information and education on the physical, cognitive, and emotional/ behavioral changes associated with TBI. Family members expressed a desire to have this information presented honestly and in language that they could understand. This series of studies also investigated the extent to which family members perceived their needs as being met. While family members largely reported their needs for information as being met, they reported needs for emotional support and instrumental support (e.g., help with practical things like housekeeping) as primarily unmet. These needs were reported as unmet by most family members up to 2 years post-injury. This is significant, as unmet needs are related to greater distress for caregivers [32]. Understanding caregiver needs and accurately assessing their emotional functioning is important for neuro-psychologists in order to facilitate appropriate referrals and to maximize the likelihood that the person with TBI will benefit from recommendations and/or treatment.

# Neuropsychologists' Roles in Treating and Collaborating With Family Caregivers

The role of neuropsychologists in working with family caregivers can differ by setting. Therefore, the roles will be discussed in reference to inpatient/ acute trauma, inpatient rehabilitation, post-acute rehabilitation, and private practice settings.

#### Inpatient/Acute Trauma Setting

The inpatient acute trauma experience is a particularly difficult one for family members. The catastrophic nature of the injury is usually emotionally overwhelming, and family members can be confused about the information that they are often bombarded with from neurosurgeons and the rest of the trauma team. Particularly in the initial days following injury, their emotions may be up and down. They may move from fear over the possibility of losing their loved one, to joy over their survival, to concern for their post-injury functioning. The role of the neuropsychologist during this phase is to provide early education and emotional support to the family. Initial education should be brief, as family members may have difficulty processing and recalling detailed information when they are under such emotional distress. Simple information on how the brain is affected by TBI and why certain medical procedures are being performed should be provided in language that they can understand rather than in medical or psychological jargon. Emotional support should be provided as needed.

The need for education becomes more intensive as the person with TBI emerges from the critical period, regains consciousness, and moves from the neurosurgery critical care unit to a regular hospital floor. At this point, family members may be surprised and confused by some common TBI-related behavior. For example, many persons with TBI demonstrate a phase of agitation, which can result in them pulling out medical tubes and devices and, in some cases, physically lashing out at treatment staff and/or family members. The neuropsychologist is often the professional who is best equipped to explain this behavior to family members as a normal part of the recovery process, to educate them on the temporary nature of these symptoms, and to advise them on ways to help reduce the agitated behavior by maintaining reduced stimulation in the environment, limiting the number of visitors at any one time, speaking quietly, and avoiding surprise approaches.

Family members are also confused by the period of posttraumatic amnesia or confusion

that typically follows in the hours, days, or weeks after injury. They do not initially understand why their loved one seems to be unaware of the passage of time or is unable to hold on to information from one moment to the next. Education and reassurance that these symptoms will resolve over time is important. Neuropsychologists can play an important role during this time by tracking patients' emergence from posttraumatic amnesia and informing family members of progress. Family members should also be educated on what information to provide patients with while they are in the stage of posttraumatic amnesia. The following case demonstrates how confusing posttraumatic amnesia can be for family members and the role that the neuropsychologist can play in helping them through it.

MP was a 20-year-old woman who sustained a TBI in a motor vehicle accident. Her boyfriend of 2 years was the driver of the vehicle and did not survive. MP remained in coma for 6 days. When she emerged from coma, she was moved to a regular hospital floor and was able to move around, consistently follow commands, and communicate her needs. She was not able to recall that she had been in a motor vehicle accident and did not hold on to the information when it was explained to her. She intermittently realized that she was in a hospital, but at times, thought that she was in the college dormitory where she had resided prior to injury. She repeatedly asked her parents where her boyfriend was and why he was not visiting. Her parents were confused by her inability to recall events and were distressed about having to tell her that her boyfriend had passed away. The neuropsychologist explained why MP was experiencing confusion and let the parents know that this is a normal stage of recovery that would resolve over time. As she was unable to retain information told to her and was in the very early stages of recovery, the neuropsychologist advised the family to redirect her attention when she asked about her boyfriend. This action would avoid the parents having to experience the distress of retelling the story to her on multiple occasions and also avoid distressing MP at a time when she did not have the cognitive capacity to process the news. Fortunately, MP was easy to redirect, which the neuropsychologist was aware of based on their interactions. Over the following two weeks, MP emerged from posttraumatic amnesia and began to recall that she had been at a party with her boyfriend and they had gotten in the car to drive home. At that point, the neuropsychologist

advised her parents that it was appropriate to inform her of her boyfriend's death and provided emotional support during the process.

A subset of persons with TBI remain in a vegetative or minimally conscious state for an extended period of time. The neuropsychologist's role for these families is to provide education related to the prognosis for recovery in patients with prolonged duration of unconsciousness and to assist them with decision-making regarding post-rehabilitation placement. Family members making these decisions will need counseling to manage their grief and distress and to avoid guilt and self-blame if they decide that they are unable to provide in-home care. In cases where family members decide to care for a person in vegetative or minimally conscious state at home, the neuropsychologist can assist with developing a plan for daily physical and cognitive stimulation. The neuropsychologist can also provide information on resources and referrals that family members may need to sustain care in the long term.

#### **Inpatient Rehabilitation Setting**

Many patients with TBI who receive inpatient rehabilitation services do so within the first 6 months of recovery. This is also the most rapid period of recovery, when persons with TBI tend to show frequent improvements in physical, cognitive, and emotional functioning. Family members often react to this rapid recovery with great hope for the future. Their initial distress can turn to happiness and enthusiasm for each step that the person with TBI takes toward improvement. While this is a positive outlook for family members, it can sometimes be an obstacle for preparing them adequately for discharge. They are often under the impression that the current rate of improvement will continue until the person with TBI achieves their pre-injury level of function. Such a belief may render them unlikely to fully process and/or retain information on the problems they may face following discharge. Many rehabilitation staff members talk about family caregivers being in denial when they hold onto these hopes for the future and appear to ignore advice regarding long-term problems. It is important to recognize that family members are reacting to what they are seeing and experiencing on an everyday basis, and that they may not be at a stage where they can process information that is inconsistent with their observations. Furthermore, many family members have developed a distrust of medical advice based on dire prognosis that they may have received from neurosurgery staff during the acute hospital stay. Family members who were told that their loved ones would never walk or eat on their own, yet witness them doing these very things, may come to believe that medical providers always give the worst case scenario and that their loved one is the exception to the rule. This can be a powerful belief to overcome, as it provides them with much-needed hope.

During this phase of in-patient rehabilitation, the neuropsychologist should monitor the daily cognitive and emotional progress of the person with injury. Serial assessment of the changes in cognitive and emotional status by a neuropsychologist can provide important information to family members, as well as to members of the interdisciplinary rehabilitation team, regarding how to improve function and to increase potential to benefit from therapy. The neuropsychologist should meet with each patient's family members at least weekly to provide education on progress. Family members also benefit from advice on how to interact with their loved one to maximize success. This is especially the case when the person with injury exhibits difficult to manage behaviors such as agitation or disinhibition. Family members often have difficulty understanding the impaired awareness of deficits that is typical in the early stages of recovery following TBI [33, 34]. The fact that the person with injury may seem unbothered by decreased abilities and may even argue about their existence can be confusing and stressful for family members. The neuropsychologist can assist by providing education about the cause and prognosis for impaired awareness. The neuropsychologist can also assist family members in determining ways to assist the person with injury in becoming more aware of problems and how to cope with them. The following example illustrates the role of the neuropsychologist in this process.

RH was a 19-year-old Hispanic man who sustained a gunshot wound to the right frontal lobe during an attempted car-jacking. He was admitted to a comprehensive inpatient unit for rehabilitation of physical and cognitive deficits. Neuropsychological evaluation revealed impairments in left hand motor speed and dexterity, organization of visual scanning, learning and retention of word lists, abstract verbal reasoning, and higher level problem-solving. He also sustained mild left-sided weakness that impacted balance. He was cooperative and communicated in a polite manner with the hospital staff. His parents were pleased with the progress he was making; however, they expressed distress regarding his seeming unawareness of his cognitive deficits and balance problems. He would often attempt to transfer from his wheelchair to bed on his own, and had already fallen twice. When he forgot information, he often confabulated or filled in his memory gaps with inaccurate information. For example, he believed that his physical therapist had cleared him to do transfers on his own, even though she had clearly informed him that he should have assistance. RH also insisted that he would be able to return to living independently following discharge, in spite of the team's recommendation that he should be supervised. He had never argued with his parents before injury, but was frequently doing so now, mainly over how independent he should be in activities.

The neuropsychologist met with the family to provide education regarding impaired awareness as a stage of recovery from TBI. He encouraged them to avoid engaging in arguments with RH, as it could result in distress for everyone involved. Instead, he helped them to start a memory notebook where they could help RH to record all therapists' recommendations. They would remind him to look at it at regular times throughout the day, so that he could begin to learn the recommendations through repetition. His parents were also taught to help RH make a chart that showed his progress in physical therapy, so that he could visualize how he was doing at balance activities and how he was improving. Having these concrete examples of functioning and progress helped to anchor RH in the present reality and to avoid arguments. Over his 4 weeks on the rehabilitation unit, he gradually began to understand that while he was making progress, he would need his parents' assistance for a while following discharge.

As discharge from inpatient rehabilitation approaches, family members may need increased education and support from the neuropsychologist. It is not atypical that certain cognitive deficits manifest less in the structured therapy environment than they do in the home or community environments, when the person with TBI is responsible for imposing his or her own structure. Based on neuropsychological evaluation conducted close to the time of discharge, family members should be provided with education on the key impairments noted in the person with injury and on how these are likely to manifest in their home and community environments. Family members should then be instructed in how to employ compensatory strategies and/or how to structure the home environment to maximize success. For example, a person with impairments in selective attention could benefit from completing tasks or learning information with minimal distractions and with frequent repetition. A person with impairments in initiation could be provided with a checklist of regular daily activities to complete. Helping family members to plan these strategies in advance of discharge can help to ease their transition to serving as the sole caregivers for the person with injury. They should also be provided with a list of resources that they may need, including outpatient cognitive rehabilitation programs, counseling and psychotherapy services, and Internet sites, books, and fact sheets for gathering information as needs arise.

#### **Post-Acute Rehabilitation Setting**

The post-acute phase of rehabilitation can begin at any time following discharge from either acute trauma care or from acute inpatient rehabilitation. Persons with TBI receiving services in the post-acute rehabilitation setting are typically residing at home or in another private residence, with the assistance of a family member or other caregivers. During this phase, family caregivers are attempting to adjust to the impact of injuryrelated impairments on the everyday functioning of the person with injury. For many family members, the feelings of hope that they experienced during the early recovery process begin to wane during the first 6 to 9 months after injury. Emotional distress can increase as they become more aware of the limitations of the person with injury and how these limitations will impact their lives. Information told to family members during the earlier stages of recovery may not be recalled when they are actually facing the realities in their home environment. Neuropsychologists in the post-acute rehabilitation setting play a crucial role in helping family members through this difficult time and preparing them for the end of rehabilitation services.

One of the most important goals of postacute rehabilitation is to teach the person with injury skills needed to function more independently in the home and community environments. Toward this end, there is a focus on training in strategies to compensate for cognitive impairments in areas such as attention, memory, planning, and problem-solving. The family is an integral part of this training process for a few reasons. First, family members spend the most time with the person with injury and have first-hand knowledge of their strengths and weaknesses in their everyday environments. Second, family members can help the person with TBI to learn compensatory cognitive strategies. Some of the greatest obstacles to successful use of compensatory strategies are difficulties recognizing when they should be used and trouble recalling that they have the strategy available and how to use it. Family members can help increase success by prompting the person with TBI to use the strategy at appropriate times and assisting them in recalling the mechanics of strategy use. Finally, family members can play an important role in helping the person with injury to generalize the strategy to settings outside of rehabilitation. The neuropsychologist can work with the family to set functional goals for activities at home or in the community and then choose compensatory strategies to help achieve those goals. Family members can then assist the person with injury to use the strategy properly by practicing it on a regular basis. Family members can also help them to adapt the strategy for use in different situations in the home and community. The following example demonstrates how the neuropsychologist can

involve a family caregiver to maximize success of compensatory strategy training.

VM was a 20-year-old woman who was in her junior year of college when she was injured in an auto-pedestrian accident. She sustained bilateral frontal and left temporal lobe contusions. Following three weeks of inpatient rehabilitation, she was discharged home to live with her mother and younger brother. She was referred to a comprehensive post-acute cognitive rehabilitation/community re-entry program and started therapies approximately seven months after her injury. The neuropsychologist conducted an evaluation during her first two days in the program. The results showed strengths in receptive and expressive language, visuo-perceptive skills, immediate auditory memory, recall of stories, and abstract reasoning. Impairments were noted in visuo-constructive skills, organization, sustained and selective attention, learning and recall of word lists, retention of stories following a delay, cognitive flexibility, and higher level problem-solving. The neuropsychologist met with VM and her mother to set treatment goals. Her mother provided input that VM seemed unable to complete tasks. She would start one thing, stop, forget what she was doing, and move on to something else. She was also having difficulty remembering to complete the household chores that her mother had assigned to help her stay active. Another problem that was stressful for her mother was VM's persistent interruption of others during conversations. She did not wait her turn, but simply jumped in whenever she wanted, often changing topic. This had become embarrassing for her mother during social outings.

With the mother's input, the neuropsychologist decided to focus on improving VM's organization by developing a checklist strategy to help her keep track of steps in each task she was doing and to keep track of assigned household chores. They also worked on developing a signal strategy that the mother could use to quietly let VM know when she was interrupting others. When she saw this signal (a slight wave of the hand from her mother), she would be cued to stop whatever she was saying and say, "I'm sorry I interrupted you. What were you saying?" VM's mother played a crucial role in her learning and using both of these strategies outside of the rehabilitation setting. Through frequent practice encouraged by the neuropsychologist, she was able to move from initially using the checklist strategy with her mothers' regular prompting and guidance, to using it independently by the end of the 2-month treatment program. Toward the end of treatment, the neuropsychologist trained the mother to help VM adapt the checklist strategy for the steps that she would need to accomplish in order to resume school. This generalization would not have been possible without family involvement. VM continued to need her mother's signal to be aware of when she was interrupting a conversation, but she began to respond to this signal more rapidly and consistently.

In this case, lack of family involvement in the treatment planning and implementation of strategies could have led to development of compensatory strategies based on neuropsychological test results alone. These strategies may have had relevance in the rehabilitation setting, but may not have generalized to the everyday environment. The mother's input allowed the neuropsychologist to set functionally relevant goals and to assist VM in developing strategies that could be applied and practiced in her everyday environment to improve her functioning. In turn, the mother's involvement in planning and implementing compensatory strategies reduced her emotional distress and that of other family members, as VM became more independent and could assist with the household chores and could complete more tasks without family members' help.

In addition to cognitive impairments, family members of persons with TBI also have a difficult time coping with changes in their loved ones' emotional functioning and personality. Indeed, changes in personality and emotional response have been noted to be the most distressing injury-related change for family members [20, 21]. While family members may be aware that cognitive impairments are related to the brain injury, it is often difficult for them to understand that changes in personality are related to impaired brain function. Even when they are educated in the relationship between personality and brain function, they may continue to express irritation and frustration in interactions with the injured family member. They may continue to personalize certain behaviors. For example, some family members interpret impaired initiation as poor motivation on the part of the person with injury. They may become frustrated and angry, and these emotions filter into their interactions with the person with injury. Impaired social communication is another injury-related change that can be very stressful for family members and that they often

have negative reactions to. Behaviors such as non-responsiveness to facial expressions and body language, overly long eye contact, and difficulty taking turns and listening can be difficult for family members to cope with. The tendency is to forget the contribution of the brain injury and become exasperated with the behavior of their loved one. This can have a negative impact on family interactions and can ultimately lead to family dysfunction.

Neuropsychologists can assist family members in coping with changes in emotions and personality by educating them as to how the injury is contributing to these changes. They can also help family members to develop better ways to respond to these behaviors. For example, a combination of a checklist and a phone alarm may be used to help a person with impaired initiation to complete regular activities each day. Family members are likely to be less frustrated and less likely to attribute decreased motivation to the person with injury if they see them engage in more activity. Family members also feel more in control of the situation when they have tools that they can use to impact the behavior of the person with injury and assist them in achieving goals.

As during the acute trauma and inpatient rehabilitation phases, neuropsychologists should attend to the emotional needs of family members during the post-acute rehabilitation process. Tools, such as compensatory strategies, can help family members feel less overwhelmed and more in control; however, these tools will not help them to overcome the feelings of grief associated with perceived loss of their former lives. Many family members feel that the event of the injury changes the course of their life in an unsatisfactory way. For some, these feelings of loss and grief will resolve over time and they will adjust relatively well to their post-injury lifestyle. For others, the feelings of loss will lead to emotional distress that may be long-lasting unless treated. When possible, neuropsychologists can assist by providing psychotherapy to help them through this process; however, many insurance companies will not reimburse for therapy provided to family members, unless it includes the person with injury and is incorporated into the rehabilitation goals for that person. When it is not possible for the neuropsychologist to directly provide treatment to family members, they should be referred to other therapists as needed. Unfortunately, there is a lack of strong empirical support for the effectiveness of psychotherapy with family caregivers of persons with TBI. While minimal research has been devoted to this topic, there is no reason to believe that psychotherapies that have proven to be effective in treating emotional distress in other populations would not be effective with family caregivers following TBI. Small studies have indicated that a therapy program including education about consequences of injury, stress management, and training in coping strategies and problem-solving can result in a reduction in anxiety and use of escape-avoidance [35] and a reduction in unmet needs [36].

## **Private Practice Setting**

Many persons with TBI do not have the benefit of receiving formal rehabilitation in an acute or post-acute setting. Therefore, family members' first encounter with a neuropsychologist may be in the context of a referral to a private practice neuropsychologist for assessment and recommendations. While the setting differs, the principles of working with families in rehabilitation still apply in private practice. The neuropsychologist should strive to involve the family members by educating them regarding the cognitive, emotional, and behavioral impairments noted during assessment. When providing this education, the neuropsychologist should seek family members' input into how impairments are being manifested in the home and community environments and whether there are functional problems that may not have emerged during the testing session. The neuropsychologist should then provide recommendations for compensatory strategies and/or environmental changes that could lead to improved functioning. Neuropsychologists should also include questions in their interview to ascertain information on how family members are coping with the injury and how this may be impacting the person with injury. It is understandable that neuropsychologists in private practice may not be able to conduct formal assessments of family functioning; however, the addition of a few structured interview questions to the usual interview with the person with injury can be helpful. Inclusion of family caregivers in the interview process is recommended when possible.

The following case description illustrates how knowledge of family members' functioning and family dynamics can be a crucial piece of information for the neuropsychologist to consider when making recommendations.

EC was a 35-year-old father of three who was injured in a motor vehicle accident. He sustained bilateral frontal lobe contusions and subarachnoid hemorrhage. He was discharged home from the acute care hospital. He had been the driver of the vehicle that had gone through a stop sign and had hit another vehicle. His 8-year-old son had been in the back seat wearing a seatbelt and had sustained a pelvic injury, but no documented TBI. EC had not received formal rehabilitation services. EC presented for neuropsychological testing at the request of his neurosurgeon who had seem him in the follow-up clinic. At the time of testing, he was still not working and was at home alone every day. His wife, an ER nurse, had rearranged her schedule to be able to take her son to physical therapy in the mornings. She then worked an 11 a.m. to 7 p.m. shift and returned home after dinner. EC's neuropsychological test performance revealed average scores in all areas of functioning except for auditory recognition memory (characterized by a high number of false alarm errors), poor organization when copying a complex figure, and impaired performance on a card-sorting task. While his learning and recall of a word list was within normal limits for the numbers of words recalled, he made a high number of intrusion errors.

EC's wife presented as being under a great deal of stress. She verbalized anger at EC for not driving safely with their son in the car. She also expressed anger that he was unable to have dinner and homework completed by the time that she arrived home from her shift at 7:30 p.m. He would often be in the middle of cooking dinner and the children would be watching television instead of doing their homework. She was also angry that he seemed to be unaware that there was any problem. She was exhausted from working, but felt that she could not relax, as she "must keep the household running." When the neuropsychologist began to discuss use of compensatory strategies, EC's wife began to cry, stating that she could not handle being in charge of anything else. She perceived the strategies as another thing that she would need to "stay on top of." She also expressed the belief that her husband was not working hard enough and was not organizing his time effectively. She stated that he had a problem organizing his time prior to injury and that this had been a source of conflict between them. Based on this information, the neuropsychologist realized that simply prescribing compensatory strategies and expecting the wife to help EC implement them would not be effective. He set up a series of cognitive therapy sessions, during which he worked with EC to develop some organization strategies independently. He also referred EC and his wife for couples counseling with a colleague who had some experience in working with couples after TBI. He also referred EC to the state vocational agency to apply for vocational rehabilitation services.

# Summary and Conclusions

Family caregivers who are under emotional distress can be an obstacle to the home and commuwith nitv reintegration of persons TBI. Neuropsychologists working with persons who have sustained TBI must include family caregivers to be truly effective. The services needed by family caregivers differ by setting, including acute care, inpatient rehabilitation, post-acute rehabilitation, and private practice. Common across all settings are the needs for education about injury-related changes and how to manage them, and the family caregivers' need for assistance in managing their own emotional distress and feelings of grief and loss. The method of approaching these needs and family members' emotional responses varies by setting and time since injury. Involvement of family caregivers in the rehabilitation process can maximize the person with injury's benefit from rehabilitation and can help to ensure generalizability of what is learned in the rehabilitation setting to the everyday environment. Adequately meeting family caregivers' needs can also improve their own mental and physical health, which is important in its own right.

# Additional Tips for Working with Family Caregivers After TBI

- Family members' ability to process and recall information given to them early during the recovery process may not be optimal. Providing them with written materials that they can refer to later is recommended. The Traumatic Brain Injury Models Systems investigators, funded by the National Institute on Disability and Rehabilitation Research, have created a series of consumer fact sheets. There are fact sheets on a variety of problems specific to TBI, including depression, sexuality, sleep disturbance, and driving ability. These can be printed and distributed to persons with injury and family members at no cost. http://www.msktc.org/ tbi/factsheets
- Simply informing family caregivers of impairments in the person with injury is not usually helpful. It is most beneficial for them to have tools that they can use to help to manage functional impairments in everyday life. The following resources contain information on strategies for family caregivers to manage injury-related problems.
  - "Picking Up the Pieces After TBI: A Guide for Family Members": This is an informational booklet written for family caregivers. It contains information on typical everyday problems (physical, cognitive, and emotional/behavioral) resulting from TBI and ideas for how to manage them. It also contains information on stress management, coping, and problem-solving for family caregivers. http://www.tbicommunity.org/ resources/publications/famEducManual.pdf (English version) http://www.tbicommunity. org/resources/publications/famEducManual-Spanish.pdf (Spanish version)
  - "Understanding TBI Issues for Caregivers": This is an online course for caregivers. It presents compensatory strategies and management techniques for cognitive and emotional/behavioral problems. http://www. tbicommunity.org/resources/courseAvenue/ caregivers\_course.htm

- Cultural factors can influence how family caregivers perceive their roles and react to the person with TBI. For example, adult children of an Asian parent with TBI may be reluctant to direct their father's activities, even when the neuropsychologist recommends doing so. In certain cultures, family members may consider caring for a family member with injury to be an obligation, and may reject any attempt to discuss emotional distress associated with the caregiving role. Neuropsychologists should be sensitive regarding these cultural differences and should adapt their feedback and education accordingly.
- Pre-injury problems in family dynamics and functioning seldom improve after injury. Including some questions about pre-injury family relationships as part of the interview process can aid in planning how to approach education and treatment, both for the person with TBI and their family caregivers.

Acknowledgement Preparation of this chapter was partially supported by the U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grants H133A070043, H133B090023, and H133A120020.

# References

- Brooks, N., Campsie, L., Symington, C., Beattie, A., & McKinlay, W. (1986). The five year outcome severe blunt head injury: A relative's view. *Journal of Neurology, Neurosurgery, and Psychiatry*, 49, 764–770.
- Brooks, N., Campsie, L., Symington, C., Beattie, A., & McKinlay, W. (1987). The effects of severe head injury on patient and relatives within seven years of injury. *The Journal of Head Trauma Rehabilitation*, 2, 1–13.
- Gillen, R., Tennen, H., Affleck, G., & Steinpreis, R. (1998). Distress, depressive symptoms, and depressive disorder among caregivers of patients with brain injury. *The Journal of Head Trauma Rehabilitation*, 13, 31–43.
- Kreutzer, J. S., Gervasio, A. H., & Camplair, P. S. (1994). Primary caregivers' psychological status and family functioning after traumatic brain injury. *Brain Injury*, 8, 197–210.
- Livingston, M. G., Brooks, D. N., & Bond, M. R. (1985). Three months after severe head injury: Psychiatric and social impact on relatives. *Journal of Neurology, Neurosurgery, and Psychiatry,* 48, 870–875.
- Livingston, M. G., Brooks, D. N., & Bond, M. R. (1985). Patient outcome in the year following severe

head injury and relatives' psychiatric and social functioning. *Journal of Neurology, Neurosurgery, and Psychiatry,* 48, 876–881.

- Marsh, N. V., Kersel, D. A., Havill, J. H., & Sleigh, J. W. (1998). Caregiver burden at 6 months following severe traumatic brain injury. *Brain Injury*, 12, 225–238.
- Marsh, N. V., Kersel, D. A., Havill, J. H., & Sleigh, J. W. (1998). Caregiver burden 1 year following severe traumatic brain injury. *Brain Injury*, *12*, 1045–1059.
- Kreutzer, J. S., Rapport, L. J., Marwitz, J. H., Harrison-Felix, C., Hart, T., Glenn, M., et al. (2009). Caregivers' well-being after traumatic brain injury: A multicenter prospective investigation. *Archives of Physical Medicine and Rehabilitation*, 90, 939–946.
- Ponsford, J., & Schonberger, M. (2010). Family functioning and emotional state two to five years after traumatic brain injury. *Journal of International Neuropsychological Society*, 16, 306–317.
- Perlesz, A., Kinsella, G., & Crowe, S. (1999). The impact of traumatic brain injury on the family: A critical review. *Rehabilitation Psychology*, 44, 6–35.
- Ponsford, J., Olver, J. P., Ponsford, M., & Nelms, R. (2003). Long-term adjustment of families following traumatic brain injury where comprehensive rehabilitation has been provided. *Brain Injury*, 17, 453–468.
- Hall, K. M., Karzmark, P., Stevens, M., Englander, J., O'Hare, P., & Wright, J. (1994). Family stressors in traumatic brain injury: A two-year follow-up. *Archives* of *Physical Medicine and Rehabilitation*, 75, 876–884.
- Sander, A. M., Sherer, M., Malec, J. F., High, W. M., Thompson, R. N., Moessner, A. M., et al. (2003). Preinjury emotional and family functioning in caregivers of persons with TBI. Archives of Physical Medicine and Rehabilitation, 84, 197–203.
- 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. *The Journal of Head Trauma Rehabilitation*, 24(3), 145–154.
- Allen, K., Linn, R. T., Gutierrez, H., & Willer, B. S. (1994). Family burden following traumatic brain injury. *Rehabilitation Psychology*, 39, 29–48.
- Kreutzer, J. S., Gervasio, A. H., & Camplair, P. S. (1994). Patient correlates of caregivers' distress and family functioning after traumatic brain injury. *Brain Injury*, 8, 211–230.
- Perlesz, A., Kinsella, G., & Crowe, S. (2000). Psychological distress and family satisfaction following traumatic brain injury: Injured individuals and their primary, secondary, and tertiary carers. *The Journal of Head Trauma Rehabilitation*, 15, 909–929.
- Gosling, J., & Oddy, M. (1999). Rearranged marriages: Marital relationships after head injury. *Brain Injury*, 13, 785–796.
- 20. Anderson, M. I., Parmenter, T. R., & Mok, M. (2002). The relationship between neurobehavioral problems of severe traumatic brain injury (TBI), family functioning and the psychological well-being of the

spouse/caregiver: Path model analysis. *Brain Injury*, 16, 743–757.

- Wells, R., Dywan, J., & Dumas, J. (2005). Life satisfaction and distress in family caregivers as related to specific behavioural changes after traumatic brain injury. *Brain Injury*, 19, 1105–1115.
- 22. Gill, C. J., Sander, A. M., Robins, M. S., Mazzei, D. K., & Struchen, M. A. (2011). Exploring intimacy from the viewpoint of individuals with traumatic brain injury and their partners. *The Journal of Head Trauma Rehabilitation*, 26, 56–68.
- 23. Hammond, F. M., Davis, C. S., Whiteside, O. Y., Philbrick, P., & Hirsch, M. A. (2011). Marital adjustment and stability following traumatic brain injury: A pilot qualitative analysis of spouse perspectives. *The Journal of Head Trauma Rehabilitation*, 26, 69–78.
- Wood, R. L., & Yurdakul, L. K. (1997). Change in relationship status following traumatic brain injury. *Brain Injury*, 11, 491–501.
- Kreutzer, J. S., Marwitz, J. H., Hsu, N., Williams, K., & Riddick, A. (2007). Marital stability after brain injury: An investigation and analysis. *NeuroRehabilitation*, 22(1), 53–59.
- Sady, M. D., Sander, A. M., Clark, A. N., Sherer, M., Nakase-Richardson, R., & Malec, J. F. (2010). Relationship of pre-injury caregiver and family functioning to community integration in adults with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 91, 1542–1550.
- Sander, A. M., Caroselli, J. S., High, W. M., Becker, C., Neese, L., & Scheibel, R. (2002). Relationship of family functioning to progress in a post-acute rehabilitation program. *Brain Injury*, *16*(8), 649–657.
- Sander, A. M., Maestas, K. L., Sherer, M., Malec, J. F., & Nakase-Richardson, R. (2012). Relationship of caregiver and family functioning to participation

outcomes following post-acute rehabilitation for traumatic brain injury: A multicenter investigation. *Archives of Physical Medicine and Rehabilitation, 93*, 842–848.

- Kreutzer, J. S., Serio, C. D., & Bergquist, S. (1994). Family needs after brain injury: A quantitative analysis. *The Journal of Head Trauma Rehabilitation*, 9, 104–115.
- Serio, C. D., Kreutzer, J. S., & Gervasio, A. H. (1995). Predicting family needs after brain injury: Implications for intervention. *The Journal of Head Trauma Rehabilitation*, 10, 32–45.
- Witol, A. D., Sander, A. M., & Kreutzer, J. (1996). A longitudinal analysis of family needs following traumatic brain injury. *NeuroRehabilitation*, 7, 175–187.
- Nabors, N., Seacat, J., & Rosenthal, M. (2002). Predictors of caregiver burden following traumatic brain injury. *Brain Injury*, 16, 1039–1050.
- Sherer, M., Bergloff, P., Boake, C., High, W., & Levin, E. (1998). The Awareness Questionnaire: Factor structure and internal consistency. *Brain Injury*, 12, 63–68.
- Sherer, M., Oden, K., Bergloff, P., Levin, E., & High, W. M. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *NeuroRehabilitation*, 10, 25–37.
- 35. Sander, A. M. (2005). A cognitive-behavioral intervention for family members of persons with TBI. In N. Zasler, R. D. Zafonte, & D. Katz (Eds.), *Brain injury medicine* (pp. 1117–1130). New York: Demos Publishing.
- 36. Kreutzer, J. S., Stejskal, T. M., Ketchum, J. M., Marwitz, J. H., Taylor, L. A., & Menzel, J. C. (2009). A preliminary investigation of the brain injury family intervention: Impact on family members. *Brain Injury*, 23, 535–547.

# Comprehensive Brain Injury Rehabilitation in Post-hospital Treatment Settings

James F. Malec

# Abstract

The mission of comprehensive brain injury rehabilitation (CBIR) is to assist individuals with traumatic brain injury and their close others to resume full participation in family, work, and community life. In pursuing this goal, CBIR addresses the needs of participants holistically including cognitive and metacognitive impairments, neurobehavioral dysfunction, and interpersonal and affective issues, and identifies barriers and resources in their physical and social environments. Treatment is integrated and, in its most intensive form, relies heavily on developing a positive transdisciplinary therapeutic milieu with an emphasis on group treatment. This chapter describes characteristics of participants best suited for CBIR and reviews interdisciplinary team evaluations and the operation and care of the rehabilitation team. Important components of CBIR include the development of a therapeutic milieu and therapeutic alliance, dynamic group treatment, cognitive rehabilitation, and interventions to address impaired self-awareness as well as co-morbid and premorbid conditions. This chapter describes how to provide these components in an integrated fashion in collaboration with close others and how to further integrate treatment with transitional interventions, such as, vocational rehabilitation, work and independent living trials, and resource facilitation. Methods for systematic measurement of progress and outcome both in the individual case and at a programmatic level are recommended in a continuous quality improvement model.

#### Keywords

Brain injuries • Rehabilitation • Postacute • Outpatient • Interdisciplinary • Outcome measurement

J.F. Malec, Ph.D. (🖂)

Professor and Research Director, Physical Medicine and Rehabilitation, Indiana University School of Medicine and Rehabilitation Hospital of Indiana, Indianapolis, IN, USA e-mail: jmalec@rhin.com Emeritus Professor of Psychology, Mayo Clinic, Rochester, MN, USA

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_15, © Springer Science+Business Media, LLC 2014
# Overview

The methods and concepts of holistic comprehensive post-hospital brain injury rehabilitation were originally defined through the work and writings of Ben-Yishay and Prigatano [1-3]. A consensus conference in Zionsville, Indiana in 1994 codified the key elements of this approach (see Table 1) [4]. Ben-Yishay, as well as Pamela Klonoff from George Prigatano's program and other developers of this approach to rehabilitation, participated in this conference. The Ben-Yishay/Prigatano model stressed the importance of developing a therapeutic milieu in which a highly integrated, transdisciplinary team provided neurosychologically focused rehabilitative treatment based in group process. Although goals included reduction of impairments, the primary emphasis of this form of rehabilitation is to assist the participant in reintegrating into family, community, and work life. This holistic milieu-oriented treatment is clearly and extensively described in a book by Ben-Yishay and Diller [5] which includes a number of compact discs illustrating practices and methods in actual participant groups.

At the present time, few programs exist that include all the features of Ben-Yishay's original program at the Rusk Institute in Manhattan. Due both to shrinking reimbursement and advances in rehabilitation intervention research, comprehensive programs have become more streamlined. Nonetheless, the basic principles identified during the Zionsville Conference continue to characterize current comprehensive programs. The nature of the milieu has become more varied as well. With increased interest in community-based programming, the rehabilitation team may seek to develop a more stable therapeutic milieu within the participant's own family or social network rather than within a treatment facility. In this chapter, component interventions and team interactions that characterize comprehensive brain injury rehabilitation (CBIR) will be reviewed in detail. The types of individuals best served through this approach will be described, as will methods for monitoring process and outcome. As Table 1 indicates, continuous quality **Table 1**Defining features of holistic day treatment (fromMalec JF, Basford JS [45]; based on text from Trexler LE,et al. [4])

I. Neuropsychological orientation focusing on
A. Cognitive and metacognitive impairments
B. Neurobehavioral impairments
C. Interpersonal and psychosocial issues
D. Affective issues
II. Integrated treatment that includes
A. Formal staff meetings with core team in attendance four times/week
B. A team leader or manager for each participant
C. A program leader or manager with at least 3 years experience in BI rehabilitation
D. Integrated goal setting and monitoring
E. Transdisciplinary staff roles
III. Group interventions that address
A. Awareness
B. Acceptance
C. Social pragmatics
IV. Dedicated resources, including
A. An identified core team
B. Dedicated space
C. A participant to staff ratio no greater than 2:1
V. A neuropsychologist is part of the treatment team, not just a consultant
VI. Formal and informal opportunities for involvement of significant others, including systematic inclusion of significant others on a weekly basis
VII. Inclusion of a dedicated vocational or independent living trial
VIII. Multiple outcomes are assessed, including
A. Productive activity
B. Independent living
C. Psychosocial adjustment
D. Emotional adjustment

improvement through monitoring and evaluating individual and programmatic outcomes is a key feature of CBIR.

#### Types of Participants

Comprehensive and holistic evaluation and treatment arguably characterize all high-quality rehabilitation and medical care. However, a significant proportion, if not a majority, of individuals with traumatic brain injury (TBI) do not require a CBIR day or residential treatment program.

- Limited self-awareness of disabilities
- Cognitive impairments: e.g., concentration, memory, generalization, problem-solving, initiation, reasoning, planning
- Poor communication and social skills
- · Limited emotional/behavioral self-control
- Unemployed or failing in employment
- No imminent risk of harm to self or others

Many individuals with TBI, even those with moderate to severe injuries, emerge from the acute period of recovery with reasonable selfawareness of a limited number of circumscribed deficits. Problems with memory and attention and difficulties in emotional control are perhaps the most common of these. Such participants with a few circumscribed disabilities and reasonable self-awareness will typically benefit from rehabilitation and associated medical care provided on a more limited scope (3–5 h per week) that is less costly both financially and in time demands on the participant and their family and close others [6].

On the other hand, CBIR is required in more complex cases in which a number of cognitive, behavioral, and often physical disabilities are present, interact, and are compounded by limited self-awareness and co-occurring or pre-injury conditions, such as, a history of substance abuse, psychiatric disorder or family dysfunction. Definitive research studies identifying which types of participants benefit from specific forms of brain injury rehabilitation are not available. However, based on the author's clinical experience, characteristics of the type of participant most suited for comprehensive treatment are listed in Table 2.

These more complex cases require a comprehensive approach because their disabilities frequently interact. They also require a transdisciplinary approach in which all therapists collaborate in an overall plan of rehabilitation treatment. Although individual therapists bring their specific expertise to their interventions, each must be aware of the goals and approaches of the other therapists involved and able to assume each other's roles, as needed, to keep the rehabilitation "on mission." Transdisciplinary team process will be discussed in more detail later in this chapter.

#### Interdisciplinary Evaluation

A thorough interdisciplinary evaluation will help determine who requires CBIR and who may benefit from more limited and focused rehabilitation. Ideally, this evaluation includes individual evaluations by the following disciplines: a rehabilitation physician (physiatrist) and other medical specialties as required; a clinical neuropsychologist; speech/language pathologist; occupational and physical therapists; a family liaison; and a resource facilitator. Although most participants who enter post-hospital rehabilitation are medically stable, a thorough re-evaluation of the participant's medical status by a physician specializing in medical rehabilitation is important for several reasons: (1) to determine any overlooked medical problems related to the brain injury; (2) to identify co-occurring or pre-injury conditions that may require additional treatment or special attention during rehabilitation; and (3) to identify medical risk factors and assist the participant to develop a medical life care plan. Masel and DeWitt [7] have noted that individuals with TBI may be more vulnerable to medical conditions and benefit from ongoing medical care to minimize these risk factors. Physical therapy (PT) evaluation assesses possible motoric disabilities as well as general cardiovascular fitness and the need for intervention in these areas.

A thorough neuropsychological evaluation that includes both neuropsychometric testing and a clinical interview will identify cognitive impairments as well as emotional and adjustment issues, more serious psychiatric disorders including substance abuse issues, and possible family and social concerns. Speech/language pathology evaluation focuses specifically on cognitive communication, and occupational therapy (OT) evaluates functional abilities that affect everyday activities. Both speech and OT evaluations assess the degree to which cognitive impairments contribute to functional disability in interpersonal communication and complex activities of daily living. These functional cognitive and communication evaluations are important because the ecological validity of neuropsychological testing is not perfect. That is, cognitive impairments identified on neuropsychometric testing do not always translate into functional disability; conversely, cognitive deficits that are apparent in more real life or interpersonal settings may not be apparent in the highly structured and supportive setting in which neuropsychometric testing is conducted.

Although a complete neuropsychological evaluation also touches on the participant's family and social support network, these are important enough to the long-term success of rehabilitation that a specific evaluation is critical to rehabilitation planning. In most cases, further development of the participant's network of social and practical support will be required for community reintegration. This is the role of the resource facilitator and will be discussed in greater detail later in this chapter. The assessment of the participant's network of family, social, and practical support and resources may be conducted by a social worker, family counselor, or other individual with training and experience in working with family, social and community systems. This assessment of the participant's integration in home and community life includes leisure, recreational, and work interests and activities. Ideally such evaluations are conducted by experts in these areas, i.e., vocational counselor, recreational therapist, but in some settings these evaluations may be included in the evaluations of other rehabilitation team members.

This interdisciplinary evaluation is focused on current functional abilities. Several studies [8– 10] have shown that current functional abilities are better predictors of long-term rehabilitation outcomes than initial injury severity as measured by, for instance, the Glasgow Coma Scale or duration of post-traumatic amnesia. In two studies, an initial assessment on admission to posthospital rehabilitation with the Mayo-Portland Adaptability Inventory (MPAI-4) accounted for over 60 % of the variance on progress and outcome, also assessed by the MPAI-4 [11, 12].

# **The Rehabilitation Team**

Rehabilitation teams may be organized in several ways: multidisciplinary, interdisciplinary, and transdisciplinary. A *multidisciplinary* team is one in which each of the team members work independently with the participant in their area of expertise and do not coordinate their therapeutic activities or treatment plans. Multidisciplinary teams are perhaps most common in traditional outpatient rehabilitation settings in working with participants who have a small number of clearly defined goals. For instance, a participant poststroke will see the Speech/Language Pathologist for dysarthria, PT for ambulation, and OT to improve use of the affected hand.

In an *interdisciplinary* team, each member works with the participant in their area of expertise but in a coordinated manner and with an integrated treatment plan. Each team member is aware of, and reinforces the goals and methods of, other team members. Interdisciplinary teams are most appropriate in working with more complex or acute participants when the objectives of each discipline overlap and are affected by those of other team members. Inpatient rehabilitation teams are most commonly organized in an interdisciplinary fashion.

In a transdisciplinary team, members not only work in a coordinated manner from an integrated treatment plan and reinforce each other's efforts, but are also able to temporarily assume each other's roles. In other words, the PT can assume the role of the psychologist if a participant begins to exhibit inappropriate anger in the PT session, and the psychologist can remind the participant about the current parameters of the aerobic conditioning program that the PT has prescribed. To be maximally effective, the organization of the rehabilitation team delivering CBIR must be transdisciplinary. Being able to assume each other's roles requires a good deal of information sharing among team members, exquisite confidence in one's own and each other's professional abilities, and a high degree of trust among team members.

The ability to assume each other's roles is critical because most participants with brain injury who require CBIR have severe difficulty in acquiring and generalizing new learning. Individuals with significant cognitive impairment need to have learning experiences in close temporal proximity to their expression of problematic behaviors. For instance, when a participant exhibits inappropriate behavior, it will not be effective to note this and bring it to the attention of the psychologist for discussion at a later point in time. The participant's behavior and its negative consequences must be addressed in the hereand-now. Then alternative, appropriate behaviors must be prompted or coached, and the more positive consequences of these behaviors identified. This type of training in the here-and-now can be applied to every type of cognitive and behavioral problem. Arguably new learning of this nature is most effective in assisting individuals with problematic behaviors to learn more adaptive behaviors whether or not they have TBI. However, for individuals with TBI, organizing new learning in this way is critical because of their limited capacity to remember and to generalize new learning.

# Operation of the Transdisciplinary Team

Not uncommonly rehabilitation team members exhibit the typical human characteristic of protectiveness of their "territory," i.e., their disciplinary knowledge and skills, and anxiety about working outside of their comfort zone. While these types of feelings are understandable and normative for human beings, a maximally effective transdisciplinary team is able to function beyond this level of self-interest and keep their eye on the mission. In this regard, the transdisciplinary team functions like other high performance teams, such as, elite surgical teams or military squads. Raemer [13] in Simulators in Critical Care and Beyond recommends four routines of military commando teams for emulation by high performance medical teams: (1) practice, (2) briefing, (3) debriefing, and (4) celebration.

*Practice* is essential for the efficient and effective operation of a high performance team. Nonetheless, busy schedules can restrict the amount of time that teams have to practice their roles as a team with a given participant. In order to develop the capacity for the members of transdisciplinary teams to assume each other's roles, it is helpful for the team to discuss and role play the appropriate response to critical participant events. For instance, team members might review how to respond to expressions of anger from a participant, or how to respond to memory failures. In the former instance, the psychologist may be the primary team member to guide colleagues through the appropriate responses to the participant. In the latter, the occupational or speech therapist who is primarily responsible for organizing the cognitive rehabilitation program may be the primary guide. However, in the end, each team member should be able to, at least temporarily, respond as capably as a psychologist to expressions of anger and as ably as the memory expert on the team to memory failures.

*Briefing* refers to preparing for the mission, or in the case of rehabilitation teams, the coordinated treatment program with a given participant. In rehabilitation settings, this means designing and reviewing the integrated treatment plan. Documentation of a rehabilitation treatment plan is required in most rehabilitation settings. Regular review and appropriate updating of the treatment plan may be challenged by busy schedules of the treatment team members but is essential to assure high team performance.

Debriefing refers to regular examination of the functioning of the team in completing its mission with the participant. While most rehabilitation teams have team conferences on a regular basis, these conference are often focused on participant progress. The most informative debriefing sessions include not only participant status and performance (i.e., to what degree are the goals of the mission being accomplished) but also careful examination of the methods that are being used. What is working and what is not working? How can the team be more effective in working with this participant both as individual practitioners and as a team? A high performance CBIR team needs to have a formal debriefing conference three to five times a week. The objective of these debriefing conferences is to examine in what ways specific interventions as well as their overall plan with specific participants is effective as well as ineffective, and to revise their transdisciplinary rehabilitation plan to improve progress and outcome. Debriefing conferences typically occur without the participant present. The focus is primarily on team process and function and on participant status and progress only as these represent the functioning of the team.

 Table 3
 Team communication ground rules

- 1. We will periodically review our effectiveness as a Team
- 2. We will not judge, challenge, nor evaluate an idea until we hear or understand the <u>whole</u> idea
- 3. We will attack problems, not people (each other)
- 4. We will disagree without becoming disagreeable
- 5. Once the Team reaches a decision, and I have the opportunity to be heard, I will support the Team decision 100 %
- 6. We each participate in discussions, fully and openly. We do not use silence as a weapon or as a defense
- 7. We deal with our conflicts and frustrations directly and promptly
- 8. We feel free to bring up problems and invite possible solutions
- 9. We say nothing about any third party that we would not say if that person were present

10. We respect each other's work, tasks, and	
contributions without regard for titles or status	,

Finally, effective, high performance teams regularly *celebrate* their successful missions. Central to the concept of celebration is tying the celebration to a specific accomplishment. Some CBIR teams formalize this celebration with a graduation party that includes the participant who has successfully completed the program, their close others, and the treatment team. However, to maintain *esprit de corps* and avoid burnout, even minor victories merit celebration.

#### **Care of the Team**

Providing transdisciplinary CBIR can often be challenging and stressful. The participants selected for this type of intensive rehabilitation typically lack self-awareness, are disinhibited and intermittently aggressive, and can split the team through dramatic and frustrating behaviors. Briefing and debriefing sessions provide opportunities for team members to support each other, particularly team members who may be showing signs of stress and burnout. The team leader, who is often a neuropsychologist, has a primary role in supporting the healthy psychological functioning of the team.

Explicit ground rules for team interactions can also be helpful in avoiding inappropriate exchanges and harmful splitting among team members under stress. Disagreements and differences of opinion are healthy and constructive in brainstorming approaches to challenging participant behaviors. However, interactions with participants must be unified and consistent within the team. In the CBIR at Mayo Clinic, we developed a set of guidelines (Table 3) for team interactions. These were posted in the team conference area, and not infrequently referred to during heated team discussions.

#### **Transdisciplinary Treatment**

#### The Therapeutic Milieu

A fundamental concept underlying CBIR is development of a *therapeutic milieu*. In a nutshell, a therapeutic milieu is a treatment environment in which virtually every action and interaction has a therapeutic value, that is, assists participants in accomplishing the goals of treatment. In addition to formal treatments, the therapeutic value of all other activities in the treatment setting, such as, informal conversations among participants, with staff, with family/close others; going to lunch; and formal and informal outings, is recognized and reinforced. The rest of this section examines how the various elements of CBIR create a therapeutic milieu.

#### **Group Therapy**

Most CBIR programs provide treatments in groups. Providing therapy to more than one participant simultaneously creates efficiency and lessens the personnel costs of providing treatment. However, this is not the primary rationale for group therapy. Group therapy also improves clinical care. Developing a positive dynamic for participants in a therapeutic group can be a powerful intervention to develop self-awareness, reinforce effort and progress, and create a therapeutic milieu.

Providing therapy in a group is not necessarily group therapy. In less effective groups, therapy is provided to one person in the group at a time. Others in the group may benefit from observation of the therapeutic process and serve to encourage and reinforce the intervention. However, what is missing in this scenario is the use of the powerful group dynamic. In a relatively short period of time, every group develops an identity that goes beyond the identity of the individual members. Each member finds a role in the group that is consistent with the identity of the group. Group identity and the roles can be positive or negative. A skilled group therapist will work with the group to create a positive and constructive milieu.

Examples of common roles in a group are: the rational one (who tries to be the voice of reason), the helper (who tries to be supportive), the challenger (who tends to question or challenge the "common wisdom" of the group or the leader), and the quiet one (who has difficulty speaking up in the group). This list is not exhaustive; roles in the group can be many, highly nuanced, and changing. These examples illustrate, however, how roles may be positive or negative. For instance, the rational one may in fact only think they are being rational, while they advance idiosyncratic ideas. The challenger can play an important function in making other group members think about recommendations and suggestions but, without a good group leader, can easily turn into a naysayer who enjoys the attention afforded to a "rebel."

Typically, individuals find roles in the group that are consistent with their interpersonal style. In a psychotherapeutic group, getting members to reflect on the roles they play in the group, and how and when these roles are effective or ineffective, creates important learning experiences to improve the effectiveness of their interpersonal style. For instance, the quiet one may be a very good listener, but needs to learn to be more assertive to share the perceptions that they have for the benefit of the group and themselves. The helper offers valuable support to others, but may also feel, at times, that they are "always giving" (which they are). Like the quiet one, the helper may be able to learn through practice in the group to be more assertive in getting their own needs met as well as in helping others.

Most CBIR groups, however, are not fundamentally psychotherapeutic but are focused on other goals, such as, developing cognitive, social, or functional skills. In these other CBIR groups, understanding and developing the identity of the group and the roles of the members is critical to using the group process to accomplish the goals of the group. For example, in a cognitive group in which the primary goal is to help members develop and use memory notebooks, a positive group process can be a powerful tool. With skilled guidance and reinforcement from the therapist, the rational one will explain the sense of using a memory notebook; the challenger will question this and voice the objections that others have-so these can be addressed; the quiet one may need to be drawn out, but will often be the swing vote in the process to keep things moving in a positive direction; and the helper will reinforce everybody for using the memory notebook. This quick summary is of course an oversimplification but may give the basic idea of how group process can be used to accomplish the goals of any group, not just psychotherapeutic groups.

A basic premise underlying group process is that the members of the group will respond to their peers more readily than to therapists and that the guidance and reinforcement that they receive from each other is more powerful than that of a therapist. Like most people, people with TBI tend to listen most closely to their peers, to those people who they identify are most like themselves and who they feel share their life experience. The therapist's skill is required to manage the group process and to keep its energy focused on moving its members positively toward accomplishing their goals.

#### **Therapeutic Alliance**

Therapeutic alliance is the bond of trust and collaborative working relationship that develops between therapist and participant in effective therapy. The concept of therapeutic alliance originally developed through studies of psychotherapy where it was identified as a "necessary but not sufficient (NBNS)" condition for therapeutic change [14, 15]. That is, a therapeutic alliance does not in and of itself produce positive behavioral change; however, change will not occur or will occur only minimally if a bond between therapist and participant is not present. In recent years, therapeutic alliance has been increasingly studied in brain injury rehabilitation and has been found to have a similar positive effect on outcome [16–18]. Although therapeutic alliance often involves feelings of liking and affection between participant and therapist, it is more than this. Therapeutic alliance is "mission-oriented" in that both therapist and participant see themselves as a team that is working collaboratively to accomplish the participant's goals. The participant develops trust in the therapist's treatment recommendations and feedback; the therapist also develops trust that the participant is dedicated and committed to the therapy despite the inevitable ups-and-downs of therapeutic progress.

#### Addressing Self-Awareness

Self-awareness of disability is present in a significant minority of cases of moderate to severe brain injury. Most likely because of cognitive impairments affecting both their ability to conceptualize as well as to remember changes in themselves due to their injuries, participants with impaired selfawareness (ISA) act as if they are the same people they were before their injury. Sherer and Fleming present a thorough discussion of ISA in this volume. The focus here will be on addressing ISA through CBIR and the therapeutic milieu.

Most participants selected for CBIR have some degree of ISA, and ISA is often the overarching disability that will most dramatically interfere with community reintegration for them. ISA can be effectively addressed through CBIR and typically cannot be addressed through more specific individual therapies, for instance, cognitive rehabilitation alone. Participants with no selfawareness of disability cannot be engaged in rehabilitation. In their minds, they are unimpaired; so rehabilitation is of no value. However, most participants are able to identify a specific problem for which they will acquiesce that they may need a little help, often with the encouragement of family or close others. Addressing the identified disability for participants with ISA is the hook to engage the participant; the CBIR treatment plan,

however, can be more comprehensive and include addressing ISA as part of the rehabilitation program. Working to develop self-awareness is a delicate operation of balancing feedback with support. The trusting, collaborative working relationship that characterizes *therapeutic alliance* is essential to this work. The *therapeutic milieu* is also particularly important to the development of accurate self-awareness after TBI because the development of more accurate self-appraisal is most effectively accomplished if appropriate and consistent feedback and support are provided throughout the day rather than only in a few therapy sessions dedicated to this process.

ISA is challenging to address and may be complicated by pre-injury personality tendencies to respond to stress with denial or support from close others who are also coping by a degree of denial. In almost all cases, it is unreasonable to expect that self-awareness that is impaired due to brain injury will ever completely return to normal after a brain injury. Goals for CBIR should be to improve self-awareness to the degree that the participant can (1) participate effectively in rehabilitation, (2) set realistic goals for rehabilitation and community reintegration, and (3) not engage in behaviors in which they are at risk for harm because of their disabilities. Nonetheless, CBIR provides an effective means to achieve these goals through interventions described below.

Education about brain injury generally and specific to the individual. Explaining the nature of brain injury in general and how it has affected the individual with TBI specifically typically will not in and of itself greatly improve ISA. However, this knowledge is a NBNS condition for improving ISA. A participant cannot be expected to understand how their brain injury has affected them-particularly the more subtle effects-if no one has taken the time to thoroughly explain this to them. General education about brain injury is provided in CBIR groups with easily readable and understandable written material provided as a reference. Education specific to the individual participant can also be provided in a group context, including review of neuropsychological test results, neuroimaging, and how the two connect.

It may be psychologically less stressful to learn about the effects of brain injury from review of a peer's case. Group members also provide mutual support to confronting the stressful realities of brain injury and the sense that none of the members "are alone" in struggling with the effects of brain injury. Educational information will likely need to be repeated several times over the course of a CBIR program—as the participant's selfawareness improves, they will become more able to assimilate this information.

Family/close other education and participation. Brain injury education should also include the participant's close others. Close others also often have very limited knowledge about how the brain works and how it recovers from injury. Misconceptions about brain function and recovery may lead to unrealistic expectations. As mentioned previously, close others can also have biased and inaccurate assessments of the participant's status. In order for any ISA intervention to be successful, it is very important that the rehabilitation team and the participant's close others are "on the same page" regarding the participant's abilities, goals, and expectations for recovery. This is important so that the participant's close others can become allies of the CBIR team in reinforcing more accurate self-assessments and realistic expectations by the participant as well as appropriate progress toward realistic goals. The most common situation is one in which the rehabilitation team and the participant's close others generally agree on the participant's status and the participant tends to minimize their disabilities and their impact on their activities. However, the author has observed every possible variation of discrepancy in the appraisal of the participant's current abilities by the participant, close others, and the rehabilitation team. The MPAI-4 (to be discussed in more detail later) is designed primarily as an evaluation and outcome measure to be completed by consensus of a rehabilitation team. Nonetheless, during the initial interdisciplinary evaluation, we have routinely asked participants and a close other to complete the MPAI-4 independently of each other. Comparing these self- and close other assessments with the assessment of the CBIR team on

the same measure gives a clear idea of where areas of agreement and disagreement are present regarding the participants' abilities, adjustment and community reintegration. We have found it more productive to know from the beginning where we agree and where we disagree, rather than to be surprised by these discrepancies in perception or expectation once a rehabilitation treatment plan has been set in motion. Sometimes disagreements are not easily or quickly resolved. Our approach has been to begin focusing on areas in which there is relatively good agreement about the need for rehabilitation and gradually work on coming together in areas where expectation of needs are more discrepant.

Structured repeated learning experiences with feedback. While education may be NBNS for improving self-awareness, repeated exposure to situations in which the participant's disabilities and their consequences are made apparent to the participant is very likely the active ingredient in treatment to improve self-awareness. CBIR offers numerous opportunities each day for this type of learning to occur. These types of experiences are potentially very stressful to participants-no one likes to be confronted with their failures or mistakes. Consequently, very direct or harsh feedback to participants about their disabilities, failures, and mistakes is usually not constructive. To the contrary, confronting participants about, for example, their memory problems, is most likely only going to reinforce denial and resistance. A more productive approach is to structure these experiences as supportive learning opportunities. It is not uncommon, however, for CBIR team members to disagree about how direct and confrontational feedback to a given participant should be and can lead to heated debate that threatens to split the team. Agreeing on the most constructive approach with a given participant will appropriately occupy many briefing/debriefing sessions and require skill and sensitivity from the team leader to help team members deal with their frustrations in working with participants with severe ISA.

*Peer feedback.* Feedback may take many forms: feedback from therapists, results of objective tests or measures, recorded video. However, the

most effective feedback is from peers. Like most of us, people with TBI tend to listen most closely to people who they feel are much like themselves. Direct confrontation is also better tolerated when delivered by peers than when delivered by authority figures like therapists. Shrewd and skillful management of group process and dynamics and the therapeutic milieu by the CBIR team will result in many constructive opportunities for this type of feedback.

Individual and group psychotherapy. The development of more accurate self-appraisals is a double-edged sword. More accurate self-appraisal will lead to more appropriate goal setting and avoidance of activities in which current disabilities will frustrate success or put the participant at risk. However, more accurate self-appraisal can also lead to feelings of discouragement, depression, anger, and other emotional reactions as the participant begins to recognize that life has changed because of brain injury and that some activities and goals that they had prior to the injury may be forever out of reach. For this reason, psychotherapeutic interventions to address feelings of loss and to develop coping skills are another essential component of any intervention to address self-awareness. As self-awareness increases, sometimes depression can become marked, and intensive treatment, including medication should be considered.

In this author's experience, the transition between increased self-awareness and reactive emotional distress is not clearly staged. That is, the participant does not suddenly develop selfawareness and then become depressed or angry. Rather the process is one in which self-awareness improves a little; negative emotional reactions occur; and the participant defends against these unpleasant feelings with a degree of minimization and denial. With psychotherapeutic intervention, these defensive reactions diminish and self-awareness improves a little more-only to begin the cycle again. Support and feedback of peers in psychotherapy and other CBIR groups can be very effective in helping participants to work through these cycles of psychological growth.

#### **Cognitive Rehabilitation**

Interventions to improve cognitive function are an integral part of CBIR programs. The most common targets for cognitive rehabilitation are attention, memory, problem-solving, and goal-setting. Because cognitive abilities interact, a thorough neuropsychological evaluation is essential to planning a targeted cognitive rehabilitation program. For example, almost all individuals presenting for CBIR (or their close others) will report that they have "memory problems." However, for many, the problem is not so much with storing and retrieving new information but with attending to the information when it is presented so that it can be stored in memory. Participants whose primary cognitive disability is attention will benefit from different cognitive rehabilitation methods than those whose primary problem is long-term storage and retrieval. For others, difficulty in organizing new information for memory storage may be the primary problem. These individuals typically also have great difficulty organizing other aspects of their lives and consequently, organization may be the primary target for intervention and memory only a secondary target.

The functional impact of cognitive impairments is also important to evaluate. While cognitive impairments may be a very significant frustration and impediment to community reintegration for many people with TBI, this is not true for all. Some individuals have learned to compensate for low average or mildly below average cognitive abilities throughout their lives, and have found work and other activities where strong cognitive abilities are not required. Such individuals often do not require intensive cognitive rehabilitation in order to re-engage with their communities—regardless of the results of their neuropsychological testing.

A number of evidence-based methods for rehabilitation of various cognitive functions have been identified through a series of reviews [19–21]. Haskins and colleagues [22] have recently published a manual that clearly describes in practical terms how to apply these techniques in practice. The Haskins book also provides a number of exercise guides and materials for use by therapists. A

recent volume by Sohlberg and Turkstra details the most effective processes for organizing cognitive rehabilitation interventions. Cognitive rehabilitation techniques are reviewed in this volume as well (see Chaps. 9–11). The interested reader is directed to these other sources for a more in-depth treatment of this topic. The focus here is on integrating cognitive rehabilitation into the therapeutic milieu.

Attention Process Training (APT). Originally developed by Sohlberg and Mateer [23], APT involves practice in which the complexity in auditory and visual modalities of the foci of attention is gradually increased. In their volume on cognitive rehabilitation, Sohlberg and Mateer [24] also describe a number of exercises in which attentional focusing and shifting, dividing attention, and shifting attention can be practiced in everyday life. Similar exercises can be practiced in a group setting and throughout the day in the CBIR therapeutic milieu.

Group therapy and the milieu also provide opportunities to address emotional reactions that may interfere with attention. Depression, anxiety, anger, and worry all interfere markedly with the range of attentional functions. This is true for the normal population and doubly so for individuals with TBI. In some cases, the frustration of loss of attention may set off strong negative emotions and create a downward spiral for the participant in which the negative emotions lead to further difficulty regulating attention-leading in turn to increased frustration, anxiety, dysphoria or angercreating further loss of attentional control. Through cognitive-behavioral therapy, individuals who experience such disruptive emotional reactions learn to identify the thought processes that lead to these negative emotions and interrupt them with more constructive self-talk. In a group milieu, other participants and therapists can assist by prompting this kind of self-talk when they observe a loss of attention accompanied by emotional upset.

The Memory Notebook. Development of a "memory notebook" for each participant is a standard component of CBIR. These notebooks should be individualized to the needs of each participant with sections designed to help organize their schedule and make frequently used information readily accessible (see Chap. 9 for further detail). Although the "memory notebook" appears to be almost universally used as a name for this tool, one of the participants with whom we worked challenged this. He made the point that the use of daily planners, smart phones, and other memory assists has become ubiquitous in the normal population, and asked why these aids should be called something different when used by people with TBI. In fact, his point is well taken. Simply referring to the "memory notebook" as what it is, i.e., a calendar, planner, or smart phone, may normalize the experience of its use for the person with TBI and help increase its acceptance.

As alluded to above, many participants experience some resistance to the use of an external memory aid. Very likely these aids serve as reminders of their injuries and disabilities, particularly if they were not in the habit of using such aids before their injury. Addressing such emotional issues in group therapy and in the therapeutic milieu is critical to remove these obstacles to developing individualized aids to assist participants to compensate for their impairments in organization and memory. Group treatment and the milieu also provide many opportunities to reinforce the use of memory aids through planning and scheduling group and individual activities, as well as opportunities to bring the consequences of either using or not using these aids to the awareness of participants.

*Systematic Problem-solving*. Teaching simple, systematic approaches to problem-solving is important to help participants learn ways to analyze and prioritize possible solutions for life problems. Teaching problem-solving also works well in groups since it closely resembles a brainstorming exercise. Input from other group members and therapists may be very helpful to participants in identifying, selecting, and prioritizing various solutions to problems. As with other cognitive rehabilitation interventions, identifying and addressing emotional issues that may arise during a problem-solving exercise are just as important as teaching participants the mechanics of a systematic approach to problem-solving.

Goal Management Training (GMT). Goal identification and attainment is an essential element of any rehabilitation program and a critical skill for success in life. However, the capacity to set and systematically pursue realistic goals is often diminished due to brain injury. GMT [25] has emerged as an evidence-based method to develop these skills. GMT can be applied as a method to engage the ownership of program goals by CBIR participants. GMT can also help participants develop goals that reach beyond the program and are useful in structuring their current and future lives. In all forms of rehabilitation, treatment goals are best set collaboratively by participant and therapist. Agreeing on goals, however, is challenging with individuals who lack awareness of their impairments and of the impact of these impairments on their functional abilities and activities. The process of developing realistic and attainable goals, both within and beyond the CBIR program, is integral to building more realistic and accurate self-appraisals and self-awareness of impairments.

Perhaps even more so than in other types of cognitive rehabilitation, the goal setting process frequently precipitates strong negative feelings from participants, as they begin to recognize (with feedback from their peers as well as from therapists) that goals that they had prior to their injuries are no longer realistic because of the impairments resulting from their injuries. In many cases, even repeated feedback will not be sufficient to convince CBIR participants that former goals are no longer realistic-they will have to try themselves out in pursuing these goals in real life structured independent living or work trials. This process will be discussed later in the chapter, as will a formal process for program goal identification and monitoring progress and achievement: Goal Attainment Scaling (GAS).

# **Social Communication Skills Training**

It has long been recognized that, while basic language abilities are often spared in frontal lobe injuries, these types of injuries frequently result in dramatic impairment in other types of communication skills. These other skills, such as turn taking in conversation, sequencing, gestures and facial expression, and active listening, have become known as *pragmatic communication* skills. These skills comprise the array of nonverbal and social interaction skills that support or enhance communication through language. There is good evidence that these pragmatic social-communication skills can be developed through group treatment that includes instruction, guided rehearsal, personal and videotaped feedback [26]. A social communication group is an important feature of CBIR. In addition, social communication skills are other targeted skills to be practiced and reinforced in all interactions in the therapeutic milieu.

#### **Behavior Management Training**

The focus of CBIR is on developing self-awareness and self-management skills. Participants who have a very limited capacity for self-awareness and self-management (at least initially) may be inappropriate for outpatient or community-based CBIR. Such participants may be more effectively treated in a residential setting in which a carefully controlled program of *applied behavior analysis* can be consistently implemented. Such a program carefully orchestrates environmental stimuli and behavioral consequences to assist individuals in gaining better control over problematic behaviors [27].

Nonetheless, some elements of applied behavior analysis may be beneficially introduced into an outpatient or community-based CBIR program. Participants can learn and be assisted by others within the therapeutic milieu to identify stimuli that reliably precipitate problematic behaviors. For instance, does discussion of the participant's memory problems usually make them angry or withdrawn? Do particular activities or interactions tend to lead to expressions of anger? As these stimuli are identified, other stimuli-typically more constructive self-talk-can be prompted. Other aids can be introduced to prompt constructive and rewarding behaviors-ranging from a simple label on the participant's planner that reminds them to "Stop and Think" to more elaborate prompts contained in a planner, schedule, or smart phone.

Reinforcers of problematic behaviors can also be identified and examined. What is the payoff when an individual gets angry about their memory problems? Does it simply feel good to discharge the frustration? Or does it also get them attention or sympathy from others or get them out of the difficult work of developing methods to compensate for these problems? As reinforcers of problematic behaviors are identified and reduced, more constructive behaviors and more positive naturally occurring reinforcers can be introduced. For instance, teaching a newer participant about using a daily planner effectively may be a more gratifying kind of attention than the kind of attention elicited through raging about having to use a daily planner.

Ultimately, the CBIR participant will become an active participant in identifying, analyzing and modifying the stimulus–response-reinforcer sequences that form the core of behavior management training. However, initially in cases in which self-management and self-awareness are more limited, the process may begin with therapists taking more responsibility for regulating stimulus– response-reinforcer dimensions and gradually transferring this control to the participant.

# Vocational Interventions and Resource Facilitation

Treatment of the impairments and disabilities that occur after TBI set the stage for return to work. However, it is a mistake to assume that reduction in impairment and disability alone will assure a successful return to work. Return to work is a separate step in the process of CBIR that requires a specialized focus and intervention. Reviews of naturalistic and controlled studies [28, 29] reveal that without specialized vocational services, less than 30-40 % of individuals with moderate to severe TBI successfully return to work; whereas, with specialized vocational services, 60-70 % or more can maintain employment in the community [28, 30–32]. Supportive employment services (such as job coaching, job shadowing, work trials, environmental adaptations, employer and co-worker education and work peer support) are typically required in work reintegration after TBI. In most cases, these supportive services can be discontinued over the first year following initial vocational placement.

Buffington and colleagues [33] outline the fundamental features and key elements (see Table 4) of what they termed to be a "medicalvocational case coordination system." In many ways this is a "whatever it takes" model in which a designated service provider assists the individual with TBI and their close others to develop a network of medical, rehabilitation, and community-based supports and services that assist them in obtaining and maintaining employment. More recently this type of intervention has been termed "resource facilitation." Although the target of resource facilitation is often return to work, Trexler et al. [32] demonstrated in a randomized controlled trial that this model not only almost doubles return to work rates compared to controls but also improves community reintegration more generally.

For complex cases requiring CBIR, the process of vocational reintegration is typically and best begun while the individual is still actively receiving rehabilitation. This allows individuals to receive more focused therapy in areas that emerge as particularly critical to return to a specific job. Identification of how therapeutic activities and compensation techniques are important for return to a specific job also increases the relevance of these activities for participants and their motivation. The CBIR program provides a safety net for individuals during initial work trials. Because of ISA, many CBIR participants overestimate their work skills and fail to appreciate the way disabilities secondary to TBI will interfere with their return to work. In many cases, no amount of discussion, feedback, and simulation in a clinical setting will convince these individuals that they may be unable to successfully perform in a job that they held previously. In such cases, a work trial in which the individual has the opportunity to try themselves out in a job very similar to their previous or desired job is the only way to help them to improve their self-awareness.

The risk of such work trials is that the participant will be demoralized and even become

Fi	undamental features				
•	Resource facilitator to assist in developing:		• Early intervention		Family/significant others, employer, and coworker education
	<ul> <li>Self-directed plan</li> </ul>		<ul> <li>Work/independent living trials</li> </ul>		Medical, rehabilitative,
	<ul> <li>Network of medical center and community services</li> </ul>	•	Temporary or long-term supports and coaching		independent living, and vocational intervention
K	ey elements				
•	Develop a comprehensive plan that addresses a range of issues:	•	Focus early on community and vocational reintegration	•	Provide reasonable accommodations before
	<ul> <li>Sleep and nutrition</li> </ul>	•	Identify residual impairments that may interfere with vocational reintegration and refer for appropriate medical rehabilitation services		placement
	<ul><li>Fatigue and activity tolerance</li><li>Mood</li></ul>	•	Integrate real-life goals with rehabilitation therapy goals	•	Provide education to family/ significant others, employers, coworkers, and community service providers
	<ul> <li>Substance use</li> </ul>	•	• Use real-life and on-the-job evaluations to gather the best information about a person's skills and need for further training		Provide coaching and
	<ul> <li>Cognitive limitations and compensation</li> </ul>				training for individual served to become their own advocate
	<ul> <li>Social-communication needs</li> </ul>	eds • Provide ap	Provide appropriate support	•	Clearly identify a resource
	- Social and physical environment		during evaluations		person to contact for questions and concerns
•	Provide a smooth transition from medical to community-based services	•	Provide continued training and support after placement including coaching and extended real-life trials		Provide regular, frequent follow-up after placement
•	Facilitate communication among family/significant others, community agency and volunteer services, and medical rehabilitation services to develop a team approach				

 Table 4
 Fundamental features and key elements of resource facilitation

depressed by their failure to be successful on the work trial. However, if skillfully and supportively managed by the team, these experiences can be critical to developing more accurate selfawareness. The team's job is to help the participant see failure in a work trial as a learning experience rather than as a failure experience. The team helps the participant analyze what features of the job presented insurmountable obstacles and what features were within their competencies, and then steers further job search toward occupations in which the participant can use his or her strengths and minimize his or her weaknesses. In many cases, several brief work trials of 1-2 weeks are required in order to develop sufficient self-awareness and assist the participant to find a niche in the world of work where he or she can be successful.

# **Work and Independent Living Trials**

Practice in real life situations is critical to successful outcomes for CBIR in most cases. These trials allow participants to assess their abilities in a concrete way. Because of cognitive impairments in abstraction and generalization, many CBIR participants are unable to recognize how their disabilities will interfere with community integration in a conceptual way. They have to experience the consequences of their disabilities in real world settings. As mentioned previously, these supported trials can be developed to assist participants to become more aware of their strengths and weaknesses in the world of work. Supported trials can also be developed to identify assets and limitations in independent living, including use of transportation, shopping, and leisure time activities. No matter what the setting, the goal of these real life trials is to allow the participant to try themselves out and see for themselves whether they are up to the tasks required.

This is not, however, a matter of "throwing them in at the deep end and seeing if they can swim." To the contrary, real life trials are carefully orchestrated so that the participant indeed has to accomplish the tasks required in the setting with limited assistance but also has support from one or more members of the CBIR team to help them avoid any disastrous consequences. The CBIR team's role is also to help participants identify their successes and failures and use these observations to be more successful. A better appreciation of assets and limitations in various real life settings should also lead to (1) identification of required environmental adaptations, (2) more focused therapy in areas where this may be helpful to improve success, (3) identification of required social and community supports, and (4) development of more realistic goals.

# Family/Close Others Involvement and Intervention

A supportive family or well-established social support network can be an unparalleled resource for the CBIR participant in approaching the goal of community reintegration. On the other hand, an unsupportive or dysfunctional family or support system can be an unparalleled liability. Some CBIR programs require involvement of a family member or close other. However, it is probably better advised to do an evaluation of the family prior to making a decision regarding their level of involvement. Sander et al. [34] reported that 25-33 % of families with a member who has a brain injury are experiencing significant dysfunction at the time of their injury, regardless of socioeconomic background. This finding indicates that not all the distress that families experience after TBI is in reaction to the injury. In a substantial minority of cases, family distress is also due to factors that predate the injury and may be longstanding. Such families may need more intensive intervention than the typical education and support that is provided to close others as part of rehabilitation. Families with longstanding and complex pathology will require intensive and specialized family intervention. If these very dysfunctional families are not receptive to intervention to address issues that will markedly interfere with the participant's rehabilitation, the best strategy may be to help the participant extract themselves from the dysfunctional family system and assume a more independent lifestyle.

In other cases, participants from very healthy families may be at an age (late adolescence/early adulthood) in which they were already moving toward a lifestyle that was more independent of their family of origin. These individuals were in a process of separation and individuation as they began to assume the role of an independent and self-sufficient adult. However, their injuries cast them back into a more dependent role in the family. In some cases, this more dependent role may be necessary and because of severe disabilities, the individual will always need to be dependent on someone else, like a family member. Parents of young adults with TBI may reassume the role of care-giving parent for a period of time. In these cases, stress will re-emerge as the parents age and enter a time of life where they are no longer able to care for their child because of their own health problems and associated disabilities. In such cases where long-term dependency and support is projected for the person with TBI, planning should begin early for transitioning care to someone else as the parents age.

In other cases, late adolescents and young adults may realistically choose to continue to pursue their quest for independent adulthood despite the disabilities that resulted from TBI, and may request that their families of origin not be involved in the rehabilitation process—even though their families may very much want to be involved. The wishes of the participant must be respected in such cases; however, counseling with the family and participant to make this process of separation and individuation a healthy and supportive one is recommended. In cases in which a spouse needs to assume a caregiver role with a previously independent adult, the relationship may be severely strained. Counseling for the spouse may assist them in coping and with decision-making regarding whether they can realistically assume this role. In some cases, a mutually satisfying marital relationship can continue; however, in others, the best choice may be for the care-giving role to be transferred to someone else.

As can be seen from the preceding discussion, involvement of family and close others in CBIR can involve a complicated decision-making process, requiring consideration of multiple factors. Members of the CBIR team may disagree among themselves regarding the level of involvement that is appropriate in a given case. As in all aspects of CBIR care, the goal is for rehabilitation team members to come to consensus along with the participant and family/close other on this issue and to support each other in pursuing the agreed upon plan. More detailed information on the role of the family in rehabilitation can be found in the chapter by Sander in this volume.

# Environmental Assists and Modifications

Adjustments to or enhancements of the physical and social environments in which the participant will live and work can include (1) external aids, (2) cues and prompts, (3) interpersonal supports, and (4) alterations that improve accessibility and engagement. These types of interventions are highly individualized and are limited only by the creativity of the therapist in identifying environmental modifications that are both acceptable and helpful to the participant. External aids, for instance, may include a notebook or day planner to assist memory or a smartphone or other electronic device to accomplish the same thing. Generally, electronic aids are most helpful to individuals who have used these devices prior to their injuries and are familiar with the procedures involved in their operation. To the contrary, participants who do not have previous experience with electronic aids are likely to find them frustrating.

At a less complex level, a system of prompts and cues to remind participants of routine activities, or even a more general reminder to "stop and think," will help participants stay on track as they move through their day. The rule of thumb in developing a system of cues and prompts is that "less is more." That is, a small number of carefully considered cues or prompts may be extremely helpful; whereas, a large number will be overwhelming. Other people (e.g., family members and close others, co-workers) can be enlisted to provide prompts, reminders, and coaching to the participant. In enlisting the support of other people, their roles should be clearly specified and they should be given the message that they can assist the participant but are not responsible for the participant. Being able to occasionally receive a reminder or a bit of coaching from a co-worker can be critical to a person with TBI's success in a job. However, if the person with TBI becomes very dependent on the co-worker, this will decrease the value of the person with TBI as an employee and run the risk of creating a sense of burden for the co-worker.

Common accessibility modifications (e.g., large print, ramps, ergonomic seating) that improve accessibility for individuals with physical limitations may also apply to individuals with TBI who not uncommonly have also experienced physical injury, disability, or chronic pain. Just as importantly, the environment should be carefully examined for features that may be distracting or create discomfort for the person with TBI, such as bright or distracting lighting, a high level of ambient noise, and unpredictable changes in the amount of movement and stimulation. Elimination or reduction of such environmental features can greatly enhance the person with TBI's ability to function in that environment.

#### Medication

Psychoactive mediations can be helpful to progress in CBIR. A thorough review of the application and effectiveness of such medications is beyond the scope of this chapter. The interested reader is referred to chapters by Meythaler [35] and Arciniegas [36, 37] in *Brain Injury Medicine*. Briefly, stimulant medications increase alertness, attention, and speed of processing. These medications can be helpful in cases in which attention and arousal are reduced or unreliable, as well as with participants with reduced initiation. Modern antidepressants (selective serotonin reuptake inhibitors; SSRIs) can be effective in moderating depression as well as other types of emotional distress, i.e., anxiety, anger. Other medications that have been found helpful in reducing anger and aggressiveness are amantadine and carbamazepine (an anticonvulsant). Trazodone hydrochloride and zolpidem are frequently prescribed to restore a regular sleep cycle. Regular sleep with complete sleep cycles is important to optimal cognitive functioning. Medications to support memory performance may be trialed in individual cases but, in this author's experience, results have been disappointing in most cases. Most medications have not been carefully studied as specifically applied to participants with TBI, and consequently, clear guidelines for their use with this population are not available. For this reason, a physician who is experienced in managing medications for people with TBI should supervise the use of these medications.

In this author's opinion, medications such as these are best used to support a rehabilitation process rather than as circumscribed treatments. For instance, an SSRI may help a participant with TBI and depression to experience less emotional distress, sleep better, and find the energy to participate in rehabilitation and cognitive-behavioral psychotherapy. However, learning active coping skills and increasing their involvement with other people and in valued activities is critical for them to maintain emotional stability in the long run. Similarly, other types of medications may greatly assist participants to fully participate in a rehabilitation process but this is often only the first step toward helping them to learn ways to reduce or work around their disabilities in order to reengage with their communities.

# **Co-morbidities and Prevention**

*Substance abuse.* Active substance abuse or dependence is a contraindication for CBIR since

a substance abuse disorder creates a marked barrier to a participant's ability to participate in and benefit from a rehabilitation process. Alcohol is the most commonly abused substance in the United States. Participants who have a history of alcohol or other substance abuse may effectively participate in CBIR if they are simultaneously engaged in a substance abuse program to support their sobriety or have completed such a program and continue to maintain sobriety. Participants without a history of a substance abuse or dependence will benefit from education about the appropriate use of alcohol and other drugs including prescription medications as part of CBIR (see Corrigan [38] for more detailed information about methods for increasing awareness of and addressing substance abuse issues after TBI).

Mental health. Psychiatric illnesses, particularly depressive and anxiety disorders, are another relatively frequent co-morbidity for CBIR participants. These disorders may have been present prior to or develop after injury. Psychiatric medications are best managed in such cases by a neuropsychiatrist or other physician who is familiar in working with people with TBI. The development of psychological coping skills and supported reengagement in valued activities in the community are features of CBIR that will be of particular benefit to individuals with co-morbid mood disorders. All CBIR participants and involved family/close others will benefit from education about the signs of mental health problems and appropriate actions to be taken if these signs appear.

Wellness. CBIR participants may have a wide array of other co-morbid health conditions that are related or unrelated to their injuries. Common health risk factors in the general population, such as, hypertension and hypercholesterolemia, may be increased by physical inactivity and poor nutrition resulting from physical and cognitive impairments due to TBI. Masel and DeWitt [7] have described the management of TBI as a chronic illness including assisting the individual with TBI to develop a healthy lifestyle. Individuals with TBI frequently have increased difficulty communicating their health needs and concerns to healthcare providers. CBIR programs include both (1) coaching participants about healthy lifestyles and communicating and addressing healthcare needs, and (2) assistance in developing a relationship with a primary healthcare team who can work effectively with the participant to manage their healthcare in the long term.

# **Monitoring/Measuring Outcomes**

CBIR is a highly individualized process. While many components have a firm evidence base, developing the most effective array of these components for the individual participant remains as much an art as a science. For this reason, regular and consistent monitoring of individual participant progress as well as regularly evaluating the overall performance of the CBIR program for all participants is recommended. Such processes support the assessment of the effectiveness of the program for the individual participant and the program as a whole and lead to appropriate modifications to increase effectiveness. Two methods to assess participant progress and programmatic outcomes that have gained relatively broad endorsement are GAS and the Mayo-Portland Adaptability Inventory (MPAI-4).

# GAS

GAS is a method that places individualized participant goals on a 5-point scale (see Table 5). By using the same scale for each goal and participant, progress can be compared across goals and participants, even though the goals themselves may be very different from each other. Using the 5-point GAS scale rather than a simple binary record of goal achievement, i.e., goal accomplished/not accomplished, allows participants to be recognized both for accomplishing a goal at a minimally acceptable level as well as "exceeding expectations."

Goal statements should be SMART, i.e., specific, measureable, attainable, realistic, and timelimited. Goals should also reflect achievements that are valued by the participant. The process of developing a set of GAS can itself be considered an intervention and, as such, dovetails nicely with Table 5 GAS levels and examples

- 4 Much better than expected outcome
- 3 Better than expected outcome
- 2 Expected outcome
- 1 Less than expected outcome

0 Much less than expected outcome

GAS goal: participant routinely uses problem-solving and goal management strategies to solve problems in everyday life

4 Participant learns and uses problem-solving and goal management strategies in addressing life problems almost all the time independently

3 Participant learns and uses problem-solving and goal management strategies in addressing life problems about 75 % of the time independently

2 Participant learns and uses problem-solving and goal management strategies in addressing life problems 75 % of the time with prompting

1 Participant has not learned and does not use problemsolving and goal management strategies

0 Participant refuses to engage in systematic problem-solving

GAS Goal: Participant is in part-time paid employment with support

4 Participant works full-time for pay independently without support

3 Participant works part-time for pay independently without support

2 Participant works part-time for pay with intermittent support from work peers and vocational counselor

1 Participant is unemployed but interested in employment

0 Participant is unemployed and not interested in employment

GMT. The original GAS scaling ranged from -2to +2 with zero indicating the expected level of outcome; however, many of our CBIR participants objected to the negative numbers so we rescaled using "positive" numbers as in Table 5. In scaling individual goals, we typically set "1" as the participant's level on admission to the program and "2" as the minimally acceptable level of achievement on the goal. The number of goals scaled should be relatively small. Using GAS to scale 3–5 goals per participant is recommended. Many other smaller step goals that contribute to achievement of the GAS goals will also need to be identified and monitored. These step goals are simply monitored as achieved or not achieved. Review of GAS and step goals weekly or every other week gives the participant, family/close

other, and rehabilitation team a clear indication of where progress is being made in the primary targets for rehabilitation and where adjustments in the treatment program are needed to improve the rate of progress. Greater detail about GAS and its application is available elsewhere [39].

#### MPAI-4

The MPAI-4 is a rating scale, developed using classical and modern psychometric techniques, that includes the most common areas of disability and limitation that can occur after TBI. It is composed of three subscales measuring Ability, Adjustment and Participation (see Table 6). Its well-established psychometric measurement properties have supported its increasing use to evaluate post-hospital brain injury rehabilitation programs. The MPAI-4, a manual for its use, and foreign language translations are available for free download on the web site of the Center for Outcome Measurement in Brain Injury (COMBI; http://www.tbims.org/combi/mpai). Additional information about the psychometric development and properties of the MPAI-4 is available in the manual and other sources [40-42].

The MPAI-4 is not intended to be a comprehensive list of all possible sequelae of brain injury, since such a list would be so extensive that it would make the inventory impractical for clinical use. Rather, the MPAI-4 focuses on common sequelae that indicate the range of severity of disability after brain injury. The MPAI-4 is best completed by consensus of an evaluating rehabilitation team and provides a method for the team to come to agreement about the participant's profile of disabilities and limitations as they design the rehabilitation treatment plan. A recent article by Lexell et al. [43] shows how the MPAI-4 links to the International Classification of Functioning (ICF). The ICF provides lists of more specific abilities and activities that may become goals for rehabilitation after the general areas for intervention are identified using the MPAI-4. Program staff complete the MPAI-4 again when the participant is discharged. Some programs with more extended lengths of stay may have staff complete the inventory midway through the treatment process. The Participation Index provides a brief assessment of the primary goal of CBIR-community reintegration-that can be completed over the telephone [44]. The Participation Index is used by many programs for follow-up at 3, 6, and/or 12 months to assess the resilience of their outcomes. Examples of the use of the MPAI-4 for evaluations of various types of post-hospital programs can be found elsewhere [11, 12].

Ability index	Adjustment index	Participation index
Mobility	Anxiety	Initiation
Use of hands	Depression	Social contact
Audition	Irritability, anger, aggression	Leisure activities
Vision	Pain/headache	Self-care
Motor speech	Fatigue	Residence
Dizziness	Sensitivity to mild symptoms	Transportation
Verbal communication	Inappropriate social interaction	Employment
Nonverbal communication	Impaired self-awareness	Managing money
Memory	Family/significant relationships	
Attention/concentration	Initiation	
Fund of information	Social contact	
Novel problem-solving	Leisure activities	
Visuospatial abilities		

 Table 6
 Mayo-Portland adaptability index (MPAI-4) items and subscales

Italicized items contribute to both adjustment and participation indices

#### Case Example

A thorough presentation of the complexities and intricacies of a CBIR team working with an individual participant would require another full chapter, at a minimum. However, the case description in Table 7 and the treatment plan outline in Table 8 may give a flavor for how goals are prioritized in an interdisciplinary CBIR team evaluation and how individual and group interventions are designated to achieve these goals. Review of Table 8 will also reveal interventions targeted at developing or improving family, social, and environmental systems in addition to those offered directly to the participant. For some goals, the importance of practice and reinforcement throughout the program in the therapeutic milieu is highlighted, and, for other goals, this is

extended to homework and practice outside the program.

The problem list in Table 8 is referenced to the MPAI-4 and the current status and goal level for each problem is designated using levels of the MPAI-4. In addition, four of the problem areas (novel problem-solving, social interaction, irritability, and employment) are designated for development of more specific GAS. Two of these GAS are used as examples in Table 5. These are areas that are believed to be of critical importance to the overall success of the program and for which the largest change is required. Development of these GAS in collaboration with the participant may take several weeks and, in the case example, will be an important intervention in and of itself to help develop the participant's self-awareness. In achieving all goals, numerous short-term step goals will be set on a daily and weekly basis.

#### Table 7 CBIR team evaluation summary

Shareen C. is a 29-year-old African-American woman who was injured in a motor vehicle accident approximately 3½ years ago. She was driving alone driving at the time of the accident when an approaching driver apparently lost control of his vehicle and hit Shareen head on. The other driver died in the crash. The patient was apparently in excellent health that time of the injury and has no prior history of significant medical conditions at the time of the accident and no a history of psychiatric or substance abuse disorders. Her brain injury was severe. Her initial Glasgow Coma Scale was 6. Posttraumatic amnesia of approximately 2 months was reported. Initial CT scan revealed contusions and small hematomas in the frontal lobes bilaterally; these did not require surgical intervention. MRI 2 years after her injury showed bilateral encephalomalacia in the frontal lobes and temporal poles bilaterally. In addition to her brain injury, the patient fractured her left lower extremity in the accident

She has history of a single seizure in the emergency room after her injury with no subsequent seizure history. She is on Tegretol for irritability and aggression; however her mother feels this has been minimally effective

The patient is a college-educated woman who was working as a communications specialist for a multinational corporation. She has never married. Her mother accompanied her to the evaluation and is her primary caregiver. Shareen's mother divorced her father when Shareen was 5 years old. Shareen has had no contact with her father since that time

An interdisciplinary rehabilitation evaluation reveals a number of sequelae to the patient's brain injury. Mild difficulty in ambulation is apparent associated with persistent left footdrop. However, this is remediated for most functional purposes with the use of a brace. This right-handed woman has mild fine motor impairment bilaterally which interferes with functional activities less than 25 % of the time. Motor speech is impaired. The patient is difficult to understand a minority of the time. However, language abilities are generally intact although the patient has occasional word finding problems. Nonverbal and pragmatic communication difficulties are apparent most of the time in everyday communication. The patient is hyperverbose, frequently interrupts others, and appears insensitive to the normal give-and-take of conversation. Attention and concentration are severely impaired on neuropsychometric testing and distractibility and inattention are also apparent in conversation and other everyday activities per the patient's mother. The patient's mother reports that she is no longer able to multitask. The patient also demonstrates severe new learning ability on neuropsychometric testing and her mother reports that she has difficulty retaining new information most of the time. Her general intelligence is in the lower end of the average range but appears to have declined given her educational and vocational history. Her general fund of information may be intact; however, her ability to access this information is not reliable. The patient demonstrates very significant impairment in abstract reasoning and problem-solving on neuropsychometric testing. Difficulty in managing new situations and problemsolving in everyday life is confirmed by the patient's mother. Although she has mild difficulty on complex visual spatial tasks, probably reflecting higher-order cognitive impairments noted previously, basic visual and visual perceptual abilities appear intact

#### Table 7 (continued)

There is no evidence of significant anxiety and depression; however, the patient's mother reports that she is frequently irritable and verbally aggressive when confronted with her disabilities. The patient has some awareness of the sequelae of her brain injury but does not appear to appreciate the way these disabilities may interfere in everyday life. For instance, she appears unaware of how her cognitive impairments interfere with return to work. She's convinced that she could return to work immediately. Social-communication impairments are also associated with inappropriate and disinhibited interpersonal behavior a majority of the time. Consequently, most of the patient's social interactions are problematic and other people appear to find many of her communications and manner mildly offensive. Although her mother remains very supportive, changes in the patient's interpersonal style are difficult for the mother to understand and are creating at least mild stress within their relationship. The mother describes her daughter as formerly a very goal-oriented and self-assured young woman who now appears to have "gone wild". Difficulties in social interactions, disinhibition and cognitive difficulties have had a detrimental effect on the patient's social network. The friends she had prior to her accident currently have very limited contact with her. She had a close male friend prior to her injury; however, this relationship dissolved over the years subsequent to her injury. Her leisure interests and activities are virtually nonexistent. The patient is able to manage basic self-cares (feeding, grooming, toileting) with only an occasional prompt. However, she requires assistance in many more complex activities of daily living. She currently resides with her mother who takes primary responsibility for most cooking and other household chores. Shareen is unable to drive and cannot use public transportation without assistance; for instance, her mother is required to accompany her to doctors' appointments. The patient is currently unemployed. She is able to manage small financial transactions, such as, shopping for a few items. Her mother is her legal guardian and takes responsibility for managing the savings and investments that Shareen has from before her accident and for the settlement she received from the accident

Problem	Current status	Intervention	Goal level	
Impaired ambulation	Mild problem but does <i>not</i> interfere with activities; uses assistive device	None	No further improvement expected	
Impaired use of hands	Mild problem; interferes with activities 5–24 % of the time	None	No further improvement expected	
Impaired motor speech	Mild problem; interferes with activities 5–24 % of the time	Individual speech therapy	Mild problem but does <i>not</i> interfere with activities	
Impaired word finding	Mild problem but does <i>not</i> interfere with activities	None	No further improvement expected	
Impaired novel problem-solving	Severe problem; interferes with activities more than 75 % of the	Training in systematic problem-solving <sup>a</sup>	Mild problem; interferes with activities 5–24 % of the time	
	time	Goal management training <sup>a</sup>	Develop GAS	
		Engagement in GAS development process		
Impaired attention	Moderate problem; interferes with activities 25–75 % of the time	Attention process training <sup>a, b</sup>	Mild problem; interferes with activities 5–24 % of the time	
Impaired memory	Moderate problem; interferes with activities 25–75 % of the time	Memory notebook development and training <sup>a, b</sup>	Mild problem; interferes with activities 5–24 % of the time	
Impaired nonverbal and pragmatic communication skills	Moderate problem; interferes with activities 25–75 % of the time	Social-communication group <sup>a</sup>	Mild problem; interferes with activities 5–24 % of the time	
Inappropriate social interaction	Moderate problem; interferes with activities 25–75 % of the time	Social communication group <sup>a</sup>	Mild problem but does <i>not</i> interfere with activities	
			Develop GAS	

Table 8         Treatment plan outline for Sharee
---

(continued)

(continued)			
Problem	Current status	Intervention	Goal level
Irritability	Moderate problem; interferes with activities 25–75 % of the time	Neuropsychiatric evaluation for pharmacologic treatment	Mild problem but does <i>not</i> interfere with activities
		Group and individual anger management training <sup>a</sup>	Develop GAS
Needs occasional prompts from mother to complete self-cares	Mild problem but does <i>not</i> interfere with activities; dependent on mother for prompting	Individual OT to develop self-cuing system <sup>b</sup>	Mild problem but does <i>not</i> interfere with activities; independent with cuing system
Impaired self-awareness	Moderate problem; interferes with activities 25–75 % of the time	Patient/family education Self-awareness intervention <sup>a</sup> Work trials	Mild problem; interferes with activities 5–24 % of the time
Unable to live independently	Requires moderate assistance or supervision from others (25–75 % of the time)	Address cognitive, emotional, and social issues and transition to group home situation	Requires a little assistance or supervision from others (5–24 % of the time)
Unable to travel around town independently	Requires moderate assistance or supervision from others (25–75 % of the time); cannot drive	Training in limited use of public transportation	Requires a little assistance or supervision from others (5–24 % of the time); cannot drive
Limited social contact	No or rare involvement with others (less than 25 % of normal interaction for age)	Address emotional and social problems that are obstacles to new relationships	Mildly limited involvement with others (75–95 % of normal interaction for age)
		Social communication group <sup>b</sup>	Develop GAS
Limited leisure/ recreational activities	No or rare participation (less than 25 % of normal participation for age)	Leisure skills group <sup>b</sup>	Mildly limited participation (75–95 % of normal participation for age)
Unemployment	Unemployed	Individual vocational counseling	Full-time or part-time employment with support
		Resource facilitation	Develop GAS
		Work trials	
Unable to manage money independently	Requires a little help or supervision (5–24 % of the time) with large finances; independent with small purchases	Counseling with mother and daughter to develop	No change in patient status expected
		long term plan for financial management support	Goal is to develop more viable, long term, external support system for participant in managing her financial affairs
Strained relationship with mother	Mild stress that interferes with family functioning 5–24 % of	Counseling/behavioral rehearsal with mother and daughter	Normal stress within family
	the time	Training mother to prompt/ reinforce daughter's anger management and improved social interaction <sup>b</sup>	
		Transition to group home	

#### Table 8 (continued)

<sup>a</sup>Practice/reinforcement in other groups and therapeutic milieu

<sup>b</sup>Homework/practice outside of program with review in program

Progress toward goals will be evaluated formally at conferences that include the participant and her mother every other week and more frequently in team briefing/debriefing sessions. Goals and the treatment plan will be further modified and refined through these ongoing evaluations.

# Conclusion

As can be seen from the methods and processes described in this chapter and in the brief case report, CBIR is a complex, multimodal, transdisciplinary intervention that addresses sequelae of TBI holistically and comprehensively. Not only are interventions implemented to reduce the impairments of the individual with TBI but also to modify family, social, and environmental systems in order to facilitate the participant's reentry into community life. Most of the individual interventions are supported by scientific study; however, the combination of these interventions is highly individualized depending on the needs and goals of the participant. Transdisciplinary teamwork and the development of the therapeutic alliance that are so critical to the success of CBIR are difficult to prescribe in detail or manualize. Consequently, CBIR is as much art as science and may remain so into the indefinite future.

Acknowledgement Preparation of this chapter was partially supported by the U.S. Department of Education National Institute on Disability and Rehabilitation Research (NIDRR) grant H133A120035.

# References

- Ben-Yishay, Y., & Diller, L. (1993). Cognitive remediation in traumatic brain injury: Update and issues. *Archives of Physical Medicine and Rehabilitation*, 74(2), 204–213.
- Ben-Yishay, Y., & Prigatano, G. P. (1990). Cognitive remediation. In M. Rosenthal, E. R. Griffith, M. R. Bond, & J. D. Miller (Eds.), *Rehabilitation of the adult and child with traumatic brain injury* (pp. 393– 400). Philadelphia: Davis.
- Prigatano, G. P., Fordyce, D. J., Zeiner, H. K., Roueche, J. R., Pepping, M., & Wood, B. C. (1986). *Neuropsychological rehabilitation after brain injury*. Baltimore, MD: Johns Hopkins University.

- Trexler, L. E., Diller, L., Gleuckauf, R., Anreiter, B., Ben-Yishay, Y. et al. (1994). Consensus conference on the development of a multi-center study on the efficacy of neuropsychological rehabilitation. June 1994, Zionsville, IN.
- Ben-Yishay, Y., & Diller, L. (2011). Handbook of holistic neuropsychological rehabilitation outpatient rehabilitation of traumatic brain injury. New York: Oxford.
- Malec, J. F., & Degiorgio, L. (2002). Characteristics of successful and unsuccessful completers of three postacute brain injury rehabilitation pathways. *Archives of Physical Medicine and Rehabilitation*, 83(12), 1759–1764.
- Masel, B. E., & DeWitt, D. S. (2010). Traumatic brain injury: A disease process, not an event. *Journal of Neurotrauma*, 27(8), 1529–1540.
- Bush, B. A., Novack, T. A., Malec, J. F., Stringer, A. Y., Millis, S., & Madan, A. (2003). Validation of a model for evaluating outcome after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(12), 1803–1807.
- Malec, J. F. (2001). Impact of comprehensive day treatment on societal participation for persons with acquired brain injury. *Archives of Physical Medicine* and Rehabilitation, 82, 885–894.
- Novack, T. A., Bush, B. A., Meythaler, J. M., & Canupp, K. (2001). Outcome following traumatic brain injury: Contributions from premorbid, injury severity, and recovery variables. *Archives of Physical Medicine and Rehabilitation*, 82, 300–305.
- Altman, I. M., Swick, S., Parrot, D., & Malec, J. F. (2010). Effectiveness of community-based rehabilitation after traumatic brain injury for 489 program completers compared with those precipitously discharged. *Archives of Physical Medicine and Rehabilitation*, 91, 1697–1704.
- Eicher, V., Murphy, M. P., Murphy, T. F., & Malec, J. F. (2012). Progress assessed with the Mayo-Portland Adaptability Inventory through the OutcomeInfo system for 604 participants in four types of post-inpatient rehabilitation brain injury programs. *Archives of Physical Medicine and Rehabilitation, 93*, 100–107.
- Raemer, D. B. (2009). Team-oriented medical simulation. In W. F. Dunn (Ed.), *Simulators in critical care and beyond*. Mt. Prospect, IL: Society for Critical Care Medicine.
- Cailhol, L., Rodgers, R., Burnand, Y., Brunet, A., Damsa, C., & Andreoli, A. (2009). Therapeutic alliance in short-term supportive and psychodynamic psychotherapies: A necessary but not sufficient condition for outcome? *Psychiatry Research*, 170(2–3), 229–233.
- Hewitt, J., Coffey, M., Hewitt, J., & Coffey, M. (2005). Therapeutic working relationships with people with schizophrenia: Literature review. *Journal of Advanced Nursing*, 52(5), 561–570.
- 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. *The Journal of Head Trauma Rehabilitation*, 24(3), 145–154.

- 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 post-acute brain injury rehabilitation. *The Journal of Head Trauma Rehabilitation*, 23(5), 329–338.
- Klonoff, P. S., Lamb, D. G., & Henderson, S. W. (2001). Outcomes from milieu-based neurorehabilitation at up to 11 years post-discharge. *Brain Injury*, *15*(5), 413–428.
- Cicerone, K. D., Dahlberg, C., Kalmar, K., Langenbahn, D. M., Malec, J. F., Bergquist, T. F., et al. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Arch Phys Med Rehabil*, 81(12), 1596–1615.
- Cicerone KD, Dahlberg C, Malec JF, Langenbahn DM, Felicetti T, Kneipp S, Ellmo W, Kalmar K, Giacino JT, Harley JP, Laatsch L, Morse PA, Catanese J (2005). Evidence based cognitive rehabilitation: updated review of the literature from 1998 through 2002. Arch Phys Med Rehabil 86, 1681–92.
- Cicerone, K. D., Langenbahn, D. M., Braden, C., Malec, J. F., Kalmar, K., Fraas, M., et al. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003 through 2008. *Archives of Physical Medicine and Rehabilitation*, 92, 519–530.
- 22. Haskins, E. C., Shapiro-Rosenbaum, A., Dams-Oconnor, K., et al. (2011). Cognitive rehabilitation manual: Translating evidence-based recommendations into practice. Reston, VA: American Congress of Rehabilitation Medicine.
- Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attention-training program. *Journal of Clinical* and Experimental Neuropsychology, 9, 117–130.
- Sohlberg, M. M., & Mateer, C. A. (2001). Cognitive rehabilitation: An integrative neuropsychological approach. New York: Guilford.
- Levine, B., Robertson, I. H., Clare, L., Carter, G., Hong, J., Wilson, B. A., et al. (2000). Rehabilitation of executive functioning: An experimental-clinical validation of goal management training. *Journal of the International Neuropsychological Society*, 6(3), 299–312.
- 26. Dahlberg, C. A., Cusick, C. P., Hawley, L. A., Newman, J. K., Morey, C. E., Harrison-Felix, C. L., et al. (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.
- Cattelani, 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.

- 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*. New York: Oxford.
- Van Velzen, J. M., Van Bennekom, C. A. M., Edelaar, M. J. A., Slutter, J. K., & Frings-Dresen, M. H. (2009). How many people return to work after acquired brain injury?: A systematic review. *Brain Injury*, 23, 473–488.
- Malec, J. F., Buffington, A. L. H., Moessner, A. M., & Degiorgio, L. (2000). A medical/vocational case coordination system for persons with brain injury: An evaluation of employment outcomes. *Archives of Physical Medicine and Rehabilitation*, 81, 1007–1015.
- Malec, J. F., & Moessner, A. M. (2006). Replicated positive results for the VCC model of vocational intervention after ABI within the social model of disability. *Brain Injury*, 20(3), 227–236.
- Trexler, L. E., Trexler, L. C., Malec, J. F., Klyce, D., & Parrott, D. (2010). Prospective randomized controlled trial of resource facilitation on community participation and vocational outcome following brain injury. *The Journal of Head Trauma Rehabilitation*, 25(6), 440–446.
- Buffington, A. (1997). The vocational rehabilitation continuum: Maximizing outcomes through bridging the gap from hospital to community-based services. *The Journal of Head Trauma Rehabilitation*, 12(5), 1–13.
- 34. Sander, A. M., Sherer, M., Malec, J. F., High, W. M., Thompson, R. N., Moessner, A. M., et al. (2003). Preinjury emotional and family functioning in caregivers of persons with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 84(2), 197–203.
- Meythaler, J. (2012). Neuropharmacology. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (2nd ed.). New York: Demos.
- Arciniegas, D. (2012). Pharmacotherapy of cognitive impairment. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (2nd ed.). New York: Demos.
- Arciniegas, D. (2012). Pharmacotherapy of neuropsychiatric disturbance. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (2nd ed.). New York: Demos.
- Corrigan, J. (2012). Substance abuse. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine* (2nd ed.). New York: Demos.
- Malec, J. F. (1999). Goal attainment scaling in rehabilitation. *Neuropsychological Rehabilitation*, 9(3/4), 253–275.
- 40. Kean, J., Malec, J. F., Altman, I. M., & Swick, S. (2011). Rasch measurement analysis of the Mayo-Portland Adaptability Inventory (MPAI-4) in a community-based rehabilitation sample. *Journal of Neurotrauma*, 28, 745–753.
- 41. Malec, J. F., Kragness, M., Evans, R. W., Finlay, K. L., Kent, A., & Lezak, M. (2003). Further psychometric evaluation and revision of the Mayo-Portland Adaptability Inventory in a national sample. *The Journal* of Head Trauma Rehabilitation, 18(6), 479–492.

- Malec, J. F., & Lezak, M. D. Manual for the Mayo-Portland adaptability inventory (2nd ed.). http://www. tbims.org/combi/mpai. Accessed March 24, 2014.
- 43. Lexell, J., Malec, J. F., & Jacobsson, L. L. (2012). Mapping the Mayo-Portland adaptability inventory to the international classification of functioning, disability, and health. *Journal of Rehabilitation Medicine*, 44, 65–72.
- 44. Malec, J. F. (2004). The Mayo-Portland Participation Index (M2PI): A brief and psychometrically-sound measure of brain injury outcome. Archives of Physical Medicine and Rehabilitation, 85, 1989–1996.
- Malec, J. F., & Basford, J. S. (1996). Postacute brain injury rehabilitation. Archives of Physical Medicine and Rehabilitation, 77(2), 198–207.

Part IV

Special Issues

# Pediatric Traumatic Brain Injury: Outcome, Assessment, and Intervention

# Mary R. Prasad and Linda Ewing-Cobbs

# Abstract

Traumatic brain injury (TBI) is a leading cause of disability in children. This chapter addresses the unique challenges facing children with TBI. Outcome, assessment, and intervention issues are discussed in relation to injuries sustained during two stages of development: (1) infancy and early childhood and (2) school-age and adolescence. Two cases studies are presented to illustrate issues relevant to children with TBI.

#### Keywords

Pediatric traumatic brain injury • Academic outcomes • Educational accommodations

Traumatic brain injury (TBI) is a leading cause of disability in children [1]. Severe TBI often produces alterations in many domains of daily functioning that persist throughout life. In this chapter, we will discuss outcome, assessment, and intervention issues in relation to injuries sustained during two stages of development: (1) infancy and early childhood and (2) school-age and adolescence. We provide case studies to illustrate the unique issues and challenges facing children who sustain TBI during these different stages of development.

Epidemiological studies indicate that over one million children a year sustain TBI and approxi-

mately 30,000 have long-term disabilities [2]. Each year in the United States, approximately 475,000 children between the ages of 0 and 14 years sustain a TBI [3]. Children aged 0-4 years and older adolescents aged 15-19 years have a greater likelihood of sustaining a brain injury than other age groups [4]. The external cause of injury varies according to age. In infancy to early childhood, falls and bicycle/pedestrian-motor vehicle collisions are the most frequent causes of TBI [5]. Although assault is responsible for only approximately 5 % of brain injuries in children aged 1-4 years, assault produces 90 % of serious brain injuries [6]. Head trauma inflicted by child abuse is the leading cause of death in children under the age of 3 years. In school-aged children and adolescents, motor vehicle incidents are a primary cause of injury and more recently there is a growing recognition of sports-related TBI [7].

M.R. Prasad, Ph.D. (⊠) • L. Ewing-Cobbs, Ph.D. Children's Learning Institute, University of Texas Health Science Center, Houston, TX, USA e-mail: Mary.R.Prasad@uth.tmc.edu

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_16, © Springer Science+Business Media, LLC 2014

Dennis and colleagues [8] postulated that brain injuries sustained in childhood can alter both brain reserve and cognitive reserve. Brain reserve is a proxy variable for the health of the brain and may reflect the influence of a variety of genetic/congenital factors as well as environmental insults, such as chemotherapy or TBI. Cognitive reserve is viewed as a combination of pre-injury and post-injury cognitive abilities, as well as environmental factors such as socioeconomic status and family environment. Brain reserve and cognitive reserve influence each other; their joint influence on outcomes may be moderated or mediated by injury-related variables such as the age at injury, time since injury, and type/location of parenchymal injury. The combined influence of these factors affects functional plasticity, which in turn affects physical, cognitive, and psychosocial outcomes.

The sequelae of TBI can vary greatly depending on many injury-related factors including severity of injury to the brain and body, age at injury, type and location of brain injury, as well as factors related to the child's pre-injury abilities and characteristics of the family environment before and following the injury. Although these cognitive and brain reserve factors interact in complex ways, severity of TBI is a major predictor of the quality of outcomes. Although many TBIs are considered to be mild and result in few long-term impairments, infants, children and adolescents with severe TBI sustain significant, chronic impairments [9].

Recently, the Pediatric Common Data Elements (CDE) Traumatic Brain Injury Outcomes Workgroup identified a common set of core and supplemental outcome measures for research in pediatric TBI [10]. Although initially developed for researchers, the recommended battery is an excellent resource for clinicians as well. The battery covers many domains, including adaptive and daily living skills, health-related quality of life, language and communication, attention and processing speed, executive functioning, memory, physical functioning, as well as social and psychological functioning. These domains often have overlapping influences. For example, neuropsychological deficits in executive functions, attention, memory as well as behavioral dysregulation are related to poor health-related quality of life [11], are predictive of parental stress and burden [12], and contribute to poorer social outcomes [13]. In addition to covering the same domains as the common data elements for adults, the pediatric elements include academic outcomes and family functioning/environment. Inclusion of these areas reflects the centrality of these domains for children's everyday performance. Interestingly, these latter domains were emphasized by Taylor [14], who described the role of the neuropsychologist as isolating the "signal" of TBI from the background "noise" by evaluating how post-injury functioning impacts home and school.

#### **TBI in Early Childhood**

Studies examining a variety of neuropsychological outcomes have identified widespread alteration in functioning after moderate to severe TBI. Young children who sustain significant TBI are at high risk for lifelong reduction of abilities in many domains, including cognitive and motor abilities as well as behavioral competencies. Due to a combination of injury-related and family environment factors, outcomes appear to be less favorable in infants with inflicted TBI than in children with accidental or noninflicted injuries [15]; see review by Ewing-Cobbs and Prasad [16]. Across the first few years after TBI, infants and preschool-aged children with moderate to severe accidental TBI show lower initial general cognitive scores and less recovery over time than seen following TBI in school-aged children or adolescents [9, 17–19]. In young children, recovery curves depicting the post-traumatic change in IQ scores across time are either flat, indicating no improvement in scores after the initial injury [19], or show a decline across time [20], indicating failure to develop new skills at age-appropriate rates. In particular, TBI acquired early in life may impede the progression of later-developing skills due to the combined negative impact of reduced general cognitive skills and diminished efficiency for learning and retaining new information [21].

Acquisition of academic skills, particularly reading, may be vulnerable following early brain injury. Barnes and colleagues [22] found that children who sustained a significant brain injury prior to learning how to read scored significantly lower on reading tasks than children injured at older ages. Children who sustained severe brain injuries between the ages of 3 and 7 years scored lower on measures of early academic development and cognitive functioning than children with orthopedic injuries [23]. These academic deficits appear to persist, resulting in significant academic challenges for children who sustain significant brain injury in early childhood. A follow-up study of children who sustained moderate to severe TBI prior to the age of 3 years found that nearly 50 % failed a school grade and/or required placement in self-contained special education classrooms. The odds of unfavorable academic performance were 18 times higher for children with TBI than for healthy comparison children [24]. Unfavorable vocational outcomes have been associated with children sustaining TBI early in life [25], even in some persons who achieved normal school performance [26]. Clearly, severe TBI sustained early in life is associated with a high risk for serious academic and vocational difficulties that may become increasingly evident later during development.

Recent attention has been given to the role of family environment and psychosocial factors in outcome from early TBI [27]. Authoritarian and over controlling parenting styles worsened behavioral outcomes for children who had sustained moderate to severe TBI [28]. Parenting style and family functioning were found to be related to behavioral aspects of executive function in children under the age of 7 with TBI. For children with moderate TBI, authoritarian parenting was associated with greater executive functioning difficulties at 1 year post-injury [29]. Young children with TBI were rated by caregivers as having significantly lower social competence than orthopedic controls at 6 months post-injury [30]. Children who had sustained TBI prior to the age of 4 years were found to have higher rates of social impairment at 8 years of age than children injured between the ages of 4 and 6 years [31].

# Assessment of Young Children with TBI

There are no specific neuropsychological instruments per se for assessing outcomes in infants with TBI. Measures of general cognitive and motor development such as the Bayley Scales of Infant Development-3rd Ed. [32] or Mullen Scales of Early Learning [33] are often used. Both measures have been used in studies examining outcome from early TBI [15, 34-36]. These are comprehensive measures that provide subscale scores for cognitive, motor, and language functioning. Parent rating scales are useful in assessing social or behavioral issues in children over the age of 1 year. Commonly used measures include the Child Behavior Checklist [37] and the Brief Infant and Toddler Social Emotional Assessment [38]. Adaptive functioning measures also include scales regarding social and emotional functioning and have been used to assess outcome in infants and young children with TBI [39, 40]. Several measures assessing executive functioning are available for children as young as 18 months. The Behavior Rating Inventory of Executive Function-Preschool [41] is a parent completed questionnaire assessing early components of executive functioning. The NEPSY-2 [42] also has inhibitory control and other executive tasks that begin at the age of 2 years.

# Intervention for Infants and Preschoolers with TBI

Young children are by their very nature dependent on adults and often their impairments following a significant brain injury may not be as readily apparent to families. As children age and more is expected of them, the impact of their cognitive impairments becomes more frank and families often find themselves unprepared for the challenges that the children are facing. Finding appropriate rehabilitative services for young children with TBI is challenging. We have found that most young children with severe TBI do not receive appropriate rehabilitative services. Infants and toddlers under the age of 36 months with TBI are eligible for therapeutic services under Part C of the Individuals with Disabilities Education Act (IDEA; Public Law 101-476) through state run early childhood intervention programs (ECI). To qualify for ECI services, a child must first undergo a screening evaluation conducted by ECI professionals. It is not uncommon for young children with TBI to be ineligible for intervention service because their performance on a developmental screening measure was found to be above the deficient range for their age. We have found that comprehensive developmental assessments using standardized measures such as the Bayley Infant Development Scales-III or the Mullen Scales of Early Learning are more sensitive to the cognitive and motor sequelae of TBI than screening measures. In addition, longitudinal studies do not show any significant "catch up" growth over time. Rather, severe TBI appears to reduce the level of performance in a given area; over time, the child continues to develop new skills, but at a slower rate. For example, a child performing at the 60th percentile prior to injury may score at the 20th percentile after injury and continue to develop skills at this level without closing the gap over time. As seen in the case study below, some scores may not stabilize and may continue to decline.

Changes in funding for ECI in some states have resulted in families being expected to share some of the cost of services. Unfortunately, these copayments can further burden a family that is dealing with the financial ramifications of their child's injury and hospitalization. The IDEA emphasizes the parent-child relationship as a tool for change although there is considerable variation among ECI programs as to how or if this occurs. In an informal survey of infants and preschoolers with moderate to severe TBI participants in our research study, only 13 % received direct services from ECI and 8 % received information on how to appropriately stimulate their child's development from ECI. Often parents are not aware that they can engage their children in stimulating activities that may help to improve their development. Most parents are not aware that ECI is mandated to educate parents on how to stimulate their child's development. ECI is a

valuable resource for infants and toddlers. After the age of 36 months, children are referred to their local public schools for services. The means by which children with TBI receives special education services through the public school systems is detailed later in this chapter.

# Case 1: Infant with Inflicted Brain Injury

Jessica sustained a severe inflicted brain injury at the age of 2 months. She was brought to the hospital unresponsive with a Glasgow Coma Scale score of 3T. An MRI of the brain performed 1 week post-injury revealed right hemisphere subacute subdural hemorrhage, laminar necrosis with diffuse swelling, infarction involving the right hemisphere, and leftward shift of midline structures. Jessica was discharged from the hospital to the care of her mother approximately 1 month post-injury. At the time of discharge, she had left arm and leg weakness and a preference for her right visual field. Jessica did not receive inpatient rehabilitation therapies.

The alleged perpetrator of the abuse was Jessica's mother's boyfriend. No charges were filed against the boyfriend and Jessica was returned to the care of her mother who continued to reside with the boyfriend. Prior to and for several months following the injury, Jessica's mother was gainfully employed and had health care insurance which provided Jessica with physical therapy and speech/language therapy twice weekly. Jessica's initial and 3-month post-injury evaluations are presented in Fig. 1. Jessica's baseline and 3-month post-injury scores are below average, indicating mild deficits in cognitive and motor development. However, observations of Jessica during the testing session suggested significant neurological impairments. She had significant left-sided weakness. Objects manipulated by her right rarely came to midline, objects were not passed through the midline, and her left foot was turned in and was weaker than her right foot when attempting to stand with support. More spontaneous motor activity was observed on the right side. By 1 year post-injury,



**Fig. 2** Standard area scores of 89–111 are in the average range



Jessica had mild improvement in left-sided strength and was able to bear weight on her left leg and ambulate. She was very active and readily babbled to communicate. As demonstrated in Fig. 1, Jessica demonstrated marked growth in her cognitive and motor skills by 1 year postinjury. She was above expectancy in her cognitive development and demonstrated age appropriate motor skills despite having pronounced left-sided weakness. Although Jessica was clearly benefitting from therapeutic interventions, because her scores were at or above age expectancy, her insurance carrier declined to continue to cover physical, speech/language, and occupational therapies. She was also denied services through ECI. Her mother was unable to afford to pay out of pocket for therapeutic services. Soon after the 1 year post-injury mark, the mother left her boyfriend, lost her job, had a falling out with her family, and was evicted. She lived with various family members and boyfriends for the next several years. Not only was Jessica no longer receiving much needed therapies, there was also considerable instability and stress in the family environment. Her followup assessment at 2 years post-injury indicated a marked reduction in growth in her cognitive and motor skills. Given the devastating nature of Jessica's brain injury, it is not likely that she would have been able to maintain age appropriate cognitive and motor skills as she aged; however, the early loss of therapeutic interventions coupled with high environmental stress may have undermined potential gains in her development. At the age of 6 years, Jessica was found to have below average intellectual functioning on the Stanford-Binet Intelligence Scale 4th Edition and by 8 years of age, there was a further reduction in the growth of intellectual development (see Fig. 2). By her last follow-up assessment at the age of 8 years, Jessica was in first grade for the third time. Her mother had placed her in three different schools, including one accelerated Montessori-based program. The change in



Case 1 Scores on the Woodcock-Johnson-III Tests of Achievement



schools was prompted by her mother's desire to find a school that could address Jessica's learning difficulties. Jessica's performance on the Woodcock-Johnson-III Tests of Achievement (WJ-III) is presented in Fig. 3. Jessica's academic skills relative to her age group were in the Borderline range, consistent with her level of intellectual functioning. However, relative to her grade level, her scores were in the Average range. By Jessica's third year in first grade, she was able to demonstrate grade appropriate skills. Jessica's mom was committed to keeping her in first grade until she "learned what she is supposed to learn." Her mother believed that once Jessica mastered first grade skills, she would be able to move forward with her education without further issues. However, given Jessica's cognitive impairments it was unlikely that she would be able to maintain pace with a regular education curriculum without significant educational accommodations and supports. Jessica's teachers were aware of her learning challenges and recommended that she be placed in special education services. During the feedback meeting, we discussed the impact of Jessica's brain injury on her cognitive development and the impact of these deficits on the rate at which Jessica is able to master new academic skills. Her mother acknowledged that she was ready to accept that her daughter would have lifelong impairments and that she needed special education services and stability. Jessica was very much aware of learning difficulties. By the age of 8, she was questioning why she was different from other children and why this injury happened to her. Jessica and her family were referred to a therapist to help them begin the process of coming to terms with the injury.

# TBI in School Aged Children and Adolescents

Meta-analysis of cognitive outcomes after pediatric TBI [43] found that the most significant chronic effects of moderate and severe TBI were evident in intellectual functioning, processing speed, attention, working memory, fluency, inhibition, problem solving, and delayed recall of newly learned verbal and visual information from memory. Improvement over time was greatest in Performance IQ, processing speed, and working memory [43]. In contrast, memory and learning did not show significant improvement over time. Impairments have been noted on tests of verbal and visual learning [44-48] for children with severe TBI. The persistence of learning and memory difficulties is unfortunate, since one of the major developmental tasks of childhood and adolescence involves learning the academic curriculum.

Unlike infants and preschoolers, older children and adolescent survivors of severe TBI have acquired academic and other abilities prior to their injury, which may confer greater cognitive reserve. However, previously learned skills and abilities can appear relatively intact during the early stages of school re-entry following TBI, giving the false impression to educators and parents that the ability to learn, retain, and implement new skills is intact. Academic outcome studies have found that school-aged children and adolescents with severe brain injuries scored lower on measures of reading, spelling, and mathematics than children with moderate TBI [49] and struggled with mastering new information and skills [50]. Children with severe TBI from families with fewer socioeconomic assets are at increased risk for poorer academic performance [51]. Catroppa and Anderson [52] found that premorbid academic ability (based on teacher's ratings of academic skills) and verbal memory skills were significantly predictive of academic attainment. Children with pre-injury intellectual or learning disabilities are extremely vulnerable to post-traumatic exacerbation of their academic difficulties [45].

Are there alterations in metacognitive processes that underlie the post-traumatic disruption of diverse cognitive and academic skills? Metacognition is "knowing about knowing" and includes being able to use certain strategies for problem-solving and learning. Hanten and colleagues [53–56] have found that post-traumatic difficulties in metacognitive abilities, such as categorization, using learning strategies, and directing learning resources to the most important information, contributed to poor academic performance. Similarly, Barnes [57] found that children with TBI had specific difficulties making inferences to support their comprehension. Even when they understood and remembered factual information from a story, they were inefficient at holding different sources of relevant information in working memory long enough to make inferences. They also had problems knowing when an inference was required to understand what they heard. These examples highlight ways in which specific difficulties in working memory, selective attention, and metacognitive strategies may disrupt everyday cognitive and academic performance. Metacognitive difficulties may represent fruitful targets for intervention.

Childhood TBI has been associated with significant and persistent changes in social development and adaptive functioning [58–62]. Children with severe TBI have been found to have long-term issues in developing friendships and social contacts, and are more likely to exhibit social withdrawal [63]. Certain sequelae of severe TBI such as cognitive and behavioral functioning were more marked in the context of higher compared with lower levels of family burden or dysfunction [27, 51]. Studies examining neuropsychiatric outcomes at least 1 year postinjury have found elevated rates of emotional disorders, mixed emotional and cognitive disorders, attention-deficit/hyperactivity disorder (ADHD), major depressive disorder, and conduct disorders [64, 65]. Anxiety and mood disorders are commonly identified [65–71]. Prefrontal damage, as well as injury to deep gray matter structures such as the amygdala, places children with TBI at high risk for neuropsychiatric and behavioral difficulties [68, 71–75].

Family functioning has been found to be a significant predictor of outcome from brain injury in children. In school-aged children and adolescents with TBI, Yeates and colleagues [76] found that after accounting for injury severity, pre-injury family environment significantly predicted cognitive and behavioral outcome 1 year post-injury for children and adolescents with TBI. Taylor and colleagues [12] found that higher parent stress at 6 months post-injury predicted more child behavioral difficulties at 12 months post-injury and more child behavior problems at 6 months predicted worse family outcomes at 12 months post-injury. In essence, they found a bidirectional influence of child and family on outcomes following brain injury.

# Assessment of School-Aged Children with TBI

Numerous measures are available to assess intellectual, neuropsychological, behavior, academic, and social functioning in school-aged children with TBI. The Pediatric CDE Working Group identified measures which were considered to be valid, robust, and widely used in clinical research with children with TBI [10]. Although a mainstay of neuropsychological evaluations, comprehensive assessment of intellectual functioning may not be as useful for rehabilitation planning and treatment as briefer and more focused assessment that can be repeated to track recovery. Assessment batteries should include tests that focus on areas that are commonly disrupted by TBI as well as tests of abilities that are targets of intervention.

Assessment of children with TBI includes many of the same domains as assessment of adults with TBI: executive functioning, memory, attention, processing speed, and motor skills. Perhaps the greatest distinction between the assessment of children with TBI and adults with TBI is the need for educational planning and treatment for the former. Although academic assessment is often not covered by third party payers, assessing the child's academic functioning is highly relevant for the child's habilitation. For children, school is the primary place where they are taught and are expected to perform. It is in essence their work environment. Many insurance carriers do not cover the cost of academic assessment because of an inappropriate expectation that the public schools are responsible for providing this service for the children. However, most school professionals do not have the background or familiarity with TBI to interpret the academic test findings in the context of the child's brain injury. Assessment of academic functioning should include basic word decoding, reading comprehension, math calculation and reasoning, and expressive writing skills. Given the cognitive and motor slowing that commonly occurs following severe TBI, performance on measures assessing academic fluency, or the speed and efficiency at which the child is able to work, are an essential component of any battery. Curriculum-based assessments are also valuable for assessing retention of academic material.

#### **Return to School**

A critical factor contributing to the identification of students with TBI for special education is the link between hospital and school [77]. The majority of children with severe TBI will require special education support [78]. For children with moderate to severe TBI, transition from hospital to school is done gradually. Children in inpatient rehabilitation units often receive onsite teaching services from the local school district. Following discharge, interim homebound teaching which is provided by the child's public school is recommended for many children with severe TBI. Children with mild to moderate TBI vary in their stamina, strength, and attention and some may require a gradual transition to school. Re-entry to the school environment for most children with TBI, regardless of injury, is recommended to begin with a half-day placement with time in school increasing as the child's stamina improves [79]. Children with mild TBI can suffer from physical fatigue and confusion that impact their return to school. Returning a child to full-day school too quickly can be detrimental to the child's well-being. Readjustment to the school environment depends on the transition plan designed by the student's rehabilitation or hospital team [80].

Often times, the mere presence of a brain injury is not sufficient to obtain special education services. An educational need must be demonstrated. In a study conducted by Glang and colleagues [77], only 25 % of children with TBI were identified for formal special education services. Over 41 % received informal supports (e.g., schedule change, extra time on tests). Injury severity and hospital–school transition services were predictive of provision of special education services. Clearly, children with TBI are under identified by school personnel and better linkages between medical and educational systems are needed.

Despite the high incidence of TBI in children, many school personnel are unfamiliar with TBI and this unfamiliarity with the sequelae of brain injury can lead to less than satisfactory educational services [81]. A comprehensive neuropsychological evaluation provides information that can be used to help justify educational need and help guide the family and school personnel in developing appropriate accommodations and interventions. Including links to the websites with resources for educators in the neuropsychological report provides an opportunity for educators to learn more about the challenges facing their students with TBI.

There are two paths by which a student with TBI can receive special education services in the public school systems. TBI is a qualifying condition for receipt of special education services under the IDEA, which was most recently reauthorized in 2004. Under this legislation, a child must be assessed in all areas related to his or her suspected disability. The evaluation must identify the child's needs for both special education and related services. Related services include speech-language pathology and audiology services, and physical and occupational therapy. In addition, psychological and social work services may be included. Based on the comprehensive evaluation, an individualized educational plan, often referred to as an IEP, is developed. The IEP must address the child's current level of functioning (sometimes referred to as performance), list annual goals, describe the measurement of progress to meet goals, and list specific special education services and accommodations.

The IEP is developed and written by a team that includes the parents or guardians, teachers, and other professionals at the school, most likely the school psychologist, speech/language therapist, and physical therapist. This team is referred to as the Assessment, Review, and Dismissal (ARD) committee. The ARD committee will convene a meeting to develop the IEP within 30 days of deciding that the child is eligible for special education services. The IEP must be reviewed every year to insure that it is meeting the educational needs of the student although the ARD committee can convene as often as needed. Particularly during the first year after TBI, the committee should meet periodically since improvement is likely to be uneven, with significant gains in some areas and less in others. IEP goals will need to be adjusted depending on the rate of recovery or improvement [82]. In addition, we often suggest to the IEP committee that safeguards be put in place regarding the child's safety. Children with severe TBI are vulnerable to manipulation and abuse by their peers. The school must take precautions to ensure a safe and supervised environment, taking into account the child's right to an education in the least restrictive environment.

Some students with TBI will need a Behavior Intervention Plan (BIP) to address the prevention of maladaptive behaviors associated with the brain injury. The BIP should focus on positive behavior supports and may include parent or in home training. BIPs are also important for students with TBI who are unable to follow the rules of conduct at school. Students with TBI may not respond to traditional contingency management protocols because of impaired selfregulation, poor initiation, and difficulties with contingency learning [83]. Positive behavior intervention and supports (PBIS) is an antecedent-based intervention that has been demonstrated to have some efficacy in students with TBI in a series of single subject studies [83–85]. PBIS differs from traditional behavioral intervention methods by focusing more on lifestyle changes through internal control of behavior. There is a focus on control of antecedents including events that occurred earlier as well as internal events such as loneliness. The environment is adjusted to meet the needs of the student so that that there is a high degree of success. The intervention is conducted in naturalistic settings such as home and school and involves the student's primary caregivers.

Another avenue by which students receive educational accommodations is by Section 504. Often simply referred to as "504," Section 504 is a federal civil rights law that prohibits discrimination against individuals with disabilities. Section 504 provides children with disabilities equal access to education and as such, they are allowed educational accommodations and modifications. These accommodations and modifications are for the general curriculum and do not include additional therapeutic interventions outside the classroom. The 504 plan does not provide an educational program that is tailored to the child's needs. For children with mild to moderate TBI, who are able to make adequate progress in the general curriculum with accommodations such as extended time for classroom assignments and tests, a 504 plan may be an acceptable option by which to receive accommodations in the schools. However, for children with moderate to severe TBI who have extensive cognitive and/or physical impairments, an IEP should be implemented at the school.

In our experience, children with TBI are often erroneously classified for special education services with the most common classification being ADHD. Children with TBI may have premorbid developmental disabilities such as ADHD that necessitated special education services [45, 86]. However, comorbid TBI has a significant impact on the child's abilities and functioning and these changes need to be addressed by the school in order for the child to receive an appropriate education. To illustrate, we were contacted by the family of a boy who had sustained a severe TBI several years earlier. The family lived in a small community in a rural part of the state. The student had premorbid ADHD and had an IEP in place at school under the qualifying condition of "Other Health Impairment." The child's classification to receive services was ADHD, not TBI. Despite having an IEP in place at school, he was failing most classes because he was unable to pass tests. In a phone conference with the school, it quickly became apparent that although some members of the committee were aware that the child had been in an "accident," no school personnel were familiar with TBI and the sequelae from severe brain injury. In our conversation with the school, we ascertained that the student was consistently failing short answer and essay tests formats. An earlier evaluation of the child had revealed impaired performance on free recall verbal memory and verbal learning measures but

significantly higher performance on recognition or multiple choice format tests. When the child's impairments were discussed with the ARD committee, we suggested the use of recognition format for all examinations. The accommodation was accepted and the student's test performance significantly improved.

# Case 2: Adolescent with Severe TBI

Ann was a 14-year-old adolescent who sustained a severe TBI in a motor vehicle collision. Prior to the TBI, Ann was an honor student who attended a private school. She excelled in academics and was a competitive gymnast. She had an extensive social network and was emotionally welladjusted. Ann had an admission Glasgow Coma Scale score of 6T. A CT of the brain conducted on the day of admission revealed diffuse axonal injury, bilateral frontal lobe contusions, a right subdural hematoma, and an intraventricular hemorrhage. Upon arrival at an inpatient rehabilitation unit, she was not verbal and only minimally responded to commands. After a 2-month stay in rehabilitation, she was discharged with a dense right-sided hemiplegia and dysarthria. Ann's initial neuropsychological evaluation was performed 1 day after discharge from inpatient rehabilitation, roughly 3 months post-injury. This evaluation allowed for documentation of Ann's impairments as well as data that could be used to track her recovery over the next several years. The initial evaluation was limited to 1.5 h of testing because of Ann's fatigue and attentional issues. Ann was not oriented to the day, date, or time. She was unable to name the facility she had been in for the past 2 months. She struggled with completing activities of daily living. She needed help sequencing self-care activities such as showering and brushing her teeth. The brief evaluation assessed her memory skills as well as basic academic skills. As indicated in Fig. 4, Ann had significant impairments on verbal and visual





**Fig. 4** Scaled scores of 9–11 are in the average range

memory tasks on the Wide Range Assessment of Memory and Learning (WRAML). Her performance on the Woodcock-Johnson Tests of Achievement-Revised [87] was well below age and grade expectancies. She had a left visual field cut which greatly impaired her reading. She required cueing to start at the far left side of a sentence, following with her finger till she reached the end. She was unable to consistently recognize numerical operators and struggled with basic multiplication. When she was shown how to solve a math problem, she then was able to solve the next similar problem. Based on the results of this evaluation, we recommended periodic neurobehavioral status examinations, 1:1 aide at school, supervision for mobility, placement in a self-contained classroom, and ongoing speech/language, physical, and occupational therapies. The school requested that an intelligence test be administered to qualify Ann for special education services. Although we agreed to administer the Wechsler Intelligence Scale for Children-III, we indicated in the report that for children with moderate and severe TBI this measure in isolation does not address their unique cognitive issues. Often daily functioning is impeded after TBI by cognitive impairments, such as memory, that intellectual tests are not designed to assess. Typically children will score higher on some factors, such as Verbal Comprehension, than they can demonstrate in day to day functioning. Conversely, motor deficits such as hemiplegia may artificially reduce scores on timed tasks.

Ann received homebound teaching for 1 month following her discharge from rehabilitation and was then transitioned to her public school. Ann's cognitive and physical impairments necessitated educational accommodations and supports. Ann's medical records and neuropsychological evaluation were presented to the school and an ARD meeting was convened to consider the documentation presented by the family. To receive special education services under the TBI classification, the school required Ann's physician to attest to the presence of a TBI as well as describe the nature of her impairments. Ann's IEP contained goals for her basic academic development in reading, math, and written language as well as goals for speech/language, occupational, and physical therapies provided by the school.

Transitioning to the public school was challenging in many ways for Ann. She did not know students from the school prior to her injury and she struggled with learning the layout of the school. We created a memory book for Ann that she carried with her to her classes. The book contained pictures of her current high school, pictures of her teachers and their names, her schedule, and a brief history of Ann (information about herself, her family, and her injury). On the cover of the book was a monthly calendar which was very useful in helping Ann stay oriented to date. Ann frequently referenced this book during her first year at school. Ann was placed in self-contained special education classes which had students with a variety of developmental disabilities. The teaching format in the self-contained classrooms allowed for slower presentation of information. Ann was the only female student and the only student with an acquired brain injury in the self-contained classes. For the first 6 months, Ann had a 1:1 aid throughout the school day. The aid assisted Ann with classroom activities such as finding pages in a textbook, completing worksheets, and taking notes. As Ann's cognitive status improved, the aid's support was gradually reduced and eventually eliminated by 6 months. Because of right-sided hemiplegia, her gait was unsteady and slow. She was unable to transition from class to class safely. After the fulltime aid was discontinued, an aide was assigned to walk her to and from classes and she was allowed to leave classes 10 min early in order to avoid the hallway crowds at period changes. An aide was called to the classroom to escort Ann to the restroom if needed. Ann struggled to learn to write with her right hand but she persevered with the assistance of her occupational therapist and eventually her writing was mostly legible.

A concern for her family was Ann's personal safety at school. She was a young woman who was very trusting of others and could easily be manipulated. These issues were raised with the ARD committee and they agreed that Ann would be supervised in all group settings. Because of


concerns about her safety in the school cafeteria, Ann was allowed to eat lunch in the counselor's office at school, an accommodation she maintained throughout her time in high school.

Ann was re-evaluated 18 months post-injury and these scores are presented in Figs. 4, 5, 6, and 7, along with Ann's baseline scores. Consistent with the literature, marked gains were noted on the Wechsler Intelligence Scale for Children-III Performance subtests [43]. Although Ann's performance improved by at least three scaled points on the three Performance subtests, her scores remained below average. Her performance on Wechsler Intelligence Scale for Children-III verbal subtests remained stable with a significant increase in expressive vocabulary. Ann's verbal and visual memory performance on the Wide Range Assessment of Memory and Language also significantly improved although her scores remained below average. Gains were less dramatic in Ann's academic skills as assessed on the Woodcock-Johnson Tests of Achievement-Revised. Her skills remained well below average. These findings were consistent with Ann's functional gains. By the follow-up evaluation, she was independent for activities of daily living such as bathing and dressing. She was also functioning independently in the classroom but continued to need accommodations such as extended time for completion of tests and assignments, use of a word bank for short answer tests, copies of the teacher's notes, and an aide for transitioning between classes.

Ann's parents struggled with the change in their child's functioning. As is common during the first year following injury, they held on to the belief that it was simply a matter of time before she would return to "normal." The results from our initial evaluation indicated below age and grade skills in most cognitive and academic areas. To help the parents understand the extent of their daughter's injury, we met with the parents and reviewed the various injuries sustained by the brain using a 3D model of the brain and copies of neuroimaging studies. Although the parents had been told of the CT/MRI findings over the course of the Ann's medical hospitalization, the information was presented at time when they were in acute distress about the child's survival and were not able to grasp the information. Understanding the nature of the damage sustained by their daughter's brain, although very upsetting to the parents, helped them eventually come to terms with their daughter's impairments. Often times, information regarding the injury needs to be repeated frequently with increasing details during the child's recovery [88]. Education of parents/guardians of children with TBI is an essential role of the neuropsychologist. Neuropsychological evaluations provide a critical opportunity for the child's strengths and weaknesses to be discussed in depth with the parents, to provide information about the functional impact of these deficits, to recommend appropriate interventions, and assist with long-term planning. It is important to provide parents with a listing of local, state, and national organizations for individuals with TBI. These organizations can provide an opportunity for parents to network with other families of children with TBI for support and to share information.

### Family Environment

Family environment is a crucial factor affecting outcomes. Behavioral, cognitive, and social outcomes from TBI have been found to be

moderated by positive and supportive family environments [28, 76, 89, 90]. At the time of the accident, Ann's parents had been married for 20 years and had three younger children. Her father worked in a management position and her mother was a homemaker. The family dynamics were positive and during the first year post-injury, the focus of the family was on Ann's recovery. The parents worked as a team to meet her needs at home as well as her various therapy appointments. Her family had the financial resources to provide intensive therapies to improve her communication skills and her motility. The family had an extensive support system that helped provide care for the younger children in the family as the parents tended to Ann's needs. Ann's family environment was almost ideal in encompassing positive predictors of recovery. What was not evident to those of us who worked with the family was the toll the injury was taking on the parents themselves. One year after Ann's injury, her parents separated and subsequently divorced. Both parents reported that the stress of Ann's injury and recovery had strained their relationship.

Marital discord following serious illnesses and injuries in children is well documented in the literature and many of the same issues apply to families of children with TBI. Parental stress and family functioning has been found to be related to increased behavioral issues in children with TBI [12, 91]. In Ann's case, she experienced sadness and guilt regarding her parent's divorce and worked with a therapist to process her feelings. When working with a child who has sustained a TBI, it is essential to work with the entire family and support system [92]. Even those in the best circumstances can succumb to the stress and burden of a severe injury.

#### **Transition to Adulthood**

The extent of recovery for children with TBI cannot be assessed until the impact on adult function can be determined [93]. Many individuals with severe TBI have chronic, life-long impairments in executive functions, attention, processing speed, and memory. They face significant challenges as they transition from high school to adulthood. Many young adults with TBI flounder when the familiar structure of school and daily oversight by teachers and parents is withdrawn. When entering college or a vocational program, even academically able students may struggle with the expectations for greater independence in conjunction with fewer built-in supports and less supervision. Societal expectations of independent living may not be readily met by survivors of severe TBI and often, young adults with severe TBI experience social difficulties during their school years and as they transition to adulthood, they experience social isolation. In our experience with adolescents and young adults with TBI, the proliferation of social networking sites has brought both opportunities for social interaction and potential for maladaptive behaviors. For individuals with moderate to severe TBI, impaired judgment and impulsivity can negatively impact integration into social and work environments. Clearly, supports need to be extended well into adulthood.

Several retrospective studies identified the lifelong challenges facing survivors of childhood TBI. Anderson and colleagues [94] conducted a retrospective study of 124 adult survivors of childhood TBI who were injured between the ages of 6 and 12 years. Injury severity ranged from mild to severe with the majority of participants having mild injury. Across the sample, participants were less likely to complete high school and more likely to be unemployed, with one-third of the sample not working. Mental health issues were two times more prevalent in participants with TBI. Less favorable long-term outcome was predicted by greater injury severity, younger age at injury, psychological problems, and inability to complete high school. Cattelani [95] found that social maladjustment and poor quality of life were issues for adults who had sustained TBI in childhood. Telephone interviews conducted with individuals 21 years after they sustained TBI in childhood found frequent reports of psychological and family issues as well as lower educational achievement and poor vocational attainment [96]. In all three studies, injury severity was found to be significantly related to outcome, with greater injury severity associated with worse outcomes.

Many students with significant TBI pursue post-secondary education. Identifying goals for individuals with TBI that are consistent with their long-term aspirations is a key factor in educational achievement [97]. Students with TBI are able to receive accommodations at colleges and universities who accept federal funding because these institutions must adhere to the IDEA. Developing a proper plan for accommodations and support is the first step for students with TBI. Students with TBI often benefit from the following accommodations: note taking assistance, copies of the professor's notes, extended time to complete examinations, access to scribes or computers on tests that require written responses, and audiobooks. Accommodations in college are more challenging because students are often expected to alert their professors to their need for accommodations and arrange for these accommodations. A strong support network is essential for college students with significant TBI. Students with TBI who have been successful in attaining their educational goals often draw support from peers, professors, and family members [50].

Our client, Ann, graduated from high school and was on the honor roll each semester. She expressed a strong desire to attend a large state university but because of her cognitive impairments, attending a 4-year college was not possible. She attended a junior college near her home, taking no more than two classes per semester. The college provided note taking assistance, extended time on examinations, audiotaped books, and modified tests. Her family provided transportation and she worked with a tutor for all her classes. Ann was able to successfully complete an Associate's degree. She entered a job training program with the state's rehabilitation commission and worked for several months at a state agency. With the assistance of a job coach, she was able to manage the demands of a clerical position. However since completing her degree several years ago, she has been unable to secure employment.

#### **Conclusions and Future Directions**

In regards to the Dennis [8] model, young age at injury may be associated with lower brain and cognitive reserves. In general, young children do not recover as well from diffuse or multifocal injury to the brain, such as TBI, as they do from more focal brain insults [98]. TBI sustained early in life may disproportionately reduce cognitive reserve, particularly in general cognitive functioning, learning, social competence, and executive control domains. Young children are very sensitive to environmental influences, such as family resources and parenting style, which can potentiate negative effects of injury or buffer the effects of injury and promote more positive outcomes. Even in young children, behavioral outcomes are influenced by brain injury factors; specific outcomes are related to structural neuroimaging measures characterizing the size and location of abnormalities [99].

School-aged children and adolescents can be viewed as having broader cognitive reserve assets than younger children by virtue of their greater repertoire of pre-injury skills and abilities. Other pre-injury factors also exert major influences on reserve characteristics. For example, pre-injury learning disabilities or psychiatric disorders could be a marker of lower brain and cognitive reserve. Conversely, pre-injury placement in an academic honors program and strong social skills could represent positive reserve factors associated with more favorable cognitive and behavioral outcomes. Cognitive and brain reserve can also be enhanced or diminished by environmental factors, such as level of socioeconomic advantage, access to social and material supports, and the quality of family adaptation. Environmental characteristics, including parenting behaviors, have been related to specific characteristics of children's brain and cognitive development [100–102]. Cognitive and behavioral outcomes are also significantly affected by specific characteristics of the brain injury. To illustrate, children with injury to the superior frontal gyrus in the frontal lobe [71] or to the amygdala [68] would be at increased risk for elevated anxiety. Elevated

anxiety could influence a host of outcomes ranging from social integration and competence to academic performance. Using a personalized approach, anxiety could serve as a specific target of symptom prevention and intervention efforts for this subgroup of patients.

Variables that influence outcomes have potential to inform targets of intervention. These targets may range from providing social and educational supports to children and families after injury, to specific skill-based interventions, to cognitive behavioral interventions to reduce stress and enhance problem solving. Further research is needed to identify factors that positively influence the trajectory of specific outcomes. This research would inform the development of evidence-based interventions that capitalize on variables that promote change. Personalized approaches that tailor patient characteristics, such as integrity of specific brain networks identified through neuroimaging, to specific interventions, such as attention network training, may also enhance recovery.

#### References

- 1. Pierson, E. E., & Noggle, C. A. (2010). Pediatric TBI: Prevalence and functional ramifications. *Applied Neuropsychology*, *17*, 81–82.
- Centers for Disease Control. (2004). National information center for children and youth with disabilities. Traumatic brain injury. Washington, DC: NICCYD.
- Langlois, J. A., Rutland-Brown, W., & Wald, M. M. (2006). The epidemiology and impact of traumatic brain injury: A brief overview. *The Journal of Head Trauma Rehabilitation*, 21, 375–378.
- Langlois, J. A., Rutland-Brown, W., & Thomas, K. E. (2005). The incidence of traumatic brain injury among children in the United States. *The Journal of Head Trauma Rehabilitation*, 20, 229–238.
- Kraus, J. F., Fife, D., & Conroy, C. (1987). Pediatric brain injuries: The nature, clinical course, and deadly outcomes in a defined United States population. *Pediatrics*, 79, 507.
- Guyers, B., & Ellers, B. (1990). Childhood injuries in the United States. *American Journal of Diseases* of Children, 144, 627–646.
- Keenan, H. T., & Bratton, S. L. (2006). Epidemiology and outcomes of pediatric traumatic brain injury. *Developmental Neurosciences*, 28, 256–263.
- Dennis, M., Yeates, K. O., Taylor, H. G., & Fletcher, J. M. (2006). Brain reserve capacity, cognitive

reserve capacity, and age-based functional plasticity after congenital and acquired brain injury in children. In Y. Stern (Ed.), *Cognitive reserve*. New York: Psychology Press.

- Anderson, V., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2005). Functional plasticity or vulnerability after early brain injury? *Pediatrics*, 116, 1374–1382.
- McCauley, S. R., Wilde, E., Hicks, R., Anderson, V. A., Bedell, G., Beers, S. R., et al. (2012). Recommendations for the use of common outcome measures in pediatric traumatic brain injury research. *Journal of Neurotrauma*, 29, 678–705.
- Chevignard, M. P., Brooks, N., & Truelle, J. L. (2010). Community integration following severe childhood traumatic brain injury. *Current Opinion in Neurology*, 23, 695–700.
- Taylor, H. G., Yeates, K. O., Wade, S. L., Drotar, D., Stancin, T., & Burant, C. (2001). Bidirectional childfamily influences on outcomes of traumatic brain injury in children. *Journal of International Neuropsychological Society*, 7, 755–767.
- Levin, H. S., Hanten, G., & Li, X. (2009). The relation of cognitive control to social outcome after paediatric TBI: Implications for intervention. *Developmental Neurorehabilitation*, 12, 320–329.
- Taylor, H. G. (2004). Research on outcomes of pediatric traumatic brain injury: Current advances and future directions. *Developmental Neuropsychology*, 25, 199–225.
- Ewing-Cobbs, L., Kramer, L., Prasad, M., Canales, D. N., Louis, P. T., Fletcher, J. M., et al. (1998). Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics*, 102, 300–307.
- Ewing-Cobbs, L., & Prasad, M. (2011). Outcome of abusive head trauma. In C. Jenny (Ed.), *Child abuse* and neglect. St. Louis, MO: Elsevier.
- 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, 42–57.
- Ewing-Cobbs, L., Miner, M. E., Fletcher, J. M., & Levin, H. S. (1989). Intellectual, motor, and language sequelae following closed head injury in infants and preschoolers. *Journal of Pediatric Psychology*, 14, 531–547.
- Ewing-Cobbs, L., Barnes, M. A., & Fletcher, J. M. (2003). Early brain injury in children: Development and reorganization of cognitive function. *Developmental Neuropsychology*, 24, 669–704.
- Anderson, V. A., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2000). Recovery of intellectual ability following traumatic brain injury in childhood: Impact of injury severity and age at injury. *Pediatric Neurosurgery*, 32, 282–290.
- Taylor, H. G., & Alden, J. (1997). Age-related differences in outcomes following childhood brain insults:

An introduction and overview. *Journal of the International Neuropsychological Society, 3*, 555–567.

- Barnes, M. A., Dennis, M., & Wilkinson, M. (1999). Reading after closed head injury in childhood: Effects on accuracy, fluency, and comprehension. *Developmental Neuropsychology*, 15, 1–24.
- Taylor, H. G., Swartwout, M. D., Yeates, K. O., Walz, N. C., Stancin, T., & Wade, S. L. (2008). Traumatic brain injury in young children: Postacute effects on cognitive and school readiness skills. *Journal of the International Neuropsychological Society*, 14, 734–745.
- Ewing-Cobbs, L., Prasad, M., Kramer, L., Cox, C., Baumgartner, J., Fletcher, S., et al. (2006). Late intellectual and academic outcomes following traumatic brain injury sustained during early childhood. *Journal of Neurosurgery*, 105(Suppl 4), 287–296.
- Koskiniemi, M., Kyykka, T., Nybo, T., & Jarho, L. (1995). Long-term outcome after severe brain injury in preschoolers is worse than expected. *Archives of Pediatrics and Adolescent Medicine*, 149, 249–254.
- Nybo, T., Sainio, M., & Muller, K. (2004). Stability of vocational outcome in adulthood after moderate to severe preschool brain injury. *Journal of the International Neuropsychological Society*, 5, 719–723.
- Gerring, J., & Wade, S. L. (2012). The essential role of psychosocial risk and protective factors in pediatric TBI research. *Journal of Neurotrauma*, 29, 621–628.
- 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, 345–356.
- Potter, J. L., Wade, S. L., Walz, N. C., Cassedy, A., Stevens, M. H., Yeates, K. O., et al. (2011). Parenting style is related to executive dysfunction after brain injury in children. *Rehabilitation Psychology*, 56, 351–358.
- Ganesalingam, K., Yeates, K. O., Taylor, H. G., Walz, N. C., Stancin, T., & Wade, S. (2011). Executive functions and social competence in young children 6 months following traumatic brain injury. *Neuropsychology*, 25, 466–476.
- Sonnenberg, L. K., Dupuis, A., & Rumney, P. G. (2010). Pre-school traumatic brain injury and its impact on social development at 8 years of age. *Brain Injury*, 24, 1003–1007.
- Bayley, N. (2005). Bayley scales if infant and toddler development. San Antonio, TX: Psychological Corporation.
- 33. Mullen, E. M. (1995). *Mullen scales of early learning*. Circle Pines, MN: American Guidance Service.
- 34. Badr, L. K., Garg, M., & Kamath, M. (2006). Intervention for infants with brain injury: Results of a randomized controlled study. *Infant Behavior & Development*, 29, 80–90.

- Beers, S. R., Berger, R. P., & Adelson, P. D. (2007). Neurocognitive outcome and serum biomarkers in inflicted versus non-inflicted traumatic brain injury in young children. *Journal of Neurotrauma*, 24, 97–105.
- Keenan, H. T., Hooper, S. R., Wetherington, C. E., Nocera, M. A., & Runyan, D. K. (2007). Neurodevelopmental consequences of early traumatic brain injury in 3-year-old children. *Pediatrics*, *119*, 616–623.
- Achenbach, T. (1991). Manual for the child behavior checklist/4–18 and 1991 profile. Burlington, VT: University of Vermont College of Medicine.
- Briggs-Gowan, M., & Carter, A. (2006). Brief infant toddler social emotional assessment (BITSEA). San Antonio, TX: Pearson Education.
- Sparrow, S. S., Balla, D. A., & Cicchetti, D. V. (1984). *Vineland adaptive behavior scales*. Circle Pines, MN: American Guidance Service.
- Harrison, P. L., & Oakland, T. (2003). Adaptive behavior assessment system—II. San Antonio, TX: The Psychological Corporation.
- Gioia, G. A., Espy, K. A., & Isquith, P. K. (2000). Behavior rating inventory of executive function— Preschool version. Lutz, FL: Psychological Assessment Resources.
- Korkman, M., Kirk, U., & Kemp, S. (1998). NEPSY: A developmental neuropsychological assessment. San Antonio, TX: The Psychological Corporation.
- Babikian, T., & Asarnow, R. (2009). Neurocognitive outcomes and recovery after pediatric TBI: Metaanalytic review of the literature. *Neuropsychology*, 23, 283–296.
- Catroppa, C., & Anderson, V. (2007). Recovery in memory function, and its relationship to academic success, at 24 months following pediatric TBI. *Child Neuropsychology*, *13*, 240–261.
- Farmer, J. E., Kanne, S. M., Haut, J. S., Williams, J., Johnstone, B., & Kirk, K. (2002). Memory functioning following traumatic brain injury in children with premorbid learning problems. *Developmental Neuropsychology*, 22, 455–469.
- 46. Fay, G. C., Jaffe, K. M., Polissar, N. L., Liao, S., Rivara, J. B., & Martin, K. M. (1994). Outcome of pediatric traumatic brain injury at three years: A cohort study. *Archives of Physical Medicine and Rehabilitation*, 75, 733–741.
- Hawley, C. A. (2004). Behaviour and school performance after brain injury. *Brain Injury*, 18, 645–659.
- Yeates, K. O., Blumenstein, E., Patterson, C. M., & Delis, D. C. (1995). Verbal learning and memory following pediatric closed-head injury. *Journal of the International Neuropsychological Society*, 1, 78–87.
- 49. Ewing-Cobbs, L., Barnes, M. A., Fletcher, J. M., Levin, H. S., Swank, P. R., & Song, J. (2004). Modeling of longitudinal academic achievement scores after pediatric traumatic brain injury. *Developmental Neuropsychology*, 25, 107–133.
- Hux, K., Bush, E., Zickefoose, S., Holmberg, M., Henderson, A., & Simanek, G. (2010). Exploring the

study skills and accommodations used by college student survivors of traumatic brain injury. *Brain Injury*, 24, 13–26.

- Taylor, H. G., Yeates, K. O., Wade, S. L., Drotar, D., & Klein, S. (1999). Influences on first-year recovery from traumatic brain injury in children. *Neuropsychology*, 13, 76–89.
- Catroppa, C., Anderson, V. A., Morse, S. A., Haritou, F., & Rosenfeld, J. V. (2007). Children's attentional skills 5 years post-TBI. *Journal of Pediatric Psychology*, 32, 354–369.
- Hanten, G., Levin, H. S., & Song, J. (1999). Working memory and metacognition in sentence comprehension by severely head-injured children: A preliminary study. *Developmental Neuropsychology*, 16, 393–414.
- Hanten, G., Scheibel, R. S., Li, X., Oomer, I., Stallings-Roberson, G., Hunter, J. V., et al. (2006). Decision-making after traumatic brain injury in children: A preliminary study. *Neurocase*, 12, 247–251.
- Hanten, G., Wilde, E. A., Menefee, D. S., Li, X., Lane, S., Vasquez, C., et al. (2008). Correlates of social problem solving during the first year after traumatic brain injury in children. *Neuropsychology*, 22, 357–370.
- 56. Crowther, J. E., Hanten, G., Li, X., Dennis, M., Chapman, S. B., & Levin, H. S. (2011). Impairments in learning, memory, and metamemory following childhood head injury. *The Journal of Head Trauma Rehabilitation*, 26, 192–201.
- Barnes, M. A., & Dennis, M. (2001). Knowledgebased inferencing after childhood head injury. *Brain* and Language, 76, 253–265.
- Ewing-Cobbs, L., Fletcher, J. M., Levin, H. S., Francis, D. J., Davidson, K., & Miner, M. E. (1997). Longitudinal neuropsychological outcome in infants and preschoolers with traumatic brain injury. *Journal of the International Neuropsychological Society*, *3*, 581–591.
- Jaffe, K. M., Polissar, N. L., Fay, G. C., & Liao, S. (1995). Recovery trends over three years following pediatric traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 76, 17–26.
- 60. Taylor, H. G., Yeates, K. O., Wade, S. L., Drotar, D., Stancin, T., & Minich, N. (2002). A prospective study of short- and long-term outcomes after traumatic brain injury in children: Behavior and achievement. *Neuropsychology*, 16, 15–27.
- Ganesalingam, K., Sanson, A., Anderson, V., & Yeates, K. O. (2007). Self-regulation as a mediator of the effects of childhood traumatic brain injury on social and behavioral functioning. *Journal of the International Neuropsychological Society*, 13, 298–311.
- Walz, N. C., Yeates, K. O., Wade, S. L., & Mark, E. (2009). Social information processing skills in adolescents with traumatic brain injury: Relationship with social competence and behavior problems. *Journal of Pediatric Rehabilitation Medicine*, 2, 285–295.

- 63. Kapapa, T., Pfister, U., Konig, K., Sasse, M., Woischneck, D., Heissler, H. E., et al. (2010). Head trauma in children, part 3: Clinical and psychosocial outcome after head trauma in children. *Journal of Child Neurology*, 25, 409–422.
- 64. Brown, G., Chadwick, O., Shaffer, D., Rutter, M., & Traub, M. (1981). A prospective study of children with head injuries: III. Psychiatric sequelae. *Psychological Medicine*, 11, 63–78.
- 65. Bloom, D. R., Levin, H. S., Ewing-Cobbs, L., Saunders, A. E., Song, J., Fletcher, J. M., et al. (2001). Lifetime and novel psychiatric disorders after pediatric traumatic brain injury. *Journal of the American Academy of Child Adolescent Psychiatry*, 40, 572–579.
- 66. Bombardier, C. H., Fann, J. R., Temkin, N., Esselman, P. C., Pelzer, E., Keough, M., et al. (2006). Posttraumatic stress disorder symptoms during the first six months after traumatic brain injury. *Journal* of Neuropsychiatry and Clinical Neurosciences, 18, 501–508.
- Max, J. E., Robin, D. A., Lindgren, S. D., Sato, Y., Smith, W. L., Mattheis, P. J., et al. (1997). Traumatic brain injury in children and adolescents: Psychiatric disorders at two years. *Journal of the American Academy of Child Adolescent Psychiatry*, 36, 1278–1284.
- Juranek, J., Johnson, C. P., Prasad, M. R., Kramer, L. A., Saunders, A., Filipek, P. A., et al. (2012). Mean diffusivity in the amygdala correlates with anxiety in pediatric TBI. *Brain Imaging and Behavior*, 6, 36–48.
- 69. Max, J. E., Arndt, S., Castillo, C. S., Bond, M., Bokura, H., Robin, D. A., et al. (1998). Attentiondeficit hyperactivity symptomatology after traumatic brain injury: A prospective study. *Journal of the American Academy of Child Adolescent Psychiatry*, 37, 841–847.
- Max, J. E., Castillo, C. S., Robin, D. A., Lindgren, S. D., Smith, W. L., Sato, Y., et al. (1998). Posttraumatic stress symptomatology after childhood traumatic brain injury. *Journal of Nervous and Mental Disease*, *186*, 589.
- Max, J. E., Keatley, E., Wilde, E. A., Bigler, E. D., Levin, H. S., Schachar, R. J., et al. (2011). Anxiety disorders in children and adolescents in the first six months after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 23, 29–39.
- 72. Gerring, J. P., Brady, K. D., Chens, A., Quinn, C., Herskovits, E., Bandeen-Roches, K., et al. (2000). Neuroimaging variables related to development of secondary attention deficit hyperactivity disorder after closed head injury in children and adolescents. *Brain Injury*, 14, 205–218.
- Johnson, C. P., Juranek, J., Kramer, L. A., Prasad, M. R., Swank, P. R., & Ewing-Cobbs, L. (2011). Predicting behavioral deficits in pediatric traumatic brain injury through uncinate fasciculus integrity.

Journal of the International Neuropsychological Society, 17, 663–673.

- Levin, H. S., Zhang, L., Dennis, M., Ewing-Cobbs, L., Schachar, R., Max, J. E., et al. (2004). Psychosocial outcome of TBI in children with unilateral frontal lesions. *Journal of the International Neuropsychological Society*, *10*, 305–316.
- Max, J. E., Levin, H. S., Schachar, R. J., Landis, J., Saunders, A. E., Ewing-Cobbs, L., et al. (2006). Predictors of personality change due to traumatic brain injury in children and adolescents six to twenty-four months after injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 18*, 21–32.
- Yeates, K. O., Taylor, H. G., Wade, S. L., Drotar, D., Stancin, T., & Minich, N. (2002). A prospective study of short- and long-term neuropsychological outcomes after traumatic brain injury in children. *Neuropsychology*, 16, 514–523.
- 77. Glang, A., Todis, B., Thomas, C. W., Hood, D., Bedell, G., & Cockrell, J. (2008). Return to school following childhood TBI: Who gets services? *NeuroRehabilitation*, 23, 477–486.
- Kinsella, G. J., Prior, M., Sawyer, M., Ong, B., Murtagh, D., Eisenmajer, R., et al. (1997). Predictors and indicators of academic outcome in children 2 years following traumatic brain injury. *Journal of the International Neuropsychological Society*, 3, 608–616.
- Rosen, C. D., & Gerring, J. P. (1986). *Head trauma* educational reintegration. San Diego, CA: College-Hill Press.
- Gfroerer, S. D., Wade, S. L., & Wu, M. (2008). Parent perceptions of school-based support for students with traumatic brain injuries. *Brain Injury*, 22, 649–656.
- Glang, A., Tyler, J., Pearson, S., Todis, B., & Morvant, M. (2004). Improving educational services for students with TBI through statewide consulting teams. *NeuroRehabilitation*, *19*, 219–231.
- Ewing-Cobbs, L., & Bloom, D. R. (2001). Traumatic brain injury: Neuropsychological and educational issues. In R. Brown (Ed.), *Handbook of pediatric psychology in school setting*. New Jersey: Lawrence Erblaum Associates.
- Feeney, T. J. (2010). Structured flexibility: The use of context-sensitive self-regulatory scripts to support young persons with acquired brain injury and behavioral difficulties. *The Journal of Head Trauma Rehabilitation*, 25, 416–425.
- Feeney, T. J., & Ylvisaker, M. (1995). Choice and routine: Antecedent behavioral interventions for adolescents with severe traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 10, 67–86.
- Feeney, T. J., & Ylvisaker, M. (2003). Contextsensitive behavioral supports for young children with TBI: Short-term effects and long-term outcome. *The Journal of Head Trauma Rehabilitation*, *18*, 33–51.

- 86. Arroyos-Jurado, E., Paulsen, J. S., Merrell, K. W., Lindgren, S. D., & Max, J. E. (2000). Traumatic brain injury in school-age children: Academic and social outcome. *Journal of School Psychology*, 38, 571–587.
- Woodcock, R. W., & Johnson, M. B. (1989). Woodcock-Johnson—Revised tests of achievement. Itasca, IL: Riverside Publishing.
- Semrud-Clikeman, M. (2010). Pediatric traumatic brain injury: Rehabilitation and transition to home and school. *Applied Neuropsychology*, 17, 116–122.
- Gerrard-Morris, A., Taylor, H. G., Yeates, K. O., Walz, N. C., Stancin, T., Minich, N., et al. (2010). Cognitive development after traumatic brain injury in young children. *Journal of the International Neuropsychological Society*, *16*, 157–168.
- Wade, S. L., Cassedy, A., Walz, N. C., Taylor, H. G., Stancin, T., & Yeates, K. O. (2011). The relationship of parental warm responsiveness and negativity to emerging behavior problems following traumatic brain injury in young children. *Developmental Psychology*, 47, 119–133.
- Limond, J., Dorris, L., & McMillan, T. M. (2009). Quality of life in children with acquired brain injury: Parent perspectives 1–5 years after injury. *Brain Injury*, 23, 617–622.
- Wade, S. L., Carey, J., & Wolfe, C. R. (2006). An online family intervention to reduce parental distress following pediatric brain injury. *Journal of Consulting and Clinical Psychology*, 74, 445–454.
- Forsyth, R. J. (2010). Back to the future: Rehabilitation of children after brain injury. *Archives* of Disease in Childhood, 95, 554–559.
- 94. Anderson, V. A., Brown, S., Newitt, H., & Hoile, H. (2010). Educational, vocational, psychosocial and quality of life outcomes for adult survivors of childhood traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 24, 303–312.

- Cattelani, R., Lombardi, F., Brianti, R., & Mazzucchi, A. (1998). Traumatic brain injury in childhood: Intellectual, behavioural and social outcome into adulthood. *Brain Injury*, 12, 283–296.
- Klonoff, H., Clark, C., & Klonoff, P. S. (1993). Long-term outcome of head injuries: A 23 year follow up study of children with head injuries. *Journal* of Neurology, Neurosurgery and Psychiatry, 56, 410–415.
- 97. Todis, B., & Glang, A. (2008). Redefining success: Results of a qualitative study of postsecondary transition outcomes for youth with traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 23, 252–263.
- Bates, E., Reilly, J. S., Wulfeck, B., Dronkers, N., Opie, M., Fenson, J., et al. (2001). Differential effects of unilateral lesions on language production in children and adults. *Brain and Language*, 79, 223–265.
- Ewing-Cobbs, L., Prasad, M. R., Swank, P., Kramer, L., Mendez, D., Treble, A., et al. (2012). Social communication in young children with traumatic brain injury: Relations with corpus callosum morphometry. *International Journal of Developmental Neuroscience*, 30, 247–254.
- 100. Choi, J., Jeong, B., Rohan, M. L., Polcari, A. M., & Teicher, M. H. (2009). Preliminary evidence for white matter tract abnormalities in young adults exposed to parental verbal abuse. *Biological Psychiatry*, 65, 227–234.
- 101. Hackman, D. A., & Farah, M. J. (2009). Socioeconomic status and the developing brain. *Trends in Cognitive Science*, 13, 65–73.
- 102. Raizada, R. D., Richards, T. L., Meltzoff, A., & Kuhl, P. K. (2008). Socioeconomic status predicts hemispheric specialisation of the left inferior frontal gyrus in young children. *NeuroImage*, 40, 1392–1401.

# Assessment and Treatment of Older Adults with Traumatic Brain Injuries

# Felicia C. Goldstein and Harvey S. Levin

# Abstract

In this chapter, we present special considerations when evaluating the older adult patient with TBI. First, we discuss critical information that should be obtained regarding the patient's preinjury cognitive and functional status as well as medical comorbidities and medications, because all of these can affect the neurobehavioral profile. We then review common clinical issues that arise when interpreting the neuropsychological findings and rendering a diagnostic impression. The potential impact of a previous TBI sustained years ago as a risk factor for Alzheimer's disease will also be covered. Finally, we conclude with a discussion of potential postinjury influences on outcome, and the types of recommendations that should be considered.

Keywords

TBI • Head injury • Aging • Elderly • Dementia • Mild cognitive impairment

At some point in their clinical practice, a neuropsychologist with a geriatric specialization is likely to encounter an older adult who has sustained a traumatic brain injury (TBI) and presents for assessment and treatment recommendations. Although traditionally viewed as a public health problem common in young persons, epidemiological studies indicate that TBI is also frequent in older adults [1, 2]. A Centers for Disease Control and Prevention (CDC) survey of 15 states reported an overall TBI-related hospitalization rate (per 100,000 population) of 155.9 for persons 65 years and older, 187.7 for those 75–84 years, and 85.1 for those  $\geq$ 85 years old. Another CDC survey [2] found that the rate of TBI hospitalizations for persons  $\geq$ 75 years was twice that of any other age group, including those 15–24 years [2]. As noted by the investigators,

F.C. Goldstein, Ph.D., ABPP-Cn (🖂)

Neuropsychology Program, Department of

Neurology, Emory University School of Medicine and Wesley Woods Center on Aging, Atlanta, GA, USA

e-mail: fgoldst@emory.edu

H.S. Levin, Ph.D.

Cognitive Neuroscience Laboratory, Departments of Physical Medicine and Rehabilitation, Neurosurgery and Psychiatry, Baylor College of Medicine and the Michael E. DeBakey Veterans Affairs Medical Center, Houston, TX, USA

their figures likely underestimate the scope of the problem since persons treated in emergency rooms, outpatient facilities, and those who did not seek medical care were excluded from these analyses. The above statistics, coupled with the projection that 72 million people in the United States will be 65 years and older in the year 2030 [3], underscore the importance of neuropsychologists in the assessment and treatment of older adults with TBI.

# Influences on Neurobehavioral Outcome in Older Adults with TBI

Preinjury mild cognitive impairment (MCI) and dementia. Preinjury cognitive changes may be present in the older patient with TBI who is referred for a neuropsychological evaluation, and it is therefore important to enquire about this possibility. An analysis of CDC TBI Surveillance System data [1] found that falls were the leading cause of TBI in persons 65 years and older. Persons  $\geq$ 85 years had a fall rate twice that of persons 65-84 years, and six times the rate of those 65-74 years. Risk factors for falls include MCI and dementia, with some studies demonstrating an association with specific cognitive abilities involving processing speed and executive functioning [4, 5]. Holtzer et al. [4] found that lower scores on a speed/executive function factor that comprise Trail Making [6], Digit Symbol [7], and Block Design [7] were associated with a greater risk for single or recurrent falls. Individuals in the low scoring group were almost four times more likely to fall than individuals in the high scoring group (OR = 3.9, 95 % CI=1.5-10.1, p=0.006). In contrast, a factor examining episodic [8] and semantic [9] memory was not related to increased risk. More recently, Nagamatsu et al. [5] found that persons  $\geq$ 65 years who were at-risk for falls performed more poorly than a nonrisk group on a virtual reality administered task requiring divided attention of crossing the street while talking on the phone. The at-risk group was significantly slower and had more "collisions" with oncoming traffic.

Information concerning preinjury status should ideally be gathered from a significant other in order to reduce the possibility of unreliable estimates by the patient as a result of anosognosia or memory deficit. A number of informant questionnaires are available to identify MCI and dementia [6], and the wording can be modified to clarify that the respondent is being asked about functioning prior to the injury. In our research on older adults with TBI [10-13], we used the Blessed Dementia Scale [14] to enquire about cognitive and personality changes. The Blessed Dementia Scale includes items evaluating changes in the performance of everyday activities (e.g., managing money, recalling recent events), habits (eating, dressing, sphincter control), and personality, interests, and drives (e.g., quality of social interactions, maintenance of hobbies, initiative). Another instrument, the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE), asks about changes over the last 10 years in learning and memory, orientation, financial awareness, and executive skills [15]. The IQCODE is a reliable and validated instrument for the detection of MCI and dementia, with translations available in multiple languages besides English, and evidence for cross-cultural sensitivity to impairment [16, 17]. The Concord Informant Dementia Scale (CIDS) [18] evaluates changes in everyday cognitive functioning over the previous 5 years. Many of the items were taken or adapted from the IQCODE. Domains assess memory, orientation, judgment and problem solving, language, involvement in community affairs and home and hobbies, and personal care. An additional rating form, the Functional Assessment Questionnaire (FAQ), evaluates independence in performing instrumental activities of daily living such as handling complicated financial matters and managing medications [19]. The FAQ was described by the Agency for Health Care Policy and Research as an effective means of identifying demented individuals, with sensitivities and specificities in the 85–90 % range [20].

*Medical comorbidities*. It is important to obtain a health history in order to gauge the independent

effects of certain medical conditions on neurobehavioral outcome after TBI. In their surveillance study of over 17,000 hospitalized TBI patients  $\geq 65$  years old, Coronado et al. [1] noted that approximately 80 % of the patients had comorbid medical conditions, ranging from 1 (23 %) to  $\geq 5$ (6 %). The most frequent conditions included hypertension (39 %), cardiac arrhythmias (18 %), fluid and electrolyte disorders (17 %), and diabetes mellitus (15 %) [1]. Falls are associated with numerous risk factors including disorders of gait, balance, weakness, decreased vision, and peripheral neuropathy [21, 22]. Motor vehicle crashes, the second most common mechanism of TBI in the elderly, are associated with medical conditions including stroke, heart disease, and arthritis as well as certain medications such as benzodiazepines, nonsteroidal anti-inflammatory drugs, and angiotensin-converting enzyme (ACE) inhibitors [23]. Helms et al. [24] analyzed the relationship between chronic health conditions and neuropsychological test performance in 585 elderly participants in the Canadian Study of Health and Aging. The Cumulative Illness Rating Scale (CIRS) [25] was used to determine the presence of 14 chronic illnesses. An increase in the number of conditions predicted poorer performance on measures examining visuospatial and constructional abilities, verbal memory, timed visuomotor sequencing, and set shifting. As the investigators noted, the use of a total CIRS score may have masked additional relationships that existed between specific medical conditions and cognitive performance.

Certain disorders associated with subclinical vascular ischemic disease such as hypertension, diabetes, cardiac disease, and sleep apnea may influence the TBI patient's clinical phenotype. In normal aging, these risk factors are associated with a neurobehavioral syndrome characterized by poorer attention, executive functioning, and information processing speed, as well as depression and personality changes including emotional lability [26–29]. Epidemiological studies indicate that these comorbidities are common in older adults. In the United States, the prevalence of hypertension is 67 % in persons  $\geq$ 60 years old [30]. Patients should be asked whether they have

certain vascular comorbidities. If they respond affirmatively, they should then be asked whether these conditions are well-controlled. The importance of adequate blood pressure control on cogperformance was demonstrated by nitive Waldstein et al. [31] in a cross-sectional study of community residing older adults. Irrespective of a prior diagnosis of hypertension, persons with elevated BP (systolic  $\geq$ 140 mmHg or diastolic  $\geq$ 90 mmHg), versus those with normotensive values at the time of neuropsychological testing, performed more poorly on measures of visual memory, motor speed, and visuomotor integration. Persons with both a prior diagnosis as well as elevated blood pressure levels were most vulnerable to poor performance.

Medical records, if available, may be able to verify the presence of vascular comorbidities because a reliance on self-reports can be inaccurate. The unreliability of self-report data is highlighted by findings from the National Health and Nutrition Examination Surveys which indicate that between 2005 and 2006, 7 % of the US adult population had elevated systolic blood pressure readings  $\geq$ 140 mmHg or diastolic blood pressure readings  $\geq$ 90 mgHg, yet these individuals had not been told by any health care provider that they had high blood pressure. Overall, only 78 % of hypertensive adults were aware they had this condition [30]. It is important to gauge as well whether vascular comorbidities have been adequately controlled. This can be determined by a review of available medical records to examine repeated measures such as blood pressure values. In persons with diabetes, the glycated hemoglobin (A1C) value can provide an index of the extent to which blood glucose levels have been controlled over the past several months [32].

Vitamin deficiencies are also important to assess since they may adversely affect the cognitive status of older persons. The prevalence of Vitamin B<sub>12</sub> deficiency is 10–15 % in persons >60 years [33]. Hin et al. [34] observed a two to three times risk of cognitive impairment, determined via the Mini-Mental State Examination [35], in persons  $\geq$ 75 years who had the lowest levels of serum B<sub>12</sub>. In a more recent study, Tangney et al. [36] found that specific metabolites indicative of  $B_{12}$  deficiency were associated with poorer performance on measures of episodic and semantic memory and perceptual speed in persons  $\geq 65$  years. Low levels of vitamin D can be another source of cognitive impairment in the elderly, notably on measures of executive functioning and processing speed [37, 38]. Low vitamin D levels have also been identified as a risk factor for longitudinal cognitive decline. Llewellyn et al. [39] found that persons >65 years with extremely low levels of vitamin D exhibited a faster decline, relative to those with adequate levels, in their overall cognitive status and set shifting speed over a 6-year follow-up period.

Medications. Medication use and potential sideeffects need to be evaluated in older adults because certain classes can negatively impact neurobehavioral outcome from TBI. Polypharmacy, i.e., multiple medication use, is highly prevalent in the elderly. Hajjar et al. [40] found that surveys of community-residing elderly reported that on average, two to nine prescription medications were taken daily by older adults. In a populationbased study, Kaufman et al. [41] found that during a 1-week period, 57 % of persons 65 years and older took  $\geq$ 5 drugs, whereas 12 % took  $\geq$ 10 drugs. Medications for pain, colds and coughs, and nutrition were among the most frequently non-prescribed medicines.

Medications with anticholinergic properties are used to treat several common age-related conditions including hypertension, cardiovascular and pulmonary diseases, Parkinson's disease, and incontinence. These medications can cause MCI, dementia, and delirium [42]. Moreover, some over the counter drugs such as antihistamines can have anticholinergic as well as sedating effects. In one study, Bottiggi et al. [43] examined the impact of chronic anticholinergic medication use in cognitively normal adults ≥60 years who received annual neuropsychological assessments. The investigators found that compared to a group not treated with these medications, persons taking anticholinergic medications exhibited a significantly greater longitudinal slowing on Trails A and Trails B. Another investigation [44] found

longitudinal declines as a function of continuous use of anticholinergic medications over a 6-year follow-up. Compared to women who had never taken anticholinergic drugs, women in the continuous use group were at higher risk of cognitive decline on measures of timed verbal fluency (OR, 1.5; 95 % CI, 1.1-2.0; P=0.02) and overall mental status (OR, 1.4; 95 % CI, 1.1-1.9; P=0.02). Males with continuous anticholinergic use versus males without such use were at a higher risk for a decline in visual memory (OR, 1.9; 95 % CI, 1.2-3.3; P=0.01) and timed set shifting (OR, 1.9; 95 % CI, 1.1-3.6; P=0.03).

Rating scales exist to characterize drugs according to their anticholinergic properties. One of these, the Anticholinergic Cognitive Burden (ACB) Scale [45], classifies the effects of centrally acting medications according to the severity of their cognitive impact, ranging from mild, moderate, or severe. A copy of this measure, as well as the associated medication list, can be obtained from the following website: http:// www.uea.ac.uk/mac/comm/media/press/2011/ June/Anticholinergics+study+drug+list.

Another similar scale, the Anticholinergic Drug Scale [46] uses a rating system ranging from 0 (no known anticholinergic properties) to 3 (markedly anticholinergic). The ADS has been validated against serum anticholinergic activity, with scores accounting for 9.5 % of the variance in serum activity.

# Common Diagnostic Issues in Older Adults with TBI

Are the extent and persistence of neurobehavioral deficits reasonable given the severity of the *TBI*? The neuropsychologist is commonly faced with the diagnostic dilemma of determining whether the performance of the older adult with TBI is reasonable given the severity of their injury and the time elapsed since their injury. This is an important determination from the standpoint of making effective recommendations and helping the patient and family with future planning. However, there have been relatively few studies examining the neurobehavioral

recovery of older persons to help with diagnostic issues and to estimate the time course of recovery, as much of the available literature has focused on global indices of mortality and functional outcome. At present, and consistent with what is known about outcome in young adults, the handful of studies [10, 11, 47-49] suggest that uncomplicated mild TBI (i.e., no intracranial findings) is associated with subtle deficits in the acute outcome stage, faster cognitive recovery than moderate injuries, and a return to normal cognitive functioning by 1 year. In contrast, patients with complicated mild TBI have outcomes more similar to those with moderate than uncomplicated mild TBI. The important caveat for all these studies is that they are limited to relatively healthy individuals screened for preinjury medical comorbidities, psychiatric conditions, and dementia, and therefore the results cannot be generalized to all patients who are seen in clinical practice.

In an early study, we [11] prospectively recruited patients who were  $\geq 50$  years old with uncomplicated mild head injuries (Glasgow Coma Scale (GCS) [50] scores of 13–15, loss of consciousness <20 min, and normal neurologic and neuroradiologic findings). Patients had sustained head injuries of sufficient intensity to produce a TBI (e.g., striking the head during a fall) and evidence of retrograde and/or posttraumatic amnesia (PTA). A second group of patients with moderate TBI (GCS scores 9-12, or 13-15 with intracranial complications) who had similar demographic features and injury-test intervals as the mild patients were included. Both groups received measures of attention, language, memory, and executive functioning at an average of 1 month postinjury. We found no significant differences in performance between the patients with mild TBI versus community residing controls on any cognitive measure, except for worse performance of the patients on a timed phonemic fluency task. In contrast, the mild patients and controls both performed significantly better than the moderate patients on measures of visuomotor processing speed and set shifting, verbal memory, confrontation naming, reasoning and hypothesis generation.

We subsequently examined possible differences in outcome between patients with and without intracranial complications but comparable GCS scores of 13-15 [10]. Patients were classified as having either uncomplicated mild TBI (GCS scores of 13-15, normal neuroradiologic findings), complicated mild TBI (GCS scores of 13-15, abnormal neuroradiologic findings), or moderate TBI (GCS scores of 9-12 with or without abnormal neuroradiologic findings). The uncomplicated patients with mild TBI performed significantly better on language (naming, fluency) and executive functioning (number of categories) measures than patients with complicated mild TBI. This latter group, in turn, performed similarly to patients with moderate TBI with the exception of faster set shifting ability.

Rapoport et al. [47] prospectively recruited patients  $\geq$ 50 years old who sustained mild TBI (GCS score 13-15, PTA <24 h, loss of consciousness/confusion ≤20 min), and moderate TBI (GCS 9-12, PTA <1 week, or GCS 13-15 with intracranial complication). After controlling for demographic factors and severity of medical comorbidities, it was found that the patients with TBI performed significantly worse than community controls at the 1 year assessment on measures of overall cognitive status, verbal memory, timed letter fluency, visuomotor speed, and expressive language (naming). Post hoc comparisons demonstrated that these cognitive differences occurred between patients with moderate TBI and the controls, but not between mild TBI patients and the controls. In a follow-up study, Rapoport et al. [48] investigated the 2-year cognitive outcome of the same cohort of older TBI adults, and the investigators did not find any significant differences in cognitive performance between the two groups.

Extrapolating from these findings, the neuropsychologist evaluating a relatively healthy older person who has been screened for preinjury cognitive impairment should expect mild TBI patients to exhibit a good outcome in the first year, unless their injury was complicated by intracranial pathology on acute imaging or necessitated urgent surgery. Those with moderate TBI should be performing within normal limits in most domains by about 2 years postinjury. The cognitive outcome and recovery of patients with severe TBI (GCS scores of 3–8) has not been explored, but would be predicted to be less favorable based on what is known about their poor functional outcome. In two prospective series [51, 52], only 8–15 % of patients  $\geq$ 56 years old with severe TBI, defined by GCS scores  $\leq$ 8, exhibited a Good Recovery (resumption of normal activities with possible minor deficits) or Moderate Disability (disabled but independent in daily activities) at 6 months postinjury.

Is the cognitive pattern consistent with TBI or a neurodegenerative disorder such as Alzheimer's disease? The differential diagnosis of cognitive impairments due to TBI versus Alzheimer's disease (AD) is clinically challenging. Questions posed to informants about whether the patient exhibited preinjury cognitive changes may be difficult to accurately recollect or may be biased by the injury itself. Intracranial bleeds from TBI in some older adults can slowly evolve, and therefore informants may describe an insidious onset and progressive course of cognitive changes more characteristic of AD. In other cases, the patient's pre-existing subtle cognitive deficits may be unmasked by the injury, and informants will attribute deficits to the TBI.

In a study to identify neuropsychological features that distinguish AD versus TBI, we [13] compared the cognitive profiles of older adults who sustained mild and moderate TBI or were diagnosed with probable AD. The groups were similar in demographics and overall cognitive status on the MMSE. Patients with TBI were screened for preexisting dementia and were recruited during their initial hospitalization or shortly after discharge while in an early stage of recovery. Both groups received the shortened form of the California Verbal Learning Test [53] requiring them to recall nine words over four trials, and then retain these words over time. Both patient groups demonstrated impaired recall of a word list relative to normal controls. However, those with AD also displayed poorer recall than patients with TBI. Whereas the patients with TBI and normal controls exhibited a nearly equal distribution of recall from the primacy, middle, and recency positions, the patients with AD recalled a significantly higher proportion of words from the end of the list. This latter finding could reflect more rapid forgetting of the earlier items in AD. Performance on letter and category fluency tasks also differentiated the patient groups. Patients with AD, in contrast to those with TBI or normal controls, did not show a normal facilitation in generating words belonging to categories compared with words beginning with specific letters.

Breed et al. [54] compared the cognitive profiles of patients with TBI and AD. Their patients with TBI were older than 55 years at the time of the study, they had been injured an average of 15.8 years previously, and determination of severity of injury was based on self-report information concerning length of loss of consciousness and PTA. The investigators replicated our findings of poorer timed letter fluency and memory functioning in the AD group. The patients with AD exhibited significantly lower percent retention scores for both verbal and visual material relative to TBI and normal controls, whereas the latter groups did not significantly differ from each other. This suggests that rapid forgetting is more characteristic of AD versus TBI.

What if the patient exhibits a delayed or a progressive deterioration in their neurobehavioral status?

(a) *Neurosurgical* complications. Significant others may describe a gradual worsening in the patient's cognitive and functional status, despite the older adult initially exhibiting either minimal sequelae from their TBI or demonstrating improvement over time. In other cases, the patient may have experienced a fall or other accident with no associexternal head trauma, loss ated of consciousness, or confusion. In these scenarios, the neuropsychologist should be alert to the possibility that the patient has developed a neurosurgical complication requiring immediate workup and treatment. Intracranial bleeds, including subdural hematoma (SDH), are more common in older adults due to ageassociated brain atrophy that causes stretching of the bridging veins and shearing effects from the trauma. In addition, because the subdural spaces enlarge with age, the older adult with a SDH can be asymptomatic for a long time, as a relatively large amount of fluid can accumulate before causing cerebral mass effect [55, 56]. Treatment with anticoagulant and antiplatelet medications such as warfarin is also a risk factor for SDH [57– 59]. The result is that older adults may not exhibit obvious changes in their neurobehavioral functioning until weeks or months after their injury, and they may be incorrectly diagnosed as exhibiting a neurodegenerative syndrome such as Alzheimer's disease.

Hydrocephalus is another condition that may account for delayed or worsening symptoms in some patients. Risk factors for posttraumatic hydrocephalus (PTH) include greater severity of injury and older age [60]. PTH can present clinically as the classic triad of normal pressure hydrocephalus, characterized by a Parkinsonian like gait disorder, urinary incontinence, and frontal subcortical cognitive changes of psychomotor slowing, impaired attention, and executive and visuospatial dysfunction. Personality changes of apathy, irritability, and lack of initiative may be mistakenly attributed to depression [61].

(b) Other factors that may contribute to a lack of clinically significant improvement.

Depression is a secondary condition that can adversely impact the neuropsychological profile of older persons with TBI and their long-term functional outcome. In our studies of mild and moderate TBI in persons 50 years and older, we observed that almost 20 % of patients who were not initially depressed at 1 month postinjury endorsed symptoms of new onset depression at 7 months postinjury. Increased depression was associated with a greater decline in social functioning and activities of daily living [62]. In another study [63], relatives of the patients noted a worsening at 1 year compared to 1 month postinjury in the patients' mood such as self-reports of hopelessness, worthlessness, and pessimism concerning the future. Rapoport et al. [47] found major depression to be present in 11/69 (16 %) mildlymoderately injured older adult patients within 2 months postinjury and 6/49 (12 %) at 1 year. Minor depression was observed in 15/69 (22 %) at baseline and 9/49 (19 %) at 1 year. In contrast to the figures for TBI, both minor depression and major depression were found in <4 % of a comparison group of community residing controls.

Late life depression, defined as onset  $\geq 60$ years, is associated with impaired performance in cognitive domains involving episodic and semantic memory, executive functioning, and processing speed [64–68]. Hermann et al. [66] conducted a meta-analysis of published studies comparing cognitive deficits in late onset depression (LOD) versus early onset depression. Although the number of available studies meeting inclusion criteria was small (n=10), the investigators found that executive functioning (working memory, selective attention, cognitive flexibility, set shifting, and planning/problem-solving) and motor and information processing speed (timed visuomotor sequencing) were consistently more affected in the LOD group. In contrast, both groups exhibited comparable deficits in episodic and semantic memory. In the general elderly population, those with LOD have been found to have evidence of greater white matter disease in frontal and subcortical regions, suggesting that disruption of fronto-striatal circuits is one underlying mechanism for the cognitive profile [69].

The implications of the above for the neuropsychological performance of older adults with TBI is that cognitive deficits in domains including memory, executive functioning, and processing speed may be more pronounced when depression is a comorbid condition. Yet, the detection of a mood disturbance in older adults with TBI can be clinically challenging, making it difficult at times to determine whether this is affecting the patient's performance. Some factors that complicate the detection of depression include the finding that elderly persons in general are less likely than younger persons to endorse affective symptoms (e.g., feeling sad) [70]. Therefore, negative answers to these types of symptoms may inadvertently lead the clinician to assume that depression is not present. In those with TBI, the detection of depression is further

complicated by the difficulty in distinguishing symptoms such as apathy and decreased social interaction from behaviors caused by the brain injury (e.g., "frontal lobe" syndrome). Medical conditions such as changes in sleep and appetite, weight loss, and difficulty concentrating may also be misattributed to depression [71].

To facilitate clinical diagnosis, it is recommended that the neuropsychologist attempt to determine the reason for each symptom endorsed by the patient. For example, is the patient not engaging in social activities due to physical restrictions or to a lack of interest? Moreover, given the findings that depression can worsen over time in older persons with TBI, it may be useful to examine the time course of cognitive symptoms, with a worsening perhaps pointing to the contribution of an underlying mood disturbance. Finally, information from collateral sources including significant others may help to better gauge the likelihood that depression is present. In their review of the literature on major depression following moderate and severe TBI, Seel et al. [71] recommend the Patient Health Questionnaire (PHQ-9) as being especially sensitive and specific in the TBI population. The PHQ-9 contains items based on DSM-IV criteria for major depression. The respondent is asked how often they have been bothered by problems in the last 2 weeks such as lack of pleasure, feeling down/depressed/hopeless, and feeling tired/having little energy. There is also an item assessing suicidal ideation. Answers can range from Not at All, Several Days, More than Half the Days, and Every Day. The instrument is nonproprietary and available online (http://www.phqscreeners.com). Translated versions in languages including Spanish are available online as well.

# Associations Between TBI and Alzheimer's Disease in Older Adults

Thus far we have focused on the neurobehavioral effects of TBI sustained in late life. However, there may be occasions when the neuropsychologist is asked to evaluate an elderly patient who is exhibiting new onset cognitive impairment, and the neuropsychologist learns during the interview that the patient has a past history of TBI. Is this history relevant to their current performance? TBI sustained in the younger years as a risk factor for late life cognitive decline, and specifically Alzheimer's disease, has been debated for many years, beginning with the intriguing findings of Roberts et al. [72] of beta amyloid deposition in the brains of 6/16 (38 %) patients who sustained severe TBI and died within 6–18 days postinjury. The investigators argued that since some of these patients were as young as 10 years, it was unlikely that the neuropathological features of AD preexisted. These exciting results sparked tremendous interest which continues today in the relation between TBI and AD.

In 2010, the National Institutes of Health (NIH) held a State-of-the Science Conference titled, "Preventing Alzheimer's Disease and Cognitive Decline" [73]. Prior to this meeting, a panel of experts conducted an evidence-based review of the literature on a myriad of risk factors including TBI. The findings of the NIH may be downloaded at the following website: http://consensus.nih.gov/2010/alz.htm. The NIH panel concluded that there is evidence that the risk of AD is strongest in males and that there is a doseresponse relationship between severity of TBI and risk of AD. The panel first reviewed the findings of a meta-analysis by Fleminger et al. [74] which included 15 case-control studies involving 2,653 subjects, 164 with exposure to TBI and 2,489 without a reported history of TBI. Some inclusion criteria for studies in this meta-analysis required that the definition of TBI entailed loss of consciousness, the TBI occurred prior to onset of AD, and that accepted published guidelines were used for diagnosing AD. It was found that TBI conferred an increased risk of AD in males (OR = 2.29, 95 % CI=1.47-3.58) but not in females (OR=0.91, CI=0.56-1.47). The NIH panel subsequently examined three cohort studies on TBI as a risk factor for AD, with one of the studies [75] finding a positive association (OR = 2.01, 95 % CI=1.03-3.91). In addition, the increased risk of AD was confined to those with moderate (OR=2.32, 95 % CI=1.04–5.17) and severe TBI

(OR=4.51, 95 % CI=1.77–11.47) but not mild TBI (OR=0.76, 95 % CI=0.76, 95 % CI=0.18–3.29). As the panel noted, this latter investigation comprised males only (veterans from WWII). In addition, the ascertainment of TBI may have been more reliable since it was based on a review of military records from the 1940s versus a reliance on self-report in the other two studies.

Additional investigations have examined whether the Apolipoprotein (APOE)  $\varepsilon$ 4 allele, a risk factor for AD, is associated with outcome from TBI. Studies of the association of the  $\varepsilon$ 4 allele with initial severity of TBI and quality of outcome of TBI were reviewed in a meta-analysis by Zhou et al. [76]. Based on a review of 14 cohort studies including 2,527 TBI patients ranging in severity from mild to severe TBI (736 with APOE  $\varepsilon$ 4 and 1,791 without APOE  $\varepsilon$ 4), there was evidence for an association between the presence of APOE  $\varepsilon$ 4 and a worse global outcome at 6 months postinjury as measured by the GOS or the GOS-E.

### Recommendations

This chapter has presented special considerations when evaluating older adults who sustain TBI. While not applicable to every geriatric patient with TBI who is seen for a neuropsychological exam, the following recommendations may be useful:

1. Prevent future falls: Epidemiological evidence reviewed in this chapter indicates that falls are the most common cause of TBI in older persons. As a result of this high risk, the CDC has implemented an initiative focused on preventing fall-related TBI [77]. The CDC website (www.cdc.gov) has several topics dealing with risk factors for falls in the elderly. One site (www.cdc.gov/HomeandRecreationalSafety/ Falls/adultfalls.html) lists strategies for reducing falls such as environmental modifications in the home. The neuropsychologist might recommend that an occupational therapist visit the patient's home to assess whether there is safety equipment such as grab bars in the shower and stair railings, adequate lighting, and lack of clutter.

- 2. Treat medical comorbidities. Certain medical conditions which are risk factors for cognitive decline need to be adequately treated. There is evidence, for example, that diabetes mellitus is associated with cognitive decline and Alzheimer's disease. Lu et al. [78], in their review of prospective population-based studies, found a consistent relationship between diabetes mellitus and a faster decline on measures of executive function, as well as a strong association with incident Alzheimer's disease. Based on a review of additional studies, the NIH panel [74] also concluded that diabetes was a risk factor for AD. Patients should be encouraged to routinely monitor medical conditions. Several relatively inexpensive devices for measuring vascular comorbidities such as hypertension and blood glucose control are available for purchase.
- 3. *Reduce polypharmacy*: It is not uncommon for elderly persons to have several medical specialists. Consequently, the older adult with TBI may be taking a myriad of pharmacologic agents that have adverse interactions with other medications. Sedating drugs such as pain relievers or those with anticholinergic properties such as those used for urinary urgency can interfere with cognitive functioning. Therefore, the older adult should be encouraged to have a primary clinician review their medications and determine whether some can be safely eliminated, in consultation with the prescribing physicians.
- 4. Treat depression. Studies reviewed in this chapter indicate that depression is a frequent secondary condition and may intensify over time in older persons with TBI. Treatment is especially important because cognitive performance can improve after treatment of latelife depression [79]. Butters et al. examined the pre and post-treatment performance on the Mattis Dementia Rating Scale [80] of geriatric patients who were successfully treated with anti-depressant medication for late-onset major depression. The investigators found that those individuals who had some degree of cognitive impairment before treatment showed significant improvements after treatment on the Initiation/Perseveration and

Conceptual subscales, both tapping executive functioning. While treatment of depression may not restore cognitive symptoms to their preinjury status in older adults with TBI, even small gains could be functionally meaningful and also improve quality of life.

- 5. Encourage physical exercise and cognitive stimulation. There is increasing evidence that physical exercise as well as mentally challenging activities have a beneficial effect in reducing the risk for cognitive decline and AD in community residing elderly persons. The NIH panel on Preventing Alzheimer's Disease and Cognitive Decline [74] concluded that cognitive engagement and high levels of physical activity were neuroprotective. There is empirical support for an effect of exercise in reducing neuroinflammation, promoting hippocampal neurogenesis, and improving memory performance in animal models of acquired brain injury [81, 82]. Compelling evidence, the extensive evidence in older adults for a beneficial effect of physical exercise on promoting cardiovascular health, mood, and quality of life is indisputable, and as such, this is a recommendation that should be encouraged during feedback with the patient and family. The American Heart Association (www.heart.org) recommends 150 min of moderate activity or 75 min of vigorous activity a week. Sessions can be divided into smaller durations and spread out over several days.
- 6. External memory aids. For older adults who have executive functioning and memory difficulties after TBI, it should be recommended that a caregiver monitor their safety (e.g., is the patient in danger of leaving on the stove) and other instrumental activities of daily living such as their ability to correctly take their medications. Patients may benefit from external aids such as a pillbox with an alarm and automatic dispensing capabilities. They may also benefit from using notebooks and organizers. A timing system, such as a watch with an alarm (www.cadexwatch.com), can help to prevent forgetfulness with appointments, medications, and other activities, thereby

reducing the stress on caregivers and allowing the patient with TBI to achieve independence. Ideas of other strategies to facilitate cognitive functioning are described in chapter "Rehabilitation of Memory Problems Associated with Traumatic Brain Injury".

#### References

- Coronado, V. G., Thomas, K. E., Sattin, R. W., et al. (2005). The CDC Traumatic Brain Injury Surveillance System: Characteristics of persons aged 65 years and older hospitalized with a TBI. *The Journal of Head Trauma Rehabilitation*, 20, 215–228.
- Coronado, V. G., Johnson, R. L., Faul, M., et al. (2006). Incidence rates of hospitalization related to traumatic brain injury-12 states, 2002. *JAMA*, 295, 1764–1765.
- He, W., Sengupta, M., Velkoff, V. A., et al. (2005). 65+ in the United States:2005. U.S. Census Bureau, current population reports (pp. 23–209). Washington, DC: U.S. Government Printing Office.
- Holtzer, R., Friedman, R., Lipton, R. B., et al. (2007). The relationship between specific cognitive functions and falls in aging. *Neuropsychology*, 21, 540–548.
- Nagamatsu, L. S., Voss, M., Neider, M. B., et al. (2011). Increased cognitive load leads to impaired mobility decisions in seniors at risk for falls. *Psychology and Aging*, 26, 253–259.
- Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual & Motor Skills*, 8, 271–276.
- 7. Wechsler, D. (1981). *The Wechsler Adult Intelligence Scale—Revised manual*. New York: Psychological Corporation.
- Grober, E., Buschke, H., Crystal, H. A., et al. (1988). Screening for dementia by memory testing. *Neurology*, 38, 900–903.
- 9. Goodglass, H., & Kaplan, E. (1983). *The assessment* of aphasia and related disorders (2nd ed.). Philadelphia: Lea & Febiger.
- Goldstein, F. C., & Levin, H. S. (2001). Cognitive outcome after mild and moderate traumatic brain injury in older adults. *Journal of Clinical and Experimental Neuropsychology*, 23, 739–753.
- Goldstein, F. C., Levin, H. S., Goldman, W. P., et al. (2001). Cognitive and neurobehavioral functioning after mild and moderate traumatic brain injury. *Journal of the International Neuropsychological Society*, 7, 373–383.
- Goldstein, F. C., Levin, H. S., Presley, R. M., et al. (1994). Neurobehavioural consequences of closed head injury in older adults. *Journal of Neurology, Neurosurgery, and Psychiatry*, 57, 961–966.
- 13. Goldstein, F. C., Levin, H. S., Roberts, V. J., et al. (1996). Neuropsychological effects of closed head

injury in older adults: A comparison with Alzheimer's disease. *Neuropsychology*, *10*, 147–154.

- Blessed, G., Tomlinson, B. E., & Roth, M. (1968). The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry*, 114, 797–811.
- Jorm, A. F., & Jacomb, P. A. (1989). The Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE): Socio-demographic correlates, reliability, validity and some norms. *Psychological Medicine*, 19, 1015–1022.
- Jorm, A. F. (2004). The Informant Questionnaire on cognitive decline in the elderly (IQCODE): A review. *International Psychogeriatrics*, 16, 275–293.
- Sikkes, S. A., van den Berg, M. T., Knol, D. L., et al. (2010). How useful is the IQCODE for discriminating between Alzheimer's disease, mild cognitive impairment and subjective memory complaints? *Dementia* and Geriatric Cognitive Disorders, 30, 411–416.
- Waite, L. M., Broe, G. A., Casey, B., et al. (1998). Screening for dementia using an informant interview. *Aging, Neuropsychology, and Cognition*, 5, 194–202.
- Pfeffer, R. I., Kurosaki, T. T., Harrah, C. H., Jr., et al. (1982). Measurement of functional activities in older adults in the community. *Journal of Gerontology*, 37, 323–329.
- Costa, P. T., Williams, T. F., Albert, M. S., Butters, N. M., Folstein, M. F., Gilman, S., et al. (1996) *Recognition and initial assessment of Alzheimer's disease and related dementias* (Clinical practice guideline no. 19. AHCPR Publication No. 97-0702). Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research.
- Rubenstein, L. Z. (2006). Falls in older people: Epidemiology, risk factors and strategies for prevention. *Age and Ageing*, 35(Suppl 2), ii37–ii41.
- Thurman, D. J., Stevens, J. A., & Rao, J. K. (2008). Practice parameter: Assessing patients in a neurology practice for risk of falls (an evidence-based review). *Neurology*, 70, 473–479.
- McGwin, G., Sims, R. V., Pulley, L., et al. (2000). Relations among chronic medical conditions, medications, and automobile crashes in the elderly: A population-based case–control study. *American Journal of Epidemiology*, 152, 424–431.
- Helms, E., Ostbye, T., & Steenhuis, R. E. (2011). Incremental contribution of reported previous head injury to the prediction of diagnosis and cognitive functioning in older adults. *Brain Injury*, 25, 338–347.
- Linn, B. S., Linn, M. W., & Gurel, L. (1968). Cumulative illness rating scale. *Journal of American Geriatrics Society*, 16, 622–626.
- Elias, P. K., Elias, M. F., D'Agostino, R. B., et al. (1997). NIDDM and blood pressure as risk factors for poor cognitive performance. The Framingham Study. *Diabetes Care*, 20, 1388–1395.

- Elias, M. F., Orono, M. E., Robbins, M. A., et al. (1998). A longitudinal study of blood pressure in relation to performance on the Wechsler Adult Intelligence Scale. *Health Psychology*, *17*, 486–493.
- Erkinjuntti, T., Inzitari, D., Pantoni, L., et al. (2000). Limitations of clinical criteria for the diagnosis of vascular dementia. Is a focus on subcortical vascular dementia a solution? *Annals of the New York Academy* of Sciences, 903, 262–272.
- Saxby, B. K., Harrington, F., McKeith, I. G., et al. (2003). Effects of hypertension on attention, memory, and executive function in older adults. *Health Psychology*, 22, 587–591.
- Ostchega, Y., Yoon, S. S., Hughes, J., et al (2008) Hypertension awareness, treatment, and controlcontinued disparities in adults: United States, 2005– 2006. NCHS data brief no. 3. Hyattsville, MD: National Center for Health Statistics.
- Waldstein, S. R., Brown, J. R. P., Maier, K. J., et al. (2005). Diagnosis of hypertension and high blood pressure levels negatively affect cognitive function in older adults. *Annals of Behavioral Medicine*, 29, 174–180.
- Executive summary (2010). Standards of medical care in diabetes-2010. *Diabetes Care*, 33(Suppl 1), S4–S10.
- Baik, H. W., & Russell, R. M. (1999). Vitamin B12 deficiency in the elderly. *Annual Review of Nutrition*, 19, 357–377.
- 34. Hin, H., Clarke, R., Sherliker, P., et al. (2006). Clinical relevance of low serum vitamin B<sub>12</sub> concentrations in older people: The Banbury B<sub>12</sub> study. *Age and Ageing*, 35, 416–422.
- 35. 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*, 189–198.
- Tangney, C. C., Aggarwal, N. T., Li, H., et al. (2011). Vitamin B12, cognition, and brain MRI measures: A cross-sectional examination. *Neurology*, 77, 1276–1282.
- 37. Buell, J. S., Scott, T. M., Dawson-Hughes, B., et al. (2009). Vitamin D is associated with cognitive function in elders receiving home health services. *Journals* of Gerontology. Series A, Biological Sciences and Medical Sciences, 264, 888–895.
- Lee, D. M., Tajar, A., Ulubaev, A., et al. (2009). Association between 25-hydroxyvitamin D levels and cognitive performance in middle-aged and older European men. *Journal of Neurology, Neurosurgery,* and Psychiatry, 80, 722–729.
- Llewellyn, D. J., Lang, I. A., & Langa, K. M. (2010). Vitamin D and risk of cognitive decline in elderly persons. *Archives of Internal Medicine*, 170, 1135–1141.
- Hajjar, E. R., Cafiero, A. C., & Hanlon, J. T. (2007). Polypharmacy in elderly patients. *The American Journal of Geriatric Pharmacotherapy*, 5, 345–351.
- 41. Kaufman, D. W., Kelly, J. P., Rosenberg, L., et al. (2002). Recent patterns of medication use in the

ambulatory adult population of the United States: The Slone survey. *JAMA*, 287, 337–344.

- Campbell, N., Boustani, M., Limbil, T., et al. (2009). The cognitive impact of anticholinergics: A clinical review. *Clinical Interventions in Aging*, *4*, 225–233.
- Bottiggi, K. A., Salazar, J. C., Yu, L., et al. (2006). Long-term cognitive impact of anticholinergic medications in older adults. *The American Journal of Geriatric Psychiatry*, 14, 980–984.
- 44. Carrière, I., Fourrier-Reglat, A., Dartigues, J.-F., et al. (2009). Drugs with anticholinergic properties, cognitive decline, and dementia in an elderly general population: The 3-City Study. *Archives of Internal Medicine*, 14, 1317–1324.
- 45. Boustani, M., Campbell, N., Munger, S., et al. (2008). Impact of anticholinergics on the aging brain: A review and practical application. *Aging Health*, 4, 311–320.
- 46. Carnahan, R. M., Lund, B. C., Perry, P. I., et al. (2002). The relationship of an anticholinergic rating scale with serum anticholinergic activity in elderly nursing home residents. *Psychopharmacology Bulletin*, 36, 14–19.
- 47. Rapoport, M. J., Herrmann, N., Shammi, P., et al. (2006). Outcome after traumatic brain injury sustained in older adulthood: A one-year longitudinal study. *The American Journal of Geriatric Psychiatry*, 14, 456–465.
- Rapoport, M., Wolf, U., Hermann, N., et al. (2008). Traumatic brain injury, apolipoprotein E-e4, and cognition in older adults: A two-year longitudinal study. *Journal of Neuropsychiatry and Clinical Neurosciences*, 20, 68–73.
- 49. Stapert, S., Houx, P., De Kruijk, J., et al. (2006). Neurocognitive fitness in the sub-acute stage after mild TBI: The effect of age. *Brain Injury*, 20, 161–165.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, 2, 281–284.
- Gomez, P. A., Lobato, R. D., Boto, G. R., et al. (2000). Age and outcome after severe head injury. *Acta Neurochirurgica*, 142, 373–380.
- Vollmer, D. G., Torner, J. C., Jane, J. A., et al. (1991). Age and outcome following traumatic coma: Why do older patients fare worse? *Journal of Neurosurgery*, 75(Suppl), 37–49.
- Delis, D. C., Kramer, J. H., Kaplan, E., et al. (2000). California Verbal Learning Test (2nd ed.). San Antonio, TX: Psychological Corporation.
- 54. Breed, S., Sacks, A., Ashman, T. A., et al. (2008). Cognitive functioning among individuals with traumatic brain injury, Alzheimer's disease, and no cognitive impairments. *The Journal of Head Trauma Rehabilitation*, 23, 149–157.
- Adhiyaman, V., Asghar, M., Ganeshram, K. N., et al. (2002). Chronic subdural haematoma in the elderly. *Postgraduate Medical Journal*, 78, 71–75.
- Flanagan, S. R., Hibbard, M. R., Riordan, B., et al. (2006). Traumatic brain injury in the elderly:

Diagnostic and treatment challenges. *Clinics in Geriatric Medicine*, 22, 449–468.

- Karni, A., Holtzman, R., Bass, T., et al. (2001). Traumatic head injury in the anticoagulated elderly patient: A lethal combination. *American Surgeon*, 67, 1098–1100.
- Lavoie, A., Ratte, S., Clas, D., et al. (2004). Preinjury warfarin use among elderly patients with closed head injuries in a trauma center. *Journal of Trauma*, 56, 802–807.
- Reynolds, F. D., Dietz, P. A., Higgins, D., et al. (2003). Time to deterioration of the elderly, anticoagulated, minor head injury patient who presents without evidence of neurologic abnormality. *Journal of Trauma*, 54, 492–496.
- Mazzini, L., Campini, R., Angelino, E., et al. (2003). Posttraumatic hydrocephalus: A clinical, neuroradiologic, and neuropsychologic assessment of longterm outcome. *Archives of Physical Medicine and Rehabilitation*, 84, 1637–1641.
- 61. Rousseau, G. (2011). Normal pressure hydrocephalus. *Disease-A-Month*, 57, 615–624.
- Levin, H. S., Goldstein, F. C., & MacKenzie, E. J. (1997). Depression as a secondary condition following mild and moderate traumatic brain injury. *Seminars in Clinical Neuropsychiatry*, 2, 207–215.
- 63. Goldstein, F. C., Levin, H. S., Goldman, W. P., et al. (1999). Cognitive and behavioral sequelae of closed head injury in older adults according to their significant others. *Journal of Neuropsychiatry and Clinical Neurosciences*, *11*, 38–44.
- Alexopoulos, G., Kiosses, D. N., Heo, M., et al. (2005). Executive dysfunction and the course of geriatric depression. *Biological Psychiatry*, 58, 204–210.
- Butters, M. A., Whyte, E. M., Nebes, R. D., et al. (2004). The nature and determinants of neuropsychological functioning in late-life depression. *Archives of General Psychiatry*, *61*, 587–595.
- Herrmann, L. L., Goodwin, G. M., & Ebmeier, K. P. (2007). The cognitive neuropsychology of depression in the elderly. *Psychological Medicine*, 37, 1693–1702.
- Nebes, R. D., Butters, M. A., Mulsant, B. H., et al. (2000). Decreased working memory and processing speed mediate cognitive impairment in geriatric depression. *Psychological Medicine*, 30, 679–691.
- Thomas, A. J., Gallagher, P., Robinson, L. J., et al. (2009). A comparison of neurocognitive impairment in younger and older adults with major depression. *Psychological Medicine*, 39, 725–733.
- Lesser, I. M., Boone, K. B., Mehringer, C. M., et al. (1996). Cognition and white matter hyperintensities in older depressed patients. *The American Journal of Psychiatry*, 153, 1280–1287.
- Alexopoulos, G. S., Borson, S., Cuthbert, B. N., et al. (2002). Assessment of late life depression. *Biological Psychiatry*, 52, 164–174.
- 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 and Trauma Rehabilitation*, 25, 99–112.

- Roberts, G. W., Gentleman, S. M., Lynch, A., et al. (1991). Beta A4 amyloid protein deposition after head trauma. *Lancet*, 338, 1422–1423.
- Williams, J. W., Plassman B. L., Burke, J., Holsinger, T., & Benjamin, S. (2010). *Preventing Alzheimer's* disease and cognitive decline. Evidence report/technology assessment no. 193. (Prepared by the Duke Evidence-based Practice Center under Contract No. HHSA 290-2007-10066-I.) AHRQ Publication No. 10-E005. Rockville, MD: Agency for Healthcare Research and Quality.
- 74. Fleminger, S., Oliver, D. L., Lovestone, S., et al. (2003). Head injury as a risk factor for Alzheimer's disease: The evidence 10 years on; a partial replication. *Journal of Neurology, Neurosurgery, and Psychiatry*, 74, 857–862.
- Plassman, B. L., Havlik, R. J., Steffens, D. C., et al. (2000). Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias. *Neurology*, 55, 1158–1166.
- Zhou, W., Xu, D., Peng, X., et al. (2008). Metaanalysis of APOE4 allele and outcome after traumatic brain injury. *Neurotrauma*, 25, 279–290.

- 77. Sarmiento, K., Langlois, J. A., & Mitchko, J. (2008). Help seniors live better, longer: prevent brain injury. An overview of CDC's education initiative to prevent fall-related TBI among older adults. *The Journal of Head Trauma Rehabilitation*, 23, 164–167.
- Lu, F. P., Lin, K. P., Kuo, H. K., et al. (2009). Diabetes and the risk of multi-system aging phenotypes: A systematic review and meta-analysis. *PLoS One*, *4*, 1–12.
- Butters, M. A., Becker, J. T., Nebes, R. D., et al. (2000). Changes in cognitive functioning following treatment of late-life depression. *The American Journal of Psychiatry*, 157, 1949–1954.
- Mattis, S. (1988). Dementia Rating Scale (DRS). Odessa, FL: Psychological Assessment Resources.
- Devine, J. M., & Zafonte, R. D. (2009). Physical exercise and cognitive recovery in acquired brain injury: A review of the literature. *PM & R, 1*, 560–575.
- Archer, T., Svensson, K., & Alricsson, M. (2012). Physical exercise ameliorates deficits induced by traumatic brain injury. *Acta Neurologica Scandinavica*, *125*, 293–302.

Part V

Mild TBI

# **Mild Traumatic Brain Injury**

# William B. Barr

# Abstract

There is nothing in the field of neuropsychology that is more divisive than the topic of mild traumatic brain injury (MTBI). While most will agree that an MTBI can be defined in its most basic terms as a traumatically induced alteration in mental status resulting from a physiological disruption of the brain, there is little consensus in the field about the natural course of recovery and whether persisting symptoms of MTBI are attributed to continuing effects of brain dysfunction, a result of a psychological reaction to injury, or an attempt to obtain a secondary gain through litigation or some other mechanism. Furthermore, the controversy surrounding MTBI has been heightened over the past decade through media accounts of brain injuries sustained by athletes and those serving in the military, providing the public with views that differ from what is present in the professional literature. The positive result of this exposure has been to increase public awareness about MTBI, which has provided investigators in the field the resources to perform a number of controlled investigations on athlete and military samples. The aim of this chapter is to provide a concise review on the research literature on MTBI in addition to a simple evidence-based approach to neuropsychological assessment and intervention, informed primarily by findings from studies on sports-related injuries.

#### Keywords

Mild traumatic brain injury • Concussion • Sport-related concussion • Neuropsychological assessment • Performance validity testing • Symptom validity testing • Cognitive behavioral therapy

W.B. Barr, Ph.D., ABPP (⊠) Department of Neurology, NYU Langone Medical Center, New York, NY, USA

NYU School of Medicine, New York, NY, USA e-mail: William.Barr@nyumc.org There is nothing in the field of neuropsychology that is more divisive than the topic of mild traumatic brain injury (MTBI). While most will agree that an MTBI can be defined in its most basic terms as a traumatically induced alteration in mental status resulting from a physiological disruption of the brain, there is little consensus in the field about the natural course of recovery and whether persisting symptoms of MTBI are attributed to continuing effects of brain dysfunction, a result of a psychological reaction to injury, or an attempt to obtain a secondary gain through litigation or some other mechanism. Furthermore, the controversy surrounding MTBI has been heightened over the past decade through media accounts of brain injuries sustained by athletes and those serving in the military, providing the public with views that differ from what is present in the professional literature. The positive result of this exposure has been to increase public awareness about MTBI, which has provided investigators in the field the resources to perform a number of controlled investigations on athlete and military samples. The aim of this chapter is to provide a concise review on the research literature on MTBI in addition to a simple evidence-based approach to neuropsychological assessment and intervention, informed primarily by findings from studies on sports-related injuries.

#### **Challenges in the Definition of MTBI**

According to data provided by the Centers for Disease Control (CDC), approximately 80 % of the 1.7 million traumatic brain injuries sustained yearly in the United States are mild in nature [20]. However, the most commonly reported statistics, utilizing data obtained based on emergency department visits, hospitalizations, and death, are likely to provide an underestimate of the total number of MTBIs, given the fact that many individuals sustaining this level of injury do not seek hospital-based evaluation or treatment [40]. It remains unclear how many individuals experiencing an MTBI are evaluated through outpatient medical offices and it would be next to impossible to determine how many individuals with this injury never report it to health professionals. Therefore, our understanding of the epidemiology of MTBI remains limited as a result of what McCrea [40] labels the "denominator problem," meaning that we cannot fully assess the scope and severity of the MTBI problem without knowing the total number of individuals who actually experience this type of injury.

Our ability to understand and monitor MTBI is limited further by a lack of clarity regarding its definition and its relation to the term "concussion." While some feel that there is no level of brain injury that should be considered "mild," the term MTBI is necessary as a label to distinguish this level of injury from "moderate" and "severe" levels of traumatic brain injury, both of which differ significantly from MTBI in their course and long-term prognosis. With regard to the term concussion, most will agree that it is not entirely synonymous with the term MTBI. By definition, all concussions are MTBIs. However, it is clear that not all MTBIs are concussions, particularly when one considers those rare cases where brain abnormalities, such as intracranial bleeds, are visualized on neuroimaging. Historically, the term concussion had been applied mostly in a medical setting, whereas the MTBI emerged from more of a research context [32]. However, in recent years, the convention has shifted toward using MTBI on a wider basis, except in the context of sports, where the use of concussion is preferred, reflecting the more limited scope of injury encountered in that setting. The term MTBI will be used for most of this chapter, in an effort to distinguish its contents from those chapters in this volume discussing other types of traumatic brain injury.

There have been a number of attempts to formally define MTBI through criteria developed by consensus panels or through professional organizations. The American Congress of Rehabilitation Medicine [1] definition has been the one most commonly used in the field of neuropsychology. In the ACRM definition, MTBI is characterized as "a traumatically induced physiological disruption of brain function" that results in a loss of consciousness (LOC), memory loss, or any alteration in mental status at the time of the accident (e.g., feeling dazed or confused). This definition emphasizes specific characteristics of the acute injury, including loss of consciousness (LOC<30 min) and post-traumatic amnesia (PTA < 24 h) in addition to providing clinicians with classification criteria based on scores from the Glasgow Coma Scale (GCS) [69]. A GCS score ranging from 13 to 15 is used to distinguish MTBI from more serious levels of "moderate" and "severe" brain injury. The ACRM criteria extend the scope of MTBI to include focal deficits elicited on neurological exam in addition to non-operative brain lesions observed on structural neuroimaging.

In recent years, the field has begun to move away from defining MTBI in terms of acute injury characteristics. While, at one time, the public and many professionals held the erroneous belief that LOC was a necessary component for a diagnosis of traumatic brain injury, the results of controlled investigations have demonstrated that LOC occurs in less than 10 % of the subjects with an MTBI diagnosis, while no signs of LOC or PTA are seen in over 70 % [28]. This has led, by default, to more of an emphasis on establishing, in more empirical terms, what constitutes an alteration in mental status. In 1997, a Practice Parameter from the American Academy of Neurology (AAN) defined concussion as a "trauma induced alteration in mental status that may or may not involve loss of consciousness" [31]. In a more recently published position statement, traumatic brain injury is defined in more general terms as "an alteration in brain function, or other evidence of brain pathology, caused by an external force," with further guidelines offered to define the evidence supporting a diagnosis of MTBI [45]. While most clinicians will agree that dizziness and confusion would represent symptoms of an MTBI in the setting of a head injury, the challenge lies in defining the lower limits of injury, when no such symptoms are directly observed or reported. Conversely, there remain challenges in defining the upper end of what constitutes a more severe level of MTBI, which would essentially differentiate it from a moderate level of traumatic brain injury, as in the case where a small intracranial bleed is visualized on CT imaging.

For clinical purposes, many neuropsychologists are now using the more detailed injury definitions provided in the sports concussion 
 Table 1
 Definition of concussion—consensus statement

 on concussion in sport: the third international conference
 on concussion in sport held in Zurich, November 2012

Concussion is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological and biomechanical injury constructs that may be utilized in defining the nature of a concussive head injury include:

- Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an "impulsive" force transmitted to the head
- Please provide 'Abstract' for this chapter.Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours
- Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies
- 4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. However, it is important to note that, in some cases, symptoms may be prolonged

With permission by McCroryet al. [46]

literature. While over the past 15 years there has been a plethora of definitions and grading scales developed for use in a sports setting, there has been some movement toward use of the evolving set of definitions included consensus statements developed through a series of International Conferences on Concussion in Sport [45]. The definition emanating from the most recent conference, held in Zurich in 2012, is provided in Table 1. In this definition, concussion is defined as "a complex pathophysiological process affecting the brain, induced by biomechanical forces" and extends to a brief description of possible causes and effects in addition to the expected course of recovery. In conjunction with a number of assessment tools outlined in the consensus statement, the criteria specified in this definition can be used in a model emphasizing a more empirical and multidimensional approach to documenting the signs and symptoms of injury.





# Pathophysiology and Natural Course of Recovery from MTBI

The scientific literature on MTBI supports a functional rather than a structural etiology [45]. The model commonly used to understand the neurophysiological basis of MTBI, developed on the basis of animal models, is conceptualized as a multilayered neurometabolic cascade, involving a complex of interwoven cellular and vascular changes that occur following trauma to the brain [22]. According to this model, the pathophysiology of MTBI represents a temporary disruption of brain function secondary to ionic fluxes, abnormal energy transmission, diminished cerebral blood flow, and impaired neurotransmission rather than any readily identifiable form of structural brain damage. According to most accounts the neurophysiological changes associated with the acute stage of MTBI clear within a period of 7–10 days [41, 44].

Results from research on humans with MTBI have demonstrated a rather remarkable correspondence to the findings reported from animal studies. The evidence obtained through controlled investigations, using primarily athlete samples, have demonstrated a gradual resolution of symptoms, including headache, dizziness, and cognitive disturbance, within a period of 1–7 days (see Fig. 1). Findings from these and other studies led several panels, including the World Health Organization (WHO) Collaborating Centre Task Force on Mild Traumatic Brain Injury, to con-

clude that symptoms of MTBI are generally temporary and self-limiting in nature, with resolution observed within days to weeks post-injury in an overwhelming majority of cases [15].

From a structural standpoint, it appears that most of the injuries classified as MTBI are associated with, at most, low levels of axonal stretching, resulting in the temporary neurophysiological changes described above [22]. There is now accumulating evidence, obtained from studies using advanced neuroimaging (e.g., fMRI, nuclear magnetic resonance (NMR) spectroscopy, and diffusion tensor imaging (DTI)) and electrophysiological (e.g., quantitative EEG and evoked potentials) techniques indicating that signs of this non-permanent form of traumatic axonal injury can be detected in some subjects for up to 30–40 days following injury [19, 38, 73]. From a clinical standpoint, this might represent a period where the brain is more vulnerable to re-injury or effects of fatigue with suggestions that the brain might also require recruitment of additional neuronal resources to achieve its typical level of functioning [43]. From a clinical standpoint, this might represent the underlying cause for what many patients report as difficulties with resuming their pre-injury level of activity. However, it should be emphasized that this stage of recovery is temporary with no empirical evidence of any pathophysiological abnormalities persisting for more than a several week period. Contrary to what some believe, there is little evidence supporting the existence of any





more serious form of diffuse axonal injury (DAI) resulting from a typical MTBI [3].

Patients reporting symptoms of MTBI extending for more than several weeks post-injury are often classified as having post-concussion syndrome (PCS). The exact number of patients reporting PCS remains controversial. While some argue for the existence of a "miserable minority" consisting of approximately 15 % of MTBI victims [3, 57], the results of most prospective studies and meta-analyses indicate that the number is more likely to be closer to 3 % [63]. There is no scientific evidence supporting claims that PCS symptoms are the results of direct physiologic effects of brain injury. The conclusion from the WHO task force was that symptoms extending beyond the typical recovery of several days to weeks are attributable to a number of "noninjury" factors such as depression, PTSD, chronic pain, life stress, or secondary gain [15]. There has been recent interest in identifying a number of psychological factors (e.g., misattribution, nocebo effect, "good-old-days" phenomena) underlying the tendency to report persisting symptoms following MTBI [27, 51].

Additional controversy surrounding MTBI has developed following reports of symptoms and neuropathological changes associated with dementia appearing in a small number of athletes exposed to repetitive head injury while participating in contact sports including football, boxing, and ice hockey [49]. While an

association between more severe forms of TBI and the occurrence of Alzheimer's disease has been established [67], investigators are claiming that the pattern of behavioral decline seen in this sample of athletes, in association with its unique underlying profile of pathology represents a distinctive form of dementia known as chronic traumatic encephalopathy (CTE) [49]. Based on existing evidence, the development of CTE does not appear to be related to the occurrence of discrete MTBI events, but rather the cumulative effects of repeated "sub-concussive" blows to the head. At this point, the validity of the CTE diagnosis and its relevance to the vast majority of MTBI victims remains unclear [48, 59].

In summary, the emerging evidence-based model of neurophysiological recovery from MTBI can be characterized as having three possible stages, which are depicted in Fig. 2. The first of these stages, characterized as an acute period of recovery, is based on temporary neurophysiological effects that recover within a period of 7-10 days, along with most clinical signs and symptoms. The second, "sub-acute" stage is characterized by continued brain recovery, which can be identified potentially through the use of advanced neurodiagnostic techniques obtained within a period of several weeks following the injury before full remission. Subsequently, there is a possibility of a third stage of recovery, explaining the potential for some individuals to experience a longer-term susceptibility to repeat injury and perhaps even the possibility of developing dementia later in life. While continued research is needed to confirm the presence and nature of these three stages of recovery, clinicians will find it useful to consider these stages of recovery when performing neuropsychological evaluations of individuals following a reported MTBI.

# Neuropsychological Assessment of MTBI

Patients with MTBI present with a complex combination of physical, cognitive, and emotional symptoms. When examining these individuals, neuropsychologists need to be aware of the evidence-based literature on recovery from MTBI and to be cognizant of which stage of recovery the individual falls in at the time of the assessment. With the exception of individuals working in an acute concussion clinic, sport setting, or the military, it is likely that most neuropsychologists will encounter MTBI patients long after the occurrence of the injury when patients are reporting symptoms of PCS. In that context, neuropsychologists are, by virtue of their training and use of empirically based assessment methods, uniquely qualified among health care professionals to assess the complex display of symptoms seen in individuals presenting with PCS.

When evaluating individuals with MTBI, it is important to be reminded of the distinction between neuropsychological testing and neuropsychological assessment [37]. There is perhaps no other clinical situation where it is more important to corroborate information obtained via self-report through other sources. The comprehensive assessment of an individual with MTBI should include a detailed record review, an interview with the patient, testing of neurocognitive functioning, and completion of self-report symptom inventories. Interviews with collateral sources, such as family members, employers, or witnesses to the injury are also helpful, if such individuals are available, cooperative, and knowledgeable about the patient and the reported injury.

#### **Record Review**

The level and detail of a record review performed by neuropsychologists will vary depending on the setting and situation. The most useful records will consist of those documenting the characteristics of the initial injury. These will include reports from the ambulance team, emergency room, or notes from the initial visit to the patient's primary care physician, if they received no acute hospital care following the injury. One will want to know whether there was any documented LOC or any observed alteration in behavior, as documented by trained medical professionals. It is always important to record the reported GCS score, as that continues to be the metric used for documentation of injuries in most settings.

The records will also include a description of the mechanism and severity of the injury that was reported at the time of its occurrence. There will also be important information regarding the patient's report of symptoms developing within the first few hours of the injury and whether there was treatment for any lacerations or other physical injuries involving the head or other parts of the body. Lastly, the acute injury records will include documentation of whether the individual exhibited any neurological signs or symptoms on direct examination or whether any abnormal findings were obtained on neuroimaging.

In terms of additional records, it is important to obtain documentation of the individual's subsequent medical care, particularly during the first few weeks following the injury. Again, it is important to determine the nature and level of symptoms reported during that time period. It is also important to determine whether or not there were any referrals to a neurologist or any other type of concussion specialist. In terms of pre-injury functioning, the neuropsychologist should make efforts to obtain records from medical, academic, employment, and military settings to evaluate any reported changes in post-injury functioning in addition to factors that could predispose the individual to a complicated recovery. There are indications that patients with pre-existing psychiatric conditions and/or issues with pain or other chronic medical complaints will have a more challenging recovery from MTBI [64].

# **Clinical Interview**

The clinical interview provides the neuropsychologist with an excellent opportunity to obtain useful information while making critical observations of the patient's demeanor. The patient should always be asked his or her account of the injury and its immediate impact on consciousness and behavior. It is important to make the distinction between what the individual directly recalls from what they had learned about the injury through other sources. One should be in a position to compare information provided by the patient to the contents of the medical records. Given what is known about the natural recovery from concussion, it is important to obtain a detailed description of symptoms emerging within the first few days following the injury and whether there was any full or partial resolution of the symptoms after a period of 1 week. The neuropsychologist will need to understand the symptoms experienced by the patient at the time of the assessment and understand how these symptoms are affecting the individual's ability to return to work or school.

#### Neuropsychological Test Battery

In the age of health care reform and efforts to reduce costs and increase efficiency, neuropsychologists working in a clinical setting should be in a position to assess conditions such as MTBI with a rather brief and focused test battery. While patients might report a wide range of symptoms following injury, findings from the evidence-based literature indicate that attention, processing speed, and memory are the functions most commonly affected following MTBI and these are the functions that should receive the most attention through neurocognitive testing [7]. More detailed assessment of MTBI through a comprehensive 3- to 5-h neuropsychological test battery is only likely to be needed in a forensic or disability setting.

Results of meta-analyses indicate that the cognitive deficits following MTBI are detected most readily within the first few weeks following the injury [7]. The evidence does not support the existence of long-term effects on cognitive functioning directly resulting from the physiological effects of any lasting brain injury. In fact, given the effect sizes reported in meta-analyses, cognitive impairment attributable to the effects of MTBI, if present, would be undetectable using neuropsychological testing or any other known methodology.

Based on this information, the purpose of the neuropsychological evaluation of patients following MTBI will be very different depending on the time the evaluation is performed in relation to the injury. An assessment performed within weeks of the injury would by nature focus on detection of cognitive impairment while an evaluation performed months or years following the injury would be more concerned with determining a combination of factors that are likely to be playing a role in the patient's reporting of chronic symptoms.

Ironically, when assessing the chronic effects of MTBI, the neuropsychologist should be more in a position of providing assurance and communication that the results of testing indicate no long-term cognitive consequences of brain injury, contrary to what is often reported by other health care professionals. The goal is to provide the patient with evidence-based information on recovery that will help them return to their preinjury level of functioning rather than prepare them for a long-term course of rehabilitation. The end result of the neuropsychological evaluation will be to provide to the patient and the treatment team an explanation on factors other than the physiological effects of "brain damage" that are likely to be playing a role in the maintenance of persisting symptoms and how those factors can be addressed through appropriate psychological intervention or other forms of rehabilitation.

Over the past 20 years, a number of brief neuropsychological assessment paper/pencil or computerized test batteries have been developed for assessment of patients with MTBI. It is important to note that most of these test batteries were developed for assessment of the acute injury effects in a sport setting in conjunction with data obtained from baseline testing performed before the injury. These test batteries are typically not comprehensive enough to be used for evaluations of patients in most standard clinical settings.

 Table 2
 Neuropsychological test battery for assessment

 of mild traumatic brain injury (MTBI)—NYU Langone
 Medical Center Concussion Center

- Wechsler Abbreviated Scale of Intelligence (WASI-2)
- Test of Premorbid Functioning (TOPF)
- Digit Span (WAIS-IV)
- Digit Symbol (WAIS-IV)
- Trail Making Test (DKEFS)
- Color Word Interference Test (DKEFS)
- Verbal Fluency Test (DKEFS)
- California Verbal Learning Test (CVLT-2)
- Medical Symptom Validity Test (MSVT)
- Reliable Digit Span (RDS)
- Forced Choice Recognition Trial (CVLT-2)
- Post-Concussion Scale (PCS-R)
- Beck Anxiety Inventory (BAI)
- Beck Depression Inventory (BDI-II)
- Minnesota Multiphasic Personality Inventory (MMPI-2-RF)

While there are clearly a number of advantages to using computerized tests, particularly with regard to assessment of reaction time and use of automated scoring and recording methods, there are no data indicating that these tests are any more sensitive to detecting cognitive impairments following MTBI than standard paper/pencil tests [60]. With emerging evidence regarding limitations in the reliability of these computerized test batteries and concerns about their falsepositive rates, clinicians are urged to use caution in employing these measures in most standard clinical settings until more information about psychometrics and norms is obtained [12, 62].

The neuropsychological test battery used for evaluating patients with MTBI in a non-sports setting can be conducted effectively using a brief battery of paper and pencil tests consisting of four major components. To begin with, the battery should provide a detailed assessment of cognitive functioning, with a particular focus placed on assessment of attention, processing speed, and memory. Given the range of motivational factors associated with MTBI, the battery should include multiple measures of performance validity using a combination of freestanding and embedded measures. Lastly, the neuropsychologist should employ standardized methods for evaluating symptom reporting. This will often include use of a combination of brief measures of postconcussion symptoms and mood in addition to larger inventories including formal indices for assessing symptom validity. An example of a test battery including measures of cognitive functioning, performance validity, self-report, and symptom validity used by neuropsychologists at the Concussion Center at the NYU Langone Medical Center is provided in Table 2.

Testing of cognitive functioning in patients following MTBI will often begin with a brief assessment of intellectual functioning. This might include the use of a combined reading and demographic index of premorbid functioning, such as the Test of Premorbid Functioning (TOPF) [56] or the Wechsler Test of Adult Reading (WTAR) [76]. A brief measure of current intellectual functioning such as the twosubtest version of the Wechsler Abbreviated Scale of Intelligence (WASI-2) [78] will usually suffice for assessment of current intellectual functioning, although use of the full IQ test might be required in certain forensic applications or when evaluating the need for accommodations in the workplace or school. The overall purpose of evaluating intelligence in this population is to obtain a context to interpret other test indices since there is no evidence that MTBI affects intelligence in any manner that would lead directly to a decline in intellectual functioning.

Formal assessment of attention in patients following MTBI will include measures of attention span, processing speed, and more complex attentional control. In most cases, this will begin with a measure of digit span as obtained with a subtest obtained from one of the Wechsler scales. Processing speed can be assessed effectively using one of the Wechsler coding tests or through the use the Symbol Digit Modalities Test [68]. More complex forms of attention can be evaluated with a combination of the Trailmaking Test [61] and Stroop Color Word Naming Test [23]. Further assessment might include a verbal fluency measure such as the Controlled Oral Word Association Test (COWAT) [10]. From a theoretical perspective, there is no evidence that higherorder executive functions are affected directly following MTBI. Therefore, for the sake of brevity, measures such as the Wisconsin Card Sorting Test (WCST) [29] or Category Test [61], do not typically add much value in an assessment of an individual following that level of injury.

While some patients might report specific changes in language and academic skills such as reading and spelling, there is no evidence that those functions would be affected directly or persistently through any known physiological effects of MTBI. Reports of reading and spelling disorders are more often the result of secondary effects of attentional issues stemming from anxiety and/ or distractions from somatic symptoms such as headache or pain. As a result, one might question the need to include any formal assessment of language or academic skills in within the context of a routine neuropsychological evaluation performed on an adult following MTBI. Evaluation of these functions is best performed as an occasional "add-on" to the test battery limited to instances where these symptoms are emphasized by the patient during the interview.

Similarly, some patients report changes in spatial or perceptual skills following MTBI. Again, there is no reason from a physiological or neuroanatomic standpoint to believe that these types of skills would be affected directly by MTBI. The addition of additional tests of higher order perceptual functions or similar measures would therefore only serve to increase the probability of finding "impairment" by chance as a result of committing a Type I statistical error [9, 66]. While low scores might be obtained on some tests as a result of possible neurodevelopmental factors, this situation might serve as a distraction and cause the patient and the treatment team to believe that these are acquired deficits indicating the presence of chronic brain dysfunction.

Comprehensive evaluations of memory are clearly warranted in patients following MTBI and are usually performed most efficiently with any one of a number of verbal list-learning measures. Tests such as the California Verbal Learning Test (CVLT-2) [17], Rey Auditory Verbal Learning Test (RAVLT) [66], or the Hopkins Verbal Learning Test (HVLT) [11] typically provide the clinician with a means to evaluate various stages of memory processing. Those exhibiting restrictions in their performance during initial learning trials, in combination with low scores on other measures of attention, will be identified as having difficulties with memory encoding. Low scores on delayed recall trials, in combination with higher levels of performance on yes/no recognition, will signal the presence of a retrieval deficit.

Further information regarding memory can be provided through assessment of the patient's ability to recall more contextually based material through the WMS-IV Logical Memory subtest [77], although it is debatable whether that measure adds any significant information to the evaluation of MTBI. While many clinicians prefer to add tests of nonverbal memory to their test battery, one can argue whether these tests add anything new when effects of lateralized hemispheric dysfunction are not an issue. In spite of what might be reported in some rare cases, deficits in remote memory, otherwise known as retrograde amnesia, are not seen in patients following MTBI, indicating no need for the clinician to include any tests focusing on recollection of faces or events from the distant past.

A formal evaluation of validity and response bias is critical in any test battery, particularly in one focusing on a condition such as MTBI, where a combination of many physical, psychological, and motivational factors are likely to be in play. Larrabee [34] has introduced the distinction between performance validity tests (PVT) and symptom validity tests (SVT), with the former designated as performance-based measures of effort used to assess validity of cognitive performance while the latter term is reserved for measures looking at the validity of symptom reporting, as used in self-report questionnaires. Evaluation of patients following MTBI requires the use of both PVTs and SVTs. It is important to note that these measures are not only used for detection of malingering, which is known to be seen at relatively high rates in patients alleging MTBI in forensic contexts, but are useful in helping to identify the influence that somatization, mood disorder, and other psychological disorders are having on the individual's ability to maintain

Physical	Cognitive	Emotional	Sleep
<ul><li>Headache</li><li>Dizziness</li></ul>	<ul> <li>Memory disturbance</li> <li>Concentration disturbance</li> </ul>	<ul><li>Anxiety</li><li>Depression</li></ul>	<ul> <li>Problems with falling asleep</li> <li>Problems with staying asleep</li> </ul>
<ul> <li>Fatigue</li> </ul>	<ul> <li>Forgetfulness</li> </ul>	<ul> <li>Loss of interest</li> </ul>	<ul> <li>Sleeping too much</li> </ul>
Noise sensitivity	Word finding problems	<ul> <li>Restlessness</li> </ul>	
Light sensitivity	Trouble reading		
<ul><li>Numbness</li></ul>	Feening disorganized		

Table 3 Categories of symptoms commonly associated with post-concussion syndrome (PCS)

the level of effort that is necessary to obtain valid results on neuropsychological testing.

The neuropsychological test battery should include at least one freestanding PVT using forcedchoice methodology, such as the Word Memory Test (WMT) [24] or the Test of Memory Malingering (TOMM) [72]. The Medical Symptom Validity Test (MSVT) is another freestanding measure that has been demonstrated to identify invalid levels of performance in MTBI samples in a brief and effective manner [25]. The clinician's use of combinations of other tests, such as those listed in the sections above, will also enable them to look at a number of embedded PVT measures, including the Reliable Digit Span [26] and recognition memory indices from the CVLT-2 or RAVLT. Scores below published cutoffs on at least two of the indices from the freestanding and embedded measures combined provide a signal for the clinician to question the validity of findings from the neuropsychological battery.

Symptom reporting is an important component of any evaluation of a patient following MTBI, particularly since there are no independent means to confirm the presence of injury through standard neuroimaging or electrophysiological methods. A formal evaluation of symptoms through standardized and validated assessment methods is thus an essential component of the neuropsychological evaluation so that the clinician can determine in which cases symptom magnification might be playing a role or whether the reporting of postconcussion symptoms is affected by any comorbid conditions such as chronic pain, somatization, or mood disorder. A combination of brief illness focused measures of symptom reporting and larger scale psychological inventories are recommended for use in test batteries designed for assessment of patients with MTBI.

Several measures for assessment of postconcussion symptom have been developed and validated in recent years for use in sports and non-sports setting [74]. The range of subjective complaints associated with PCS is quite large with different symptoms classified among cognitive, somatic, emotional, and sleep categories. Examples of these symptoms and their associated categories are provided in Table 3.

The Post-Concussion Scale (PCS-R) is a measure developed in the sports setting that provides a rapid assessment of symptoms that is useful for monitoring recovery. It contains 22 items scored on a 7-point (ratings, 0-6) Likert scale [35]. Based on normative information, a score of 20 would represent an elevation of symptoms while a score greater than 30 would indicate an extreme level of reporting. While brief measures of post-concussion symptoms can provide a valuable method for measuring the degree of distress reported by an individual patient, they tell us very little about the specificity of the symptoms, as many similar symptoms are known to occur in association with other clinical conditions such as chronic pain and mood disorder, both of which are frequently comorbid conditions in patients following MTBI. In many cases, the inclusion of additional brief measures of mood, chronic pain, or PTSD symptoms can provide a useful adjunct to symptom assessment, although none of the brief symptom measures provide a means of determining the presence of symptom magnification.

For many of the reasons listed above, a more comprehensive assessment of symptom reporting

needs to be performed using a larger scale self-report instrument such as the Minnesota Multiphasic Personality Inventory (MMPI-2) [14] or the Personality Assessment Inventory (PAI) [53]. The major reason for including these measures is the fact that they contain wellvalidated measures of symptom validity that can help to identify cases where patients might be over-reporting symptoms as a result of somatization or externally based motivational factors. The current author prefers to use the MMPI-2-RF [8] for this purpose as a result of its brevity and the growing literature supporting its use for assessing the validity and range of factors underlying symptom reporting in MTBI samples.

To date, the MMPI-2-RF has been shown to be sensitive to detecting symptom magnification using standard validity indices such as F-r in addition to using other measures such as the Symptom Validity Scale (FBS-r) and Response Bias Scale (RBS), which now have a rather large literature supporting their use with MTBI patients [55, 79]. The MMPI-2-RF also has a number of scales that are useful in identifying patients with features of somatization (RC1-Somatic Complaints) and a high level of cognitive complaints (COG) [80]. Results from this instrument are effective for identifying patients that might be malingering the effects of neurological illness in addition to helping identify those that are likely to be helped by specific forms of psychological intervention.

#### Interventions for MTBI

Results of the neuropsychological evaluation will provide clinicians working with MTBI patients in a clinical setting with critical information on the presence of symptoms, their likely etiology, and the degree to which they are affected by the effects of comorbid physical and psychological conditions. After obtaining this information, the next step is to develop a plan for intervention. This plan will differ significantly depending on the clinical setting and the chronicity of the injury. Those working with patients in the early sub-acute stage of recovery will utilize strategies aimed at preventing the development of persistent PCS while interventions aimed at symptom reduction will be used in patients continuing to experience longer-term effects of the injury.

Psycho-education is the key to prevention of long-term symptoms in patients following MTBI. It was not too long ago that the public at large had very little information about MTBI, making the effects of the injury somewhat of a mystery, both to patients and clinicians. Now, with the explosion of information provided by the media and the internet, most injured patients have expectations of what might be expected following MTBI, with many of these expectations guided not so much by research findings but by misconceptions based on dramatic accounts of celebrities, athletes, and soldiers who have experienced difficult recoveries. The role of the clinician is therefore to relay information from the evidence-based literature while providing reassurance to their patients that, after a brief period of headache and other short-lived symptoms, the vast majority of individuals sustaining an MTBI achieve full recovery, enabling them to resume their lives with no long-term effects.

There are indications that psycho-educational interventions aimed at patients with MTBI are best initiated as early as possible following the injury [52]. Findings from research studies have indicated that providing information sheets or a single session intervention to patients at the time of the ER visit will reduce symptom expression and level of distress at longer-term follow-up. Communications to the patient that he or she will benefit from rest while they undergo a brief period of symptoms can prevent them from suffering setbacks as a result of a premature resumption of their regular activities. Most MTBI experts advocate a common sense approach for an initial return to activity, adapting concepts borrowed from return to play guidelines developed in the sports setting. This generally involves a graduated resumption of activities, as tolerated, until a full return to a pre-injury level of functioning can be achieved unaccompanied by symptoms [47].

The issue of complete cognitive and physical rest has become a controversial topic in the clinical management of MTBI. Recommendations for rest are based loosely on concepts obtained from animal studies where it is known that a premature activation of physiological activity during a period when the brain is undergoing a restorative process can have a negative effect on many of the neural factors important for recovery. Based on this information, it makes sense to recommend a few days rest following an injury. However, the clinician must be careful not to overextend recommendations of rest, which could have the potential of placing the recovering patient at risk for developing a maladaptive focus on their symptoms. At the current time, further information is needed on the long-term benefits of rest and the optimal amount and types of rest that are required for optimal recovery from MTBI [65].

Most health care professionals are well aware that patients with persistent PCS are difficult to treat. Interventions aimed at treating persistent PCS can be divided roughly into physiological and psychological approaches. It is not surprising that there is no consensus on what physiologic approaches to treatment should be used for treatment of PCS, given the fact that its underlying physiologic causes remain undetermined. Based on findings from the research literature, there is more empirical support for the use of psychological interventions for PCS, although there is clearly a need for more work in this area.

Pharmacological approaches to the treatment of patients with PCS focus primarily on treatment of comorbid depression and anxiety, as no medication has been shown to be effective in treating the primary symptoms of PCS [36, 39]. There is a growing interest in identifying oculomotor [21] and vestibular disturbances [5] in PCS patients, based on the possibility that these types of disturbances are often overlooked in this population. However, large-scale studies looking at the efficacy of vision and vestibular therapy are clearly needed. Based on findings that long-term cognitive effects of MTBI are minimal, the use of cognitive rehabilitation approaches with this group is unwarranted. There is no convincing published evidence that neurofeedback, hypnosis, or acupuncture are effective as "alternative" treatments for patients following MTBI [36].

There is now a growing trend to use cognitive behavioral therapy (CBT) for treatment of MTBI patients who have developed PCS. The theoretical basis for CBT interventions with this population is that (1) the symptoms that predominate in PCS are subjective in nature, (2) these symptoms overlap substantially with those seen in other psychological conditions, and (3) there are a number of cognitive-behavioral processes underlying the evolution and maintenance of symptoms in PCS [58]. While previous psychotherapeutic approaches to PCS emphasized a sequence of validation and identifying alternative interpretations for the attribution of symptoms, use of CBT enables the clinician to address maladaptive behavioral responses, cognitive appraisals, and the impact of symptoms on daily life [2].

A 12-session framework for CBT with PCS patients has been developed by Potter and Brown [58]. Beginning sessions utilize and extend the use of materials from psycho-educational interventions by providing information on the course of symptom recovery seen in the majority of patients. This is followed by a number of sessions identifying problem areas and developing appropriate responses. The goal is to help develop positive expectations while limiting the negative impact of perceived errors or mistakes. Later sessions focus on the techniques that worked and did not work in addition to developing longer-term behavioral plans for the future. In a recent systematic review of the research literature, it was determined that all ten randomized controlled trials demonstrated a therapeutic benefit, although small numbers and short durations of follow-up prevent the formation of robust conclusions about the ultimate efficacy of CBT with the MTBI population.

# MTBI Presentation in Different Clinical Settings

The presentation of MTBI differs significantly across settings. For the purposes of this chapter, we will focus on a discussion of similarities and differences as presented in patients with injuries resulting from sports, personal injury, or military

	Sports setting	Personal injury	Military setting
Pre-injury condition	Measured prospectively	Estimated retrospectively	Measured prospectively
Injury documentation	Directly witnessed and recorded	Records or eyewitness reconstruction	Record or eyewitness reconstruction
Acute symptoms	Measured at time of injury	Estimated retrospectively	Dependent on medical care received
Course of early recovery	Measured and recorded	Estimated retrospectively	Dependent on medical care received
Course of chronic symptoms	Per interview and medical records	Per interview and medical records	Per interview and medical records
Possible motivation	Fake good	Fake bad	Fake good or bad, depending on time and setting

**Table 4** Differences in the presentation of mild traumatic brain injury in sports, personal injury, and military settings

service. These three diverse contexts provide markedly different means for documenting details regarding the injured person, the mechanisms of the injury, and its resulting effects. Further details regarding the differences among these three settings are listed in Table 4. For example, those in the sports and military settings are at an increased risk for sustaining an MTBI, which often provides a formal means for obtaining information on their pre-injury status through baseline testing. However, in the case of personal injury evaluations, the clinician is forced to estimate the individual's premorbid state using information from available records and various psychometric methods. The level of available documentation of the injury itself and the course of acute and subsequent recovery varies widely across these three settings. There are also marked differences by which comorbid psychiatric factors and motivation can play a role in symptom presentation, depending on the nature and social context of the injury. Additional details on the differences among these settings are provided below.

#### **Sports Concussion Setting**

When leagues and teams are provided with adequate resources, the sport setting provides the most controlled environment for obtaining details regarding an injury and its subsequent recovery. Injuries sustained by athletes participating in professional and NCAA Division I collegiate settings will often be captured on video recordings with nearly immediate care provided by athletic trainers and physicians located on site. The development of new methodology using helmet and other types of sensors placed on the athlete has also enabled investigators to capture technical information on the impacts to the head that are causing concussive injuries [13]. However, much less information about injuries and the access to immediate care is available to athletes participating at less advanced levels of competition, which often leads to reliance on retrospective accounts of the injury, in a manner that is similar to what is utilized in other settings. Most studies find that the vast majority of athletes (>80 %) exhibit a full resolution of symptoms within 7–10 days of the injury [41, 47].

By virtue of being at a known risk for concusthe practice of obtaining sion. baseline information on the athlete through some form of neuropsychological testing has become commonplace in many athletic settings. The initial rationale for performing baseline testing on athletes, which can be quite costly and time-consuming, was quite reasonable as it provided a means to control for a number of factors (e.g., age, gender, IQ, and effects of learning disability) that are likely to influence scores on neuropsychological tests administered following the injury. However, there is now growing scientific evidence, in the form of poor reliability, disappointing sensitivity/ specificity, and evidence of motivational factors influencing baseline test performance, that information on the athlete obtained at baseline has not proven to be as helpful as had been anticipated for post-concussion management [18].

At this point, there continues to be no empirical clinical or scientific data to indicate that neuropsychological testing, administered through paper/pencil means or computer, adds anything more to the management of acute effects of concussion in comparison to what can be obtained through the use of sideline administered symptoms checklists, cognitive screens, and balance measures, as found in an instrument such as the SCAT-3 [42, 44]. However, that does not mean that neuropsychological assessment plays no role in the evaluation of athletes post-concussion.

A focused approach to neuropsychological assessment using methods described earlier in this chapter can be very helpful to guide interventions aimed at athletes reporting symptoms for more than 14 days, which would take them beyond the window of vulnerability associated with physiological causes [6]. The sports community has a well-established set of guidelines for return to play based on the athlete remaining symptom-free through a predetermined set of graduated stages that eventually culminate in a full return to competition [46]. Neuropsychological testing in a sports setting provides important information from those individuals whose recovery time extends beyond the norm.

While the sports culture calls for athletes to remain "tough" in the face of injury, there is an increase in the appreciation of the level that psychological and motivational factors can influence prolonged symptom presentation in athletes as well as in other types of patients. With those factors in mind, neuropsychological consultation is critical to developing a plan for psychological and rehabilitative interventions for athletes following concussion. Feedback sessions aimed at athletes and their families will need to focus significantly on psychoeducational factors and a review of the evidence-based literature to help counteract much of the information they are likely to have received through the media and clinicians who have not kept up with the scientific literature.

#### **Clinical and Personal Injury Setting**

Nearly every neuropsychologist in clinical practice will, at some point, encounter patients who have sustained an MTBI, with most of these patients seeking services long after the injury. While many are able to provide very detailed narrative accounts of the injury and its aftermath it is imperative for the clinician to obtain information from independent medical records to help determine the nature of the initial injury and its relation to the onset of initial symptoms. In some instances, information obtained from the records will contradict what the patient reports to the provider during the clinical interview. In a routine clinical setting, documentation from the records is critical to evaluating the likelihood that the presenting symptoms are the result of physiological effects of the injury and determining whether other factors, such as pain or psychological effects, are playing a role in the presenting symptoms. In a forensic setting, the information from the records will play even a greater role in helping the neuropsychologist to determine a causal relationship between the injury and continuing symptoms and in determining the risk for longterm disability.

Neuropsychological testing of patients with reported sub-acute or long-term effects of MTBI in most routine clinical settings can be conducted successfully with the type of test battery outlined in Table 2. However, longer test batteries are often needed when the assessment is performed in a forensic capacity or as part of a long-term disability evaluation. In any case, the clinician is advised to focus on assessment of attention, processing speed, and memory, which are the cognitive functions most likely to be affected by MTBI. However, given the effect sizes reported from meta-analyses of MTBI effects [7], it is much more likely the presence of long-term cognitive impairment will be related more to non-injury factors than to the injury in question. Other details from the interview, records, and other sources will help the neuropsychologist determine what factors are likely to be playing a role in the manifestation of cognitive impairment, when it is identified on testing.

Formal assessment of symptom reporting is perhaps the most critical component to the assessment of an individual referred for neuropsychological evaluation of a personal injury secondary to MTBI. Identifying of individuals with symp-
toms resulting from comorbid anxiety and/or depression can be extremely helpful in developing a treatment plan for cases where symptoms resulting from those conditions are misattributed to the persistent effects of MTBI. Cases where symptom reporting is affected by effects of somatization or chronic pain are clearly difficult to treat, although there is growing evidence that symptom reduction is observed successfully in many of these individuals in response to CBT [4]. Given the prominent influence of psychological and iatrogenic factors in many cases of MTBI, patients are often reluctant to obtain feedback that the symptoms they have experienced are the result of emotional factors rather than the effects of an underlying brain injury. Neuropsychologists should, nonetheless, adhere to fully communicating the results of their test findings to at least "plant the seed" that other factors are playing a role in the patient's persistent symptoms.

Due to the multitude of psychological and motivational factors that are likely to influence symptom reporting, no neuropsychological evaluation is complete without inclusion of tests for assessing validity of both the cognitive and selfreport testing. Based on information combined from clinician surveys and data from patient samples, it is estimated that approximately 40 % of individuals undergoing neuropsychological testing in forensic or disability settings will "fail" validity testing while that number drops to less than 10 % in most routine clinical settings [52]. It is important to note that abnormal performance on validity testing in either setting can signal the presence of motivation for an external gain or the effects of other psychological factors. Given the availability of financial gain in the personal injury setting, no forensic evaluation of a patient following alleged MTBI should ever be conducted without a full complement of PVT and SVT.

### **Military Setting**

One can argue that the biggest challenge in neuropsychological assessment of MTBI is faced when clinicians are called upon to evaluate veterans returning from military service.

According to commonly cited statistics, approximately 12-23 % of veterans returning from service in Iraq and Afghanistan sustained an injury classified as MTBI [75]. However, there are many ongoing challenges in documenting the nature and severity of these injuries. The problem is confounded further by estimates that at least 40 % of those soldiers sustaining an MTBI are also diagnosed with PTSD secondary to the extraordinary nature by which the injuries are received [30]. Based on their use of standardized assessment methodology, neuropsychologists are in a unique position to help disentangle the complex nature of symptom reporting in this population to ensure that the veterans obtain the services they need and deserve.

Like athletes, soldiers deployed for combat service are known to be at risk for encountering an MTBI and large-scale baseline neuropsychological testing programs were initiated with the intention of assisting with injury management in this population. However, transmission and distribution of the test data to overseas medical units became a significant challenge, which prevented the use of those data in assessment of injured soldiers. Similarly, while several methods for assessment of cognitive functioning were adapted for use in theater and were, in fact, utilized successfully in many instances by medical personnel, there have been challenges in linking up those records to veterans appearing at medical clinics following their return.

Upon their discharge, military service members complete a questionnaire including many items reflecting their exposure to blasts from IEDs and other events that would expose them to risks for brain injury [70]. The questionnaires also include a number of subjective symptoms associated with possible MTBI exposure. The possible confounding factors inherent to these methods are no different than those existing in other settings. However, the relative difficulties in obtaining records corroborating the battlefield exposure and the medical care received after the injury create a challenge for assessing MTBI in veterans unlike what is seen in any other clinical setting.

Neuropsychologists working in a medical setting associated with the Department of Defense

	Case number 1	Case number 2	Case number 3
Demographics	19-year-old	58-year-old	27-year-old
	Female Soccer Player	Female Counselor	Male Veteran (Iraq)
	13 years education	18 years education	12 years education
WASI-2 FSIQ	SS=108	SS=123	SS=95
Digit span	ss=9	ss=11	ss=7
Digit symbol	ss=7	ss=12	ss=6
DKEFS			
Trailmaking			
Number-letter	ss=11	ss=12	ss=6
Verbal fluency			
Phonemic	ss=8	ss=10	ss=9
CVLT-2			
Total learning	T=48	<i>T</i> =61	T = 54
LDFR	14/16	16/16	12/16
MSVT	DR=98 %	DR=96 %	DR=100 %
RDS	8	9	9
CVLT-2 FC	16/16	16/16	16/16
PCS-R	Raw = 14	Raw=36	Raw=21
BDI-II	Raw=9	Raw = 12	Raw = 11
BAI	Raw=12	Raw = 15	Raw = 16
MMPI-2-RF			
L-Scale	T=54	T=63	T=56
F-Scale	T=50	T=58	T=90
K-Scale	T = 60	<i>T</i> =47	T = 44
• FBS	<i>T</i> =57	T=68	T=61
Profile	RC1, <i>T</i> =87	No elevations	RC2, T=77
			RC4, T=79
			RC7, T=78

 Table 5
 Neuropsychological test score profiles for case examples

(DoD) or Veteran's Administration are well equipped and experienced in evaluating the effects of MTBI in returning veterans. However, there are certain situations where veterans choose to seek evaluations outside of those institutions and end up in the offices of neuropsychologists in private practices or unaffiliated medical centers. In those instances, the clinician will be well served by taking the symptom focused assessment approach described in this chapter.

A comprehensive assessment of possible comorbid psychiatric conditions is critical to the assessment of MTBI in returning veterans. First and foremost, the clinician must evaluate for the presence of PTSD. While this can be performed successfully through the use of a comprehensive interview combined with the MMPI-2-RF, it is often helpful to employ other instruments for screening PTSD [71]. It is also important to remember that PTSD is not the only comorbid condition that might arise in these cases as returning veterans are also known to have high rates of depression and substance abuse following their return [16].

Assessment of malingering has become a controversial topic in the evaluation of medical needs and eligibility for disability in returning veterans. Some feel that it is a dishonor to question motivations behind the clinical presentation of veterans who have served their country bravely. However, the available statistics show a 10–59 % failure rate on PVTs in veteran samples with similar rates of invalid profiles on SVTs, making it clear that the use of these measures is clearly indicated in neuropsychological test batteries administered to this population [33, 54].

#### Case Examples

The following paragraphs provide descriptions of three clinical cases of MTBI as experienced in a sports, personal injury, or military setting. Details are provided in each case on the reported mechanism of injury and the challenges inherent to confirming the patient's initial report through independent documentation. Each of the three cases was evaluated through methods described in this chapter with scores from select tests listed in Table 5. The case examples also highlight how many individual factors regarding the patient, injury setting, and social context affect both symptom presentation and outcome from MTBI.

## **Case 1: Sports Concussion Setting**

This is a case of a 19-year-old Hispanic female (primarily English-speaking) college soccer player who initially sustained a concussion when she collided heads with an opponent during interscholastic competition during her first season at a small northeastern college. There was no initial loss of LOC or subsequent PTA. She experienced headache, dizziness, and nausea at the time of injury, which led the team's athletic trainer to withhold her from the final stages of the game. She continued to experience symptoms for 5 days afterwards, which was followed by a gradual return to full competition within 12 days. Two weeks later, she sustained another blow to the head, as a result of being struck by a small rubber ball that had been thrown by a male student in the context of a group's "horsing around" in the dormitory. She re-experienced the headache and dizziness following this second injury and was not able to return to any level of exercise for 3 months afterwards without experiencing these symptoms. She was advised by her doctors and school officials to withdraw from classes as a result of the continuing effects of the injury. She was referred for neuropsychological testing by her internist 7 months after the injury for assessment of cognitive functioning prior to her return to classes.

The athlete reported that she was continuing to experience a mild level of symptoms at the time of the clinical interview. She continued to be involved with the soccer team, keeping statistics and stated that she wanted to return to play with her teammates in the future. During the interview, it became clear that the athlete had initially taken up soccer on the suggestion of her father, who was an avid supporter of her high school and college teams. At one point, the father began to cry while outwardly contemplating the possibility that he would never see his daughter play another soccer game as a result of the injury. The parents also noted that the entire family was under stress as a result of a failing restaurant business and recent illnesses developed by grandparents.

Test results, as listed in Table 5, demonstrate an average level of performance on tests of intellectual functioning, cognition, and performance validity. The level of symptom reporting was slightly elevated as measured by the PCS-R, consistent with the athlete's report of continuing symptoms. Only minimal elevations were seen on the Beck Anxiety Inventory (BAI) and Beck Depression Inventory (BDI-II). The MMPI-2-RF profile was valid with evidence of an isolated elevation on RC1 (T=87), indicating a marked focus on the experience of somatic symptoms. The athlete and her family were provided with psychoeducation regarding the expected effects of an injury that occurred several months in the past and the likely effects that somatization and other psychological factors were playing a role in the persistent manifestation of symptoms.

The athlete was referred for CBT with a psychologist located in the vicinity of the college. Six months after the evaluation, the referring physician informed the neuropsychologist that the athlete was asymptomatic and had given up soccer to focus on her studies and other extracurricular activities. The physician stated that she was happy and performing well in her classes.

## Case 2: Personal Injury

The second case is a 58-year-old Caucasian, English-speaking woman who works as a guidance counselor at a private school who was struck as a pedestrian by a taxi in a crosswalk while attempting to cross a busy intersection in the New York City. She remembered being thrown into the air but did not recall many other details until her arrival by ambulance at a local hospital. She described difficulties in her communications with emergency room personnel. She was discharged to home on the same day. She reported headaches and dizziness for approximately 1 week following the accident, which followed by continuing struggles with language that were reportedly preventing her from returning to work. Her treating neurologist referred her for a neuropsychological evaluation of the reported language changes 7 weeks following the accident. The contents of medical records sent by the physician confirmed the patient's description of the injury and her initial symptoms. She was described by emergency room staff as exhibiting language difficulties during her visit, which resulting in a GCS score of 13/15.

At the time of the clinical interview, the patient reported that her language difficulties had continued to be a problem for her. She brought a written list of symptoms to the appointment describing in detail instances where she said "funny word things" or developed a tendency to "freeze" before speaking. She described the occurrence of language errors "at least once per day." She also noted problems with reading and spelling. She felt as if her thoughts were "scrambled." She had not yet determined whether she was going to retain the services of a lawyer in relation to the accident. Prior to any testing, the examiner informed the patient that the range of symptoms she was describing, and particularly those involving language, was very atypical for the effects of an MTBI. Following testing, she was shown a table of the most commonly reported MTBI symptoms listed in a scientific research publication and was given a copy of that publication in addition to an informational packet on common effects of MTBI [50].

Test results for this patient are listed in Table 5. Her scores on cognitive tests were primarily above average in relation to appropriate norms. Language tests of repetition, auditory comprehension, naming, and verbal fluency were added to the battery and her performances on these were all well within normal limits. While a high level of symptom reporting was seen on the PCS-R with most of the endorsed items relating to cognitive difficulties, only minimal elevations were seen on the two Beck inventories. There were almost no elevations seen on any scales in the MMPI-2-RF profile.

The patient returned to the office for a feedback session 1 week after the evaluation and informed the examiner that all of her language symptoms had resolved since the last meeting. She was informed of her strong performance on the neuropsychological test battery and was reassured that any residual symptoms would soon clear and that no persistent symptoms would be expected by the time she reached a point of being 2 months following the accident. She reported that she had arranged a return to work on a parttime basis for the following week with a full, anticipated return thereafter. At a time point that was 8 months following the accident, the patient contacted the neuropsychologist through a Linked-In message indicating her continued symptom-free status with thanks for the care she received at the time of the assessment.

## **Case 3: Military Setting**

The story of Case 3 highlights the complex nature of neuropsychological evaluations performed on veterans returning from Iraq and Afghanistan along with the many complications introduced by comorbid psychiatric factors and reconstruction of injuries sustained in the remote past. The veteran is a 27-year-old Hispanic (bilingual, primarily English-speaking) male who served as a Marine for two 18-month tours of duty in Iraq between 2004 and 2008. He reported that he was a participant in direct combat with opposing forces on a number of occasions and had sustained multiple small injuries to the head and one significant impact that was the result of a crash while riding in an armored vehicle during the midst of an enemy attack. He was referred for neuropsychological assessment of impairment that was reported to prevent him from returning to employment as an insurance claims consultant 2 years after his return.

The veteran reported at the time of the neuropsychological evaluation that he was experiencing chronic difficulties with attention and focusing. He had described these symptoms to a physician at a local VA hospital who had suggested that they might represent the effects of head injuries he had sustained in Iraq. When asked more questions about the injuries, the veteran provided a vague description of the frequency with which he had struck his head during training or combat. He indicated that he had also been loosely in the vicinity of IED explosions on multiple occasions. With regard to the armored vehicle accident, he stated that he had experienced at least several minutes of LOC at the time of the accident and was amnestic for his removal from the vehicle and subsequent medical care, which consisted of approximately 24 h of observation. He replied in response to attempted questioning that there were many other experiences in Iraq that he would rather not discuss.

The veteran indicated that he had undergone some form of computer testing prior to deployment, but had no way of obtaining the results. He knew of no way to access records regarding his medical care during deployment. Contents of VA hospital records were consistent with what he was reporting during the interview. Contents of other records indicated that the veteran had been experiencing chronic nightmares and signs of anger and hyper-vigilance since his return from overseas. His wife reported that it had been more difficult to get along with him following his return. He reported increasing difficulties with concentration 1 year into his job at the insurance company. He often complained of anger at the customers. The reported symptoms began to worsen once he started discussing the possible effects of the injuries he had experienced during the two tours of duty.

Neuropsychological test results from this veteran (see Table 5) were indicative of some mild and nonspecific weaknesses on tests of attention. His memory performance was relatively intact. No decrements were seen among his scores on PVT. There were mild elevations on measures of PCS symptoms and mood. The veteran was also administered the PTSD Checklist (PCL-M), which resulted in a score (raw score=77) that was well above most standard clinical cutoffs. The MMPI-

2-RF profile was marked by a high score on the infrequency scale (F-r, T=90) but no elevations were seen among other validity scales. Elevated scores were observed on three of the routine clinical scales. The results of the neuropsychological evaluation were considered indicative of PTSD. Recommendations were made to focus on treatment of the PTSD and to explain to the veteran through ongoing treatment the complex nature between that condition and the effects of MTBI.

## Conclusions

In spite of all the reputed complexities, the neuropsychological evaluation of individuals following MTBI amounts to testing cognitive functioning and symptom reporting, evaluating the validity of those tests, and integrating the results with information obtained through the interview and a review of medical records. While the context of the injury, characteristics of the injured parties, and the access to independent documentation will vary across settings, the neuropsychologist's role and the methodology employed remains relatively standard. The ultimate goal is to provide an evidence-based opinion on the nature and cause of the reported symptoms and to provide recommendations for the most appropriate form of treatment.

Philosophical views on MTBI and the nature and pattern of its recovery vary widely among neuropsychologists, as they do among most other health care professionals. However, it is important to be reminded that the available scientific evidence indicates rather overwhelmingly that the physiological effects of a single injury classified as MTBI are short lived in nature and the vast majority of the individuals sustaining these injuries recover within a matter of a few weeks or less. While there remains an active pursuit of biomarkers for the acute and longer-term effects of MTBI, there are as of yet no data indicating that information obtained from any blood tests or advanced neuroimaging techniques can be used in a valid manner to establish the diagnosis in an individual subject reporting chronic symptoms following an MTBI.

Given our current knowledge on the risk factors, comorbidity, and psychosocial stresses seen in patients reporting long-term effects of MTBI, it is clear that psychological factors play a major role in those patients classified as having PCS. Neuropsychologists, by nature of their focus on the study of brain–behavior relationships and the combined influence of psychological factors, are uniquely qualified to evaluate patients following MTBI and to offer qualified opinions on the etiology of PCS and what about it might be "neuro" versus what is "psychological." A full understanding of the syndrome will arise only from efforts at combining these two factors.

### References

- ACRM. (1993). Definition of mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 8, 86–87.
- Al Sayegh, A., Sandford, D., & Carson, A. J. (2010). Psychological approaches to treatment of postconcussion syndrome: A systematic review. *Journal of Neurology, Neurosurgery, and Psychiatry*, 81(10), 1128–1134. doi:10.1136/jnnp.2008.170092.
- Alexander, M. P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology*, 45, 1253–1260.
- Allen, L. A., Woolfolk, R. L., Escobar, J. I., Gara, M. A., & Hamer, R. M. (2006). Cognitive-behavioral therapy for somatization disorder: A randomized controlled trial. Archives of Internal Medicine, 166, 1512–1518.
- Alsalaheen, B. A., Mucha, A., Morris, L. O., Whitney, S. L., Furman, J. M., Camiolo-Reddy, C. E., et al. (2010). Vestibular rehabilitation for dizziness and balance disorders after concussion. *Journal of Neurologic Physical Therapy*, 34(2), 87–93. doi:10.1097/ NPT.0b013e3181dde568.
- Barr, W. B., & McCrea, M. (2010). Diagnosis and assessment of concussion. In F. M. Webbe (Ed.), *The* handbook of sport neuropsychology. New York: Springer.
- 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, 215–227.
- Ben-Porath, Y. S., & Tellegan, A. (2008). MMPI-2-RF: Manual for administration scoring and interpretation. Minneapolis, MN: University of Minnesota Press.
- Binder, L. M., Iverson, G. L., & Brooks, B. L. (2009). To err is human: "abnormal" neuropsychological scores and variability are common in healthy adults.

Archives of Clinical Neuropsychology, 24(1), 31–46. doi:10.1093/arclin/acn001.

- Borkowski, J. G., Benton, A. L., & Spreen, O. (1967). Word fluency and brain damage. *Neuropsychologia*, 5, 135–140.
- 11. Brandt, J. (1991). The Hopkins Verbal Learning Test: Development of a new verbal learning test with six equivalent forms. *The Clinical Neuropsychologist*, *5*, 125–142.
- Broglio, S. P., Ferrrara, M. S., Macciocchi, S. N., Baumgartner, T. A., & Elliot, R. (2007). Test-retest reliability of computerized concussion assessment programs. *Journal of Athletic Training*, 42, 509–514.
- Broglio, S. P., Surma, T., & Ashton-Miller, J. A. (2012). High school and collegiate football athlete concussions: A biomechanical review. *Annals of Biomedical Engineering*, 40(1), 37–46. doi:10.1007/ s10439-011-0396-0.
- Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A. M., & Kaemmer, B. (1990). MMPI-2: Minnesota Multiphasic Personality Inventory—2. Manual for administration and scoring. Minneapolis, MN: University of Minnesota Press.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, L., Holm, L., et al. (2004). Prognosis for mild traumatic brain injury: Results of the who collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine*, 36, 84–105. doi:10.1080/16501960410023859.
- Chapman, J. C., Andersen, A. M., Roselli, L. A., Meyers, N. M., & Pincus, J. H. (2010). Screening for mild traumatic brain injury in the presence of psychiatric comorbidities. *Archives of Physical Medicine* and Rehabilitation, 91(7), 1082–1086. doi:10.1016/j. apmr.2010.03.018.
- Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *California Verbal Learning Test* (2nd ed.). San Antonio, TX: The Psychological Corporation.
- Echemendia, R. J., Iverson, G. L., McCrea, M., Macciocchi, S. N., Gioia, G. A., Putukian, M., et al. (2013). Advances in neuropsychological assessment of sport-related concussion. *British Journal of Sports Medicine*, 47(5), 294–298. doi:10.1136/ bjsports-2013-092186.
- Ellemberg, D., Henry, L. C., Macciocchi, S. N., Guskiewicz, K. M., & Broglio, S. P. (2009). Advances in sport concussion assessment: From behavioral to brain imaging measures. *Journal of Neurotrauma*, 26(12), 2365–2382. doi:10.1089/neu.2009.0906.
- 20. 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.
- Galetta, K. M., Brandes, L. E., Maki, K., Dziemianowicz, M. S., Laudano, E., Allen, M., et al. (2011). The King-Devick test and sports-related concussion: Study of a rapid visual screening tool in a

collegiate cohort. *Journal of Neurological Sciences*, 309(1–2), 34–39. doi:10.1016/j.jns.2011.07.039.

- Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training*, 36(3), 228–235.
- 23. Golden, C. (1978). *Stroop Color and Word Test*. Wood Dale, IL: Stoelting Company.
- Green, P. (2003). Green's Word Memory Test for Windows: User's manual. Edmonton, AL: Green's Publishing.
- Green, P. (2004). Green's Medical Symptom Validity Test (MSVT) for Windows: User's manual. Edmonton, AL: Green's Publishing.
- Greiffenstein, M. F., Baker, W. J., & Gola, T. (1994). Validation of malingered amnesia measures with a large clinical sample. *Psychological Assessment*, 6, 218–224.
- Gunstad, J., & Suhr, J. A. (2001). "Expectation as etiology" versus "the good old days": Postconcussion syndrome symptom reporting in athletes, headache sufferers, and depressed individuals. *Journal of the International Neuropsychological Society*, 7, 323–333.
- Guskiewicz, K. M., McCrea, M., Marshall, S. W., Cantu, R. C., Randolph, C., Barr, W., et al. (2003). Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA Concussion Study. *JAMA*, 290(19), 2549–2555. doi:10.1001/jama.290.19.2549.
- Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). Wisconsin Card Sorting Test manual: Revised and expanded. Odessa, FL: Psychological Assessment Resources, Inc.
- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *The New England Journal of Medicine*, 358, 453–463.
- Kelly, J. P., & Rosenberg, J. H. (1997). Diagnosis and management of concussion in sports. *Neurology*, 48(3), 575–580.
- Laker, S. R. (2011). Concussion and mild traumatic brain injury: Current and future concepts. Introduction. *PM & R*, 3(10 Suppl 2), S351–S353. doi:10.1016/j. pmrj.2011.08.004.
- 33. Lange, R. T., Pancholi, S., Bhagwat, A., Anderson-Barnes, V., & French, L. M. (2012). Influence of poor effort on neuropsychological test performance in U.S. military personnel following mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, *34*(5), 453–466. doi: 10.1080/13803395.2011.648175.
- Larrabee, G. J. (2012). Performance validity and symptom validity in neuropsychological assessment. *Journal of the International Neuropsychological Society*, 18, 1–7.
- Lovell, M. R., Iverson, G. L., Collins, M. W., Podell, K., Johnston, K. M., Pardini, D., et al. (2006). Measurement of symptoms following sports-related concussion: Reliability and normative data for the

post-concussion scale. *Applied Neuropsychology*, *13*(3), 166–174. doi:10.1207/s15324826an1303\_4.

- Makdissi, M., Cantu, R. C., Johnston, K. M., McCrory, P., & Meeuwisse, W. H. (2013). The difficult concussion patient: What is the best approach to investigation and management of persistent (>10 days) postconcussive symptoms? *British Journal of Sports Medicine*, 47(5), 308–313. doi:10.1136/ bjsports-2013-092255.
- Matarazzo, J. D. (1990). Psychological assessment versus psychological testing. Validation from Binet to the school, clinic, and courtroom. *The American Psychologist*, 45(9), 999–1017.
- Mayer, A. R., Ling, J., Mannell, M. V., Gasparovic, C., Phillips, J. P., Doezema, D., et al. (2010). A prospective diffusion tensor imaging study in mild traumatic brain injury. *Neurology*, *74*(8), 643–650. doi:10.1212/WNL.0b013e3181d0ccdd.
- McAllister, T. W. (2009). Psychopharmacological issues in the treatment of TBI and PTSD. *Clinical Neuropsychology*, 23(8), 1338–1367. doi:10.1080/13854040903277289.
- McCrea, M. (2008). Mild traumatic brain injury and postconcussion syndrome. New York: Oxford University Press.
- McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., et al. (2003). Acute effects and recovery time following concussion in collegiate football players: The NCAA Concussion Study. JAMA, 290(19), 2556–2563. doi:10.1001/ jama.290.19.2556.
- 42. McCrea, M., Iverson, G. L., Echemendia, R. J., Makdissi, M., & Raftery, M. (2013). Day of injury assessment of sport-related concussion. *British Journal of Sports Medicine*, 47(5), 272–284. doi:10.1136/bjsports-2013-092145.
- McCrea, M., Iverson, G. L., McAllister, T. W., Hammeke, T. A., Powell, M. R., Barr, W. B., et al. (2009). An integrated review of recovery after mild traumatic brain injury (MTBI): Implications for clinical management. *Clinical Neuropsychology*, 23(8), 1368–1390. doi:10.1080/13854040903074652.
- 44. McCrory, P., Meeuwisse, W., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R., et al. (2013). Consensus statement on concussion in sport—The 4th international conference on concussion in sport held in Zurich, November 2012. *Physical Therapy in Sport*, 14(2), e1–e13. doi:10.1016/j. ptsp.2013.03.002.
- 45. McCrory, P., Meeuwisse, W., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R. J., et al. (2013). Consensus statement on concussion in sport–the 4th international conference on concussion in sport held in Zurich, November 2012. *Clinical Journal of Sport Medicine*, 23(2), 89–117. doi:10.1097/ JSM.0b013e31828b67cf.
- 46. McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R. J., et al. (2013). Consensus statement on concussion in sport: The 4th interna-

tional conference on concussion in sport held in Zurich, November 2012. *Journal of the American College of Surgery*, 216(5), e55–e71. doi:10.1016/j. jamcollsurg.2013.02.020.

- 47. McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, R. C., Dvorak, J., Echemendia, R. J., et al. (2013). Consensus statement on concussion in sport–the 4th international conference on concussion in sport held in Zurich, November 2012. *PM & R*, 5(4), 255–279. doi:10.1016/j.pmrj.2013.02.012.
- McCrory, P., Meeuwisse, W. H., Kutcher, J. S., Jordan, B. D., & Gardner, A. (2013). What is the evidence for chronic concussion-related changes in retired athletes: Behavioural, pathological and clinical outcomes? *British Journal of Sports Medicine*, 47(5), 327–330. doi:10.1136/bjsports-2013-092248.
- McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson, A. E., et al. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology and Experimental Neurology*, 68(7), 709–735. doi:10.1097/NEN. 0b013e3181a9d503.
- Mittenberg, W., Canyock, E. M., Condit, D., & Patton, C. (2001). Treatment of post-concussion syndrome following mild head injury. *Journal of Clinical and Experimental Neuropsychology*, 23, 829–836.
- Mittenberg, W., DiGuillo, D. V., Perrin, S., & Bass, A. E. (1992). Symptoms following mild head injury: Expectation as etiology. *Journal of Neurology, Neurosurgery & Psychiatry*, 55, 200–204.
- Mittenberg, W., Patton, C., Canyock, E. M., & Condit, D. C. (2002). Base rates of malingering and symptom exaggeration. *Journal of Clinical and Experimental Neuropsychology*, 24(8), 1094–1102. doi:10.1076/ jcen.24.8.1094.8379.
- Morey, L. C. (1991). Personality Assessment Inventory professional manual. Odessa, FL: Psychological Assessment Resources.
- Nelson, N. W., Hoelzle, J. B., McGuire, K. A., Sim, A. H., Goldman, D. J., Ferrier-Auerbach, A. G., et al. (2011). Self-report of psychological function among OEF/OIF personnel who also report combat-related concussion. *Clinical Neuropsychology*, 25(5), 716– 740. doi:10.1080/13854046.2011.579174.
- 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 Neuropsychology*, 24(4), 701–724. doi:10.1080/13854040903482863.
- Pearson. (2009). Advanced clinical solutions for the WAIS-IV and WMS-IV. San Antonio, TX: Pearson.
- Pertab, J. L., James, K. M., & Bigler, E. D. (2009). Limitations of mild traumatic brain injury metaanalyses. *Brain Injury*, 23(6), 498–508. doi:10.1080/02699050902927984.
- Potter, S., & Brown, R. G. (2012). Cognitive behavioural therapy and persistent post-concussional symptoms: Integrating conceptual issues and practi-

cal aspects in treatment. *Neuropsychological Rehabilitation*, 22(1), 1–25. doi:10.1080/09602011.2 011.630883.

- Randolph, C., & Kirkwood, M. W. (2009). What are the real risks of sport-related concussion, and are they modifiable? *Journal of International Neuropsychological Society*, *15*(4), 512–520. doi:10.1017/S135561770909064X.
- Randolph, C., McCrea, M., & Barr, W. B. (2005). Is neuropsychological testing useful in the management of sport-related concussion? *Journal of Athletic Training*, 40(3), 139–152.
- Reitan, R. M. (1979). Manual for administration of neuropsychological test batteries for adults and children. Tucson: Reitan Neuropsychology Laboratories.
- Resch, J., Driscoll, A., McCaffrey, N., Brown, C., Ferrara, M. S., Macciocchi, S. N., et al. (2013). ImPACT test-retest reliability: Reliably unreliable? *Journal of Athletic Training*, 48, 501–511.
- 63. Rohling, M. L., Larrabee, G. J., Greiffenstein, M. F., Ben-Porath, Y. S., Lees-Haley, P., Green, P., et al. (2011). A misleading review of response bias: Comment on McGrath, Mitchell, Kim, and Hough (2010). *Psychological Bulletin*, *137*(4), 708–712; authors reply 713–705. doi: 10.1037/a0023327
- 64. Rohling, M. L., Larrabee, G. J., & Millis, S. R. (2012). The "miserable minority" following mild traumatic brain injury: Who are they and do meta-analyses hide them? *Clinical Neuropsychology*, 26(2), 197–213. doi :10.1080/13854046.2011.647085.
- 65. Schneider, K. J., Iverson, G. L., Emery, C. A., McCrory, P., Herring, S. A., & Meeuwisse, W. H. (2013). The effects of rest and treatment following sport-related concussion: A systematic review of the literature. *British Journal of Sports Medicine*, 47(5), 304–307. doi:10.1136/bjsports-2013-092190.
- 66. Schretlen, D. J., Testa, S. M., Winicki, J. M., Pearlson, G. D., & Gordon, B. (2008). Frequency and bases of abnormal performance by healthy adults on neuropsychological testing. *Journal of the International Neuropsychological Society*, 14, 436–445.
- Shively, S., Scher, A. I., Perl, D. P., & Diaz-Arrastia, R. (2012). Dementia resulting from traumatic brain injury: What is the pathology? *Archives of Neurology*, 69(10), 1245–1251. doi:10.1001/archneurol.2011. 3747.
- Smith, A. (1975). Symbol digit modalities test manual. Los Angeles, CA: Western Psychological Services.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, 1974(2), 77–80.
- Terrio, H. P., Nelson, L. A., Betthauser, L. M., Harwood, J. E., & Brenner, L. A. (2011). Postdeployment traumatic brain injury screening questions: Sensitivity, specificity, and predictive values in returning soldiers. *Rehabilitation Psychology*, 56(1), 26–31. doi:10.1037/a0022685.
- Tiet, Q. Q., Schulte, K. K., & Leyva, Y. E. (2013). Diagnostic accuracy of brief PTSD screening instru-

ments in military veterans. Journal of Substance Abuse Treatment, 45, 134–142.

- 72. Tombaugh, T. N. (1996). *TOMM: Test of memory malingering*. North Tonawanda, NY: Multi-Health Systems.
- Vagnozzi, R., Signoretti, S., Cristofori, L., Alessandrini, F., Floris, R., Isgro, E., et al. (2010). Assessment of metabolic brain damage and recovery following mild traumatic brain injury: A multicentre, proton magnetic resonance spectroscopic study in concussed patients. *Brain*, 133(11), 3232–3242. doi:10.1093/brain/awq200.
- Valovich McLeod, T. C., & Leach, C. (2012). Psychometric properties of self-report concussion scales and checklists. *Journal of Athletic Training*, 47, 221–223.
- Warden, D. (2006). Military TBI during the Iraq and Afghanistan wars. *The Journal of Head Trauma Rehabilitation*, 21, 398–402.

- Wechsler, D. (2001). Wechsler test of adult reading manual. Bloomington, MN: Pearson.
- 77. Wechsler, D. (2008). *Wechsler Memory Scale* (4th ed.). San Antonio, TX: Pearson.
- Wechsler, D. (2011). Wechsler Abbreviated Scale of Intelligence-second edition manual. Bloomington, MN: Pearson.
- 79. Wygant, D. B., Sellbom, M., Gervais, R. O., Ben-Porath, Y. S., Stafford, K. P., Freeman, D. B., et al. (2010). Further validation of the MMPI-2 and MMPI-2-RF Response Bias Scale: Findings from disability and criminal forensic settings. *Psychological Assessment*, 22(4), 745–756. doi:10.1037/a0020042.
- Youngjohn, J. R., Wershba, R., Stevenson, M., Sturgeon, J., & Thomas, M. L. (2011). Independent validation of the MMPI-2-RF Somatic/Cognitive and Validity scales in TBI Litigants tested for effort. *Clinical Neuropsychology*, 25(3), 463–476. doi:10.10 80/13854046.2010.550635.

# Malingering in Mild Traumatic Brain Injury

# Maria Easter Cottingham and Kyle Brauer Boone

# Abstract

Mild traumatic brain injury (mTBI) and its congnitive sequelae (or lack thereof) are a source of controversy within the field of neurcpsychology. A primary reason for this controversy is that research examining neurocongitive function in mTBI historically has failed to include performance validity tests (PVTs), which resulted in individuals not performing to true capability being retained in study samples. This chapter summarizes newer research on cognitive outcome in mTBI, and illustrates use of miltiple PVTs and personality test data in the identification of noncredible test takers.

#### Keywords

mTBI • Malingering • Response bias • Performance validity • Concussion • Cognition

# **Overview of This Chapter**

Mild traumatic brain injury (mTBI) and its cognitive sequelae (or lack thereof) are a source of great controversy within the field of neuropsychology [1–3]. It is our belief that one of the primary reasons for this controversy is that research examining cognitive function in mTBI has often

M.E. Cottingham, Ph.D. (🖂)

Private Practice, Los Angeles, CA, USA e-mail: mecottingham@gmail.com

K.B. Boone, Ph.D. California School of Forensic Studies, Alliant International University, Los Angeles, CA, USA failed to include performance validity tests (PVTs) [4], which has resulted in individuals not performing to true capability being retained in the samples. Conclusions regarding the presence of neurocognitive deficits can only be definitive if performance validity has been verified.

This chapter will discuss how to best examine for negative response bias in neuropsychological evaluations in mTBI. We will begin by discussing the importance of performance validity testing in mTBI by presenting some prevalence data on malingering in the context of mTBI, and by highlighting some of the problems within the current literature that have led to erroneous conclusions regarding the presence of long-term cognitive deficits in this population. We will also review welldesigned research that has included PVTs, and we will make the case that these studies are more accurate in their conclusions regarding cognitive sequelae of mTBI. We will then discuss different methods that clinicians can use to detect noncredible symptoms, and we will include a section on the relevant terminology and statistical information needed to interpret failures on PVTs. Also tackled will be the interpretation of multiple PVTs used in tandem, as well as the types of response bias indicators that may be especially sensitive in individuals claiming mTBI. In addition to cognitive indices, we will briefly examine the use of personality inventories, behavioral observations, atypical data patterns, and other methods to aid in the detection of noncredible performance/malingering. Cases from our own work will be referenced to provide real-life examples of noncredible performance in mTBI. We will conclude with an overview of our comprehensive approach to the detection of malingering in mTBI.

# Prevalence of Cognitive Symptom Feigning in mTBI

Rates of malingering/negative response bias in claimed mTBI vary somewhat, but most estimates in the context of litigation hover at approximately 40 % [5, 6]. Although compensationseeking status is often used as the criterion for determining whether or not to include PVTs in one's neuropsychological battery, there is evidence that non-compensation-seeking individuals with mTBI may also fail PVTs. We have seen active duty service members present for evaluation in a clinical context and produce PVT failures, when the only apparent secondary gain was avoidance of further combat deployment. In fact, PVT failure rates of 58 % have been reported in U.S. veterans claiming mTBI and with no ostensible secondary gain [7]. A recent study examining military veterans with a self-reported history of mTBI and no motive to feign (participants were recruited specifically and solely for the research project) found that PVT scores better explained performance on cognitive tests than did demographic characteristics and so-called "postconcussive" symptoms [8]. Therefore, it is important to utilize PVTs routinely, even when there is no apparent secondary gain, particularly when evaluating mTBI patients [9, 10].

# Research Literature on Long-Term Neurocognitive Functioning After mTBI

A longstanding "myth" in neuropsychology is that 10-15 % of mTBI patients exhibit long-term, frequently disabling, neurocognitive abnormalities [11]. This assertion appears in litigation testimony, but when the expert is pressed, rarely can a research citation be provided. When a research publication is proffered, it is usually Alexander's 1995 article [12], a review of mTBI, which cites two references in support of the conclusion that 10-15 % of individuals who have sustained a mTBI do not recover. However, examination of the primary sources shows that they do not in fact support the statement. Specifically, in the Rutherford, Merrett, and McDonald study [13], presence of cognitive dysfunction was based on self-report, not objective testing, and of the 15 % who claimed continuing symptoms, almost half were involved in a lawsuit and/or judged to be malingering. Obviously, documentation of any actual long-term problems in mTBI cannot be based on a sample which has motive to feign symptoms and is in fact suspected of malingering. In the second study [14], the sample included some moderately severe head injury patients, and while a subset of subjects continued to report symptoms, no deficits were detected on objective testing at 1 month post injury. Determination of persistent cognitive abnormalities after mTBI cannot be based on selfreport, given the large literature showing little correspondence between self-report of cognitive dysfunction and objective test data [15–17], and certainly conclusions regarding chronic cognitive abnormalities after mTBI cannot be based on a sample with moderate TBI.

Other studies in addition to the Rutherford et al. [13] and McLean et al. [14] investigations have concluded, based solely on patient selfreport, that long-term cognitive symptoms are present after mTBI [18]. However, as noted above, a large literature shows very little correspondence between self-report and cognitive abnormalities identified on objective testing. Further, data show that the way in which one solicits information regarding postconcussive symptoms affects symptom reporting. For instance, Villemure and colleagues found that patients who were allowed to freely report symptoms reported fewer symptoms than did those who were queried regarding presence of specific symptoms [19, 20].

Some publications have utilized objective test data to claim that long-term effects of mTBI include memory loss [21], communication difficulties [22] including decreased verbal fluency [23, 24] and deficits in narrative discourse production [25], decreases in complex attention [26] and working memory [24], mental fatigue and processing speed abnormalities [21, 27], and reduced occupational attainments (based on military duty status) [28]. However, the above studies have generally either not administered PVTs [21-23, 25-28], and/or have failed to consider and report data on presence of secondary gain [22–27]. Further, those that have utilized PVTs have typically only administered a single measure of response bias, often with limited sensitivity (e.g., Test of Memory Malingering [TOMM]) [24], resulting in retention of an unknown percentage of noncredible individuals in the sample. Additionally, some studies that administered PVTs failed to ascertain or report whether compensation-seeking was present [29], which, given that no PVT has perfect sensitivity (i.e., 100 % detection of noncredible individuals), raises the probability that noncredible subjects were not completely excluded from the samples. Finally, studies have been contaminated by selection bias (i.e., only those individuals continuing to report symptoms present for evaluation) [30], "diagnosis threat" (i.e., studies that specifically recruit for subjects who "have" mTBI observe lower cognitive performances than those studies using "neutral" recruitment language) [31, 32], and failure to consider comorbid conditions (e.g., depression) [33].

We in fact could not identify any study purporting to describe long-term deficits in mTBI that did not suffer from at least some of the above methodological limitations. In contrast, there are now at least five meta-analyses and one review showing that there is no long-term cognitive compromise in mTBI [4, 34–39]. Additionally, an authoritative book examining the extensive literature on mTBI concluded that there are no long-term cognitive deficits associated with this condition [40].

Evidence is emerging that persistent cognitive complaints and deficits following mTBI are related to other factors, such as compensationseeking status. One of the widely cited metaanalyses, involving 1,463 mTBI cases and 1,191 healthy controls, reported that cognitive functioning in individuals with mTBI improves to baseline within 3 months, with the exception of those in litigation, who either remain stable or worsen over time [4]. Similarly, Carroll et al. [36], based on data from 120 peer-reviewed studies, concluded that mTBI resolves fully relatively quickly and only individuals seeking compensation and/or in litigation exhibit longterm cognitive symptoms. Bianchini and colleagues [41] found that compensation-seeking individuals with mTBI were more likely to fail PVTs and to produce elevated FBS scores on the MMPI-2 than were mTBI patients not seeking compensation; even compensation-seeking individuals who had sustained a moderate to severe TBI were less likely to fail SVTs than their mTBI counterparts.

Rohling and colleagues [38] suggest that additional noninjury factors (e.g., pain, poor effort, premorbid psychiatric dysfunction) must be considered when an individual continues to report and/or exhibit cognitive symptoms beyond the initial 3-month post-injury period. For example, Kashluba and colleagues [42] reported that compensation-seeking status and pre-existing psychiatric problems were the only predictors of long-term cognitive complaints after mTBI. Similarly, two additional studies observed that "postconcussive" symptoms and/or failure on PVTs and associated poor performance on cognitive tests were related to emotional distress or pre-existing psychiatric problems [43, 44]. Hoge et al. [45], surveying a large sample of recently

returned veterans, found that while soldiers with reported histories of mTBI were more likely to report somatic and postconcussive symptoms than their counterparts with no reported mTBI history, after controlling for psychiatric symptoms (posttraumatic stress disorder [PTSD] and depression), mTBI was no longer associated with these symptom reports, with the exception of headache. Rapaport et al. [46] and Chamelian and Feinstein [47] observed that in patients with mild and moderate TBI, those with major depression had more complaints of cognitive dysfunction and poorer scores on tests of processing speed, verbal memory, and working memory/executive function, but the latter disappeared when the impact of depression was controlled, leading Chamelian and colleagues to conclude that "in mild to moderate TBI, subjective cognitive deficits are linked in large measure to comorbid major depression" ([47], p. 33). As further evidence of the role of psychiatric issues in the maintenance of "mTBI" symptoms, Fann et al. [48] reported improvement in cognitive function post-mTBI with treatment of depression. A recent study of Vietnam veterans found that comorbid psychiatric disorders, including Major Depression, PTSD, and Generalized Anxiety Disorder, led to higher rates of "postconcussive" symptom endorsement than did a history of mTBI [49]. Similarly, Verfaellie et al. [50] documented that PTSD and depression symptoms better explained self-reported "postconcussive" symptoms in a sample of recent military veterans than did injury characteristics. History of sexual abuse has also been found to be associated with mTBIrelated cognitive abnormalities [51]; although *n*'s were small, subjects with histories of mTBI only underperformed in working memory, but presence of both mTBI and history of sexual abuse was associated with additional losses in executive function and memory. Finally, there is some evidence that "diagnosis threat" may impact an individual's display of "postconcussive" symptoms; a recent study of undergraduates with histories of mTBI found that male participants tended to exhibit poorer performances upon cognitive testing when placed in the "diagnosis threat" condi-

tion (i.e., specifically informed that individuals with histories of mTBI underperform on cognitive measures) as compared to individuals who did not receive this information [52].

The effect of noninjury factors may even be present in the acute stages of recovery following mTBI (i.e., <3 months post-injury). In a 2010 by Lange and colleagues study [53], compensation-seeking individuals an average of 2 months post-injury who failed the TOMM produced higher ratings on a postconcussive symptom report scale than those who passed the TOMM; those who passed the PVT performed within normal limits on cognitive measures, whereas those failing the TOMM performed poorly on measures of attention, memory, and executive functioning. Cooper et al. [54] observed that in military personnel who sustained mTBI an average of 2 months prior to evaluation, equivocal weakness in attention and processing speed (scores remained within normal limits) were moderated in large part by usage of narcotic medications. A separate study examining individuals an average of 4.5 days post-mTBI documented expected acute cognitive weaknesses but also found that postconcussive symptom report was related to emotional distress [55].

Overall, postconcussive symptoms have been shown to be too nonspecific to be reliably linked to mTBI, given that these symptoms are often reported in individuals with no history of mTBI; in a 2010 study [56], 74 % of healthy individuals reported seven or more postconcussive-like symptoms, and individuals with Beck Depression Inventory-II (BDI-II) [57] scores above 14 exhibited scores on the British-Columbia Postconcussion Symptom Inventory [58] at more than double the mean of those with lower BDI-II scores.

# Detection of Noncredible Cognitive Symptoms in mTBI

*Statistical Terminology.* In order to fully interpret PVT performance, the clinician must have an understanding of sensitivity and specificity. Sensitivity refers to the ability of a measure to

correctly classify individuals with a particular condition. In the case of PVTs, it reflects the percentage of noncredible individuals detected as noncredible (true positives). Specificity, on the other hand, refers to the measure's ability to correctly identify patients who do not have the condition. In the case of PVTs, specificity indicates the percentage of credible individuals who are correctly classified as credible. Sensitivity and specificity are interrelated, such that as cut-offs are set to increase sensitivity, specificity decreases, and vice versa. PVT cut-offs are traditionally selected to maximize specificity; the general standard is to set specificity at  $\geq 90$  % so that at most only 10 % of credible patients are misclassified, which reflects the position held in the field that it is more important to protect credible individuals from being incorrectly labeled as noncredible than to allow noncredible individuals to remain undetected [59]. The clinician should strive to utilize PVTs that have high sensitivity while maintaining adequate specificity. Several indicators of negative response bias meet or exceed  $\geq$ 70 % sensitivity while allowing  $\leq$ 10 % false positive identifications, such as the Warrington Recognition Memory Test-Words [60], Rey-Osterrieth Effort Equation [61], Digit Symbol Recognition [62], Rey Auditory Verbal Learning Test (RAVLT) Effort Equation [63], Rey-Osterrieth (RO)/RAVLT discriminant function [64], and Dot Counting [65].

The clinician might ask, why not choose PVTs that can detect all noncredible subjects? Noncredible subjects are not homogenous in feigning strategies [66], and as a result, no single PVT will capture all noncredible presentations. Because PVTs do not have 100 % sensitivity, a negative finding on a measure does not necessarily assure that a patient is in fact performing to true capability. For example, if sensitivity at a given PVT cut-off is 60 %, then 40 % of noncredible subjects will not be detected. As discussed below, the error in classification present in any single PVT can often be circumvented by use of multiple PVTs sampling various cognitive domains (and therefore differing feigning strategies).

*Classification of PVTs.* PVTs can be classified in a number of ways—embedded vs. free-standing, forced choice vs. nonforced choice, and according to the type of apparent cognitive skills required. We will examine each of these classifications in turn.

### **Embedded Versus Free-Standing**

Practice guidelines for the assessment of noncredible cognitive performance indicate that both free-standing and embedded PVTs should be employed (American Academy of Clinical Neuropsychology [10]). Dedicated PVTs are those that have the single purpose of assessing response bias (e.g., TOMM [67]). In contrast, embedded PVTs are those that have been derived from standard cognitive measures that have primary purposes in the assessment of specific cognitive skills (e.g., California Verbal Learning Test-II (CVLT-II) Forced Choice [68]; Reliable Digit Span (RDS) on WAIS-III Digit Span [69]).

There are pros and cons to consider for both types. Dedicated measures may have more extensive published validation data than do embedded measures. On the other hand, embedded indicators have several advantages. First, they are not as susceptible to coaching/education as are free-standing measures. Specifically, internet searches on names of free-standing PVTs will reveal their use as measures of response bias, but searches on standard cognitive tests will show their primary use as measures of neurocognitive function. Individuals attempting to portray themselves as more impaired than is actually the case on an embedded PVT will have to navigate around the PVT component to demonstrate credibility but at the same time underperform on the standard scores to display dysfunction, a complicated endeavor. Embedded indicators also do not add time to test battery administration; on the other hand, some free-standing measures (e.g., Victoria Symptom Validity Test (VSVT) [70]) require 15 min or more to administer. Finally, embedded measures allow the clinician to obtain data on response bias in "real-time"; in other words, if each cognitive test contains an embedded indicator, information is available regarding response bias for each task, rather than having to rely on PVT data administered at a different point in the exam. This is especially important because response bias may fluctuate across an evaluation [66].

# Forced Choice Versus Nonforced Choice

In forced choice tasks, a series of stimuli (typically visual; either verbal or nonverbal) are presented sequentially, and at the conclusion, test takers are administered trials in which a previously presented stimulus is paired with a novel stimulus, with subjects requested to identify, for each pair, the item to which they were previously exposed.

The forced choice paradigm assesses for recognition memory, and in fact is very easy although appears difficult to the test taker because of the volume of material presented (often 50 stimuli; e.g., TOMM [67]; Warrington Recognition Memory Test-Words [<mark>60</mark>]). Because an individual who has never seen the test will perform at chance levels (i.e., one has a 50/50 chance of guessing the correct answer for each pair), an individual performing significantly below chance (e.g.,  $\leq 19/50$ ) can be fairly confidently classified as not performing to true ability. This type of performance indicates that the individual knew which answer was correct and knowingly chose the wrong answer (which would demonstrate intact memory!). However, relatively few noncredible individuals actually perform this poorly ( $\leq 15\%$ ) [60, 71–73]. Fortunately, research has shown that cut-offs can be set much higher while still maintaining  $\geq 90$  % specificity, and thereby increasing sensitivity to  $\geq 70 \%$ (TOMM [74]; Warrington Recognition Memory Test—Words [60]).

Forced choice measures are the most widely used PVTs [65–77]. However, this widespread use may be a liability in that the overexposure makes them more vulnerable to coaching; for example, an attorney could simply inform a client, "Whenever you see a test in which you have to pick between two choices, perform well on that test."

Nonforced choice measures refer to tests that do not employ this response paradigm, and can include selection of correct items from a multiple choice format (Digit Symbol recognition [62]), circling items in an array (e.g., b Test [78]; Rey Word Recognition Test [79, 80]; RAVLT Effort Equation [63]; Rey 15-item plus recognition [81]; Rey-Osterrieth (RO) Effort Equation [82]), reproducing visual stimuli with paper and pencil (RO Effort Equation [82]; Rey-15 item [81]; RO/ RAVLT discriminant function [64]), verbal repetition (Digit Span indicators [69]; Sentence Repetition [83]), providing verbal answers from an infinite number of possibilities (Dot Counting [65]; Picture Completion [84]), and rapid motor movements (Finger Tapping [85]). Nonforced choice measures overall may be less sensitive than are forced choice measures, with sensitivity rates ranging from the 30s to the 70s (when cutoff specificity is set to 90 %) [74, 86, 87]. In contrast, cut-offs can be selected for most forced choice measures that will maintain at least 90 % specificity and detect at least 70 % of noncredible test takers [60, 74].

### **PVTs by Cognitive Domain**

Lastly, PVTs can be characterized according to the cognitive skills ostensibly measured. Most commonly employed PVTs involve recognition memory paradigms, and in fact, most individuals simulating cognitive deficits from mTBI appear to target verbal memory tasks on which to "display" their impairments [60, 74].

We examined PVT sensitivity rates in a large archival dataset of 135 mTBI test takers determined to be noncredible based on compensationseeking status, failure on a least two PVTs, and low standard cognitive scores at variance with evidence of normal cognitive function in activities of daily living (ADLs). As shown in the table below, the six most sensitive performance validity indicators were in the domains of memory and sensory function.

Domains	n	Sensitivity
Verbal memory	11	
$R \text{ AVI T effort equation: } \leq 12$ [63]	116	70
Wermington words correct:	100	20 20
≤42 [60]	108	82
Rey word recognition: $\leq 6$ [80]	113	63
Visual memory		
RO effort equation: $\leq 50$ [61]	100	77
Rey 15-item+recognition: <20 [81]	104	39
Digit symbol recognition: $\leq 57$ [62]	73	66
Processing speed		
Warrington—words time: ≥207 [60]	85	59
Dot counting test E-score:	127	53
≥17 [65]		
B test E-score: ≥82 [88]	117	61
Attention		
Digit span reliable digit span	135	36
$(RDS) \le 6 [69]$		
Motor speed		
Finger tapping [85]	70; 43	40; 44
Men: $\leq$ 35; women $\leq$ 28		
Visual perceptual		
Picture completion [84]	43	56
Most discrepant index ≤2		
Sensory		
Finger Agnosia errors: >3 [89]	44	66

The following case examples illustrate the heterogeneity in negative response bias displayed by noncredible mTBI test takers. The first case example involves a noncredible test taker claiming cognitive dysfunction secondary to complicated mTBI who demonstrated noncredible performance only on memory PVTs. However, as is shown in subsequent examples described below, some noncredible mTBI patients adopt feigning strategies other than poor memory performance.

*Case #1: Feigned Memory/Attention Impairment:* The patient was a 65-year-old male who had worked in food service and who had completed an Associate's degree; he was tested 7 years post complicated mTBI. Glasgow Coma Scale (GCS) was initially 13/14, but improved to 15 while still in the emergency department; a severe brachial plexus injury left the patient with an inability to use his left arm. Initial brain imaging was normal, but the following day, a small subarachnoid hemorrhage was noted as well as small petechial hemorrhages. On examination the patient failed all PVTs involving verbal (Warrington Words: total=27, 214" [60]; WMS-III Logical Memory Effort Equation=37 [90]; RAVLT Effort Equation = 4 [63]; RO/RAVLT discriminant function=-2.538 [64]; Rey Word Recognition=5 [79, 80]) and visual (Digit Symbol recognition=47 [62]; RO Effort Equation=44 [61, 82]) memory, but scored within normal limits on PVTs involving attention (Digit Span Age-Corrected Scaled Score (ACSS)=10, RDS=9; 3-digit time = 1'' [69]), visual perception (Picture Completion Most Discrepant Index = 6[84]), and processing speed (Dot Counting Test E-score=7 [65]; b Test E-score = 36 [78, 88]). Standard cognitive scores were normal with the exception of variable scores in verbal and visual memory and some timed tasks.

Case #2: Feigned language impairment: The patient was a 36-year-old female with approximately 12 years of education who was suing for damages sustained in a motor vehicle accident 3 years previous to exam; her presentation is described in more detail in Cottingham and Boone [91]. She claimed mTBI and presented with prominent language symptoms that continued to evolve and worsen over time, including decreased word-retrieval, dysarthria, telegraphic speech, loss of prosody, difficulty deciphering written words, emergence of an Eastern European-type "foreign accent," and English as a Second Language (ESL) grammatical errors ("How you say?"). However, interestingly, on examination, articulation errors were inconsistent (e.g., "turkey" was sometimes pronounced correctly and at other times was pronounced as "tur'keen" and "den tur'kee'un") and overlearned number labels were not used (e.g., "eleven" was pronounced "one-one"). Neuropsychological test performance was normal with the exception of lowered scores on some measures involving verbal abilities and processing speed. The patient passed PVTs involving verbal (Warrington Words=47 [60]; RAVLT Effort Equation=17 [63]; Rey Word Recognition=9 [79, 80]; RO/AVLT discriminant function=0.91 [64]) and visual (RO equation=60 [61, 82]) memory, attention/numbers (Dot Counting E-score=11 [65]; Digit Span ACSS=10, RDS=10 [69]), and motor speed (Finger Tapping=44 [85]), but failed indicators involving verbal repetition (time to repeat 3 digits=3", time to repeat 4 digits=4.5" [69]), letter identification (b Test E-score=481.8 [78, 88]) and finger identification (Finger Agnosia errors=5 [92]). Had only memory PVTs been employed, there would have been no psychometric evidence of noncredible symptoms.

Case #3: Feigned Processing Speed and Sensory impairment: The patient was a 42-year-old appliance repairman with 12 years of education who was suing in the context of claimed mTBI sustained in a workplace accident. On medical evaluation, GCS was 15 and the patient was alert and oriented, neurological examination was intact, brain imaging was normal, and the patient's claims of loss of consciousness were not independently verified. On neurocognitive exam 3 years after injury, the patient failed PVTs involving processing speed (Warrington Words time = 369" [60]; time per digit on forward Digit Span = 1.3" [69]), sensory function (Finger Agnosia errors = 5 [92]) and overlearned math skills (Dot Counting errors = 4 [65]), but scored within normal limits on measures involving visual memory (Rey 15-item Memorization Test plus recognition=26 [81]; RO effort equation=62 [61, 82]), verbal memory (Rey Word Recognition Test=12 [79, 80]; Warrington Words=46 [60]; RAVLT/RO discriminant function=0.096 [64]; RAVLT effort equation=16 [63]), attention (Digit Span ACSS=9, RDS=8 **[69**]), rapid letter identification (b Test E-score = 74.4 [78, 88]), visual perception ("most discrepant" index on Picture Completion=5 [84]), and motor speed (finger tapping = 53.7 [85, 93]), and standard cognitive scores were normal with the exception of scores on measures of processing speed and sensory/motor tasks.

*Use of Multiple PVTs in Combination.* It is imperative that multiple PVTs be administered

during a neuropsychological evaluation because response bias may not be constant and, as illustrated in the cases above, noncredible test takers adopt differing strategies in their approach to feigning [66]. Further, multiple PVTs are needed because none have 100 % sensitivity and specificity. Therefore, if only one PVT is administered and a passing score is obtained, it cannot be definitively concluded that the patient is performing to true capability because the individual may fall within the subset of noncredible individuals who in fact pass the test. Likewise, failure on a single PVT administered cannot be used as definitive evidence of feigning in that cut-offs are set to allow a small subset of credible patients to fail (the exception would be cut-offs set to 100 % specificity).

Rather than confusing the situation, administration of increasing numbers of PVTs actually provides the clinician with more confidence in conclusions regarding credibility of performance [94]. That is, research has shown that failure on two PVTs meets or exceeds 95 % specificity, and three failures results in near perfect specificity ( $\geq$ 98.5 %) [5, 95–97]. We frequently encounter mTBI plaintiffs who fail 8, 9, 10, 11, and even 12 PVTs, which is incontrovertible evidence of negative response bias.

Because embedded PVTs do not involve additional test administration time, their use best realizes the goal of substantially increasing available PVT data. The following three mTBI cases illustrate how examination of embedded PVT data moved the likelihood of symptom feigning from probable to definitive.

*Case #4:* The patient was a 39-year-old male attorney involved in a motor vehicle accident 5 years previous; he did not sustain loss of consciousness, head was atraumatic on evaluation, he was alert/fully oriented, and he did not complain of head symptoms in the emergency department; brain CT and MRI were normal. He subsequently returned to work, often billing 15 h in a single day. On neurocognitive examination, he failed two of five dedicated performance validity tests; specifically, he failed the Warrington Recognition Memory Test (Words: 29/50; 11'44") [60] and the

Rey Word Recognition Test (4) [79, 80], but passed Dot Counting (E-score=15) [65], the b Test (E-score=59) [79, 88], and Rey 15-item plus recognition (24) [81].

However, he was also not credible on six of six standard cognitive tests sensitive to feigned performance: Finger Tapping dominant hand (38; cut-off for TBI males) [85], Digit Span (ACSS = 5, RDS = 7, mean time to recite 3 digits = 3") [69, 98], RO Equation (30) [61, 82], RAVLT indices (Effort Equation = 4 [63]; RO/RAVLT discriminant function = -1.775 [64]), Picture Completion Most Discrepant Index (1) [84], and Finger Agnosia errors (4) [92].

Results of standard neurocognitive testing showed impaired scores in visual perception/constructional skill, verbal and visual memory, and finger dexterity, while borderline scores were documented in attention/processing speed; no scores were within normal limits.

What did the embedded PVTs contribute? Failure on two of five dedicated PVTs would be associated with 95 % specificity, but failure on eight total indicators increases specificity to 100 %. Further, the patient failed free-standing PVTs only involving verbal memory, but the failed embedded indicators revealed he was also underperforming in attention, processing speed, visual perception/memory, and motor sensory function, and showed that his symptom feigning strategy involved underperformance in most neurocognitive domains.

*Case #5:* This patient was a 52-year-old male general contractor with 12 years of education who was involved in a motor vehicle accident 3 years previous to exam. He reported loss of consciousness of unknown duration in the collision, but medical records indicated that he did not in fact lose consciousness and was ambulating at the scene, and he did not have any head complaints in the emergency department. The patient returned to work briefly, and then claimed cognitive symptoms interfered with ability to run his business. On neurocognitive examination, he failed one of four dedicated PVTs; specifically, he failed the Warrington Recognition Memory Test (Words = 39; 190") [60], but passed the Rey

Word Recognition Test (7) [79, 80], the Dot Counting Test (E-score = 10) [65], and the Rey 15-item plus recognition (30) [81].

However, he failed five of seven standard cognitive tests sensitive to feigned performance, namely, Finger Tapping dominant hand (37, cutoff for head injured males) [85], Digit Symbol recognition (57) [62], RO Equation (49) [61, 82], RAVLT indices (Effort Equation=8 [63]; RO/ RAVLT discriminant function=-1.538 [64]), and Picture Completion Most Discrepant Index (2) [84], but passed Finger Agnosia errors (3) [92] and Digit Span variables (ACSS=10, RDS=10, mean time to recite 3 digits=1") [69].

On standard neurocognitive testing, average scores or higher were obtained in attention, math skills, problem-solving/reasoning, visual spatial/ constructional skill, language (word-retrieval), and visual memory, while low average scores were found in processing speed, borderline scores were present in verbal recall, and impaired range performance was documented in motor dexterity.

What did the embedded PVTs contribute? Failure on one dedicated PVT is associated with only 59 % specificity, but failure on six total indicators increases specificity to 100 %. Additionally, the failed dedicated PVT only involved verbal memory, but the embedded indicators revealed the patient was also underperforming in processing speed, visual perception/memory, and motor function.

*Case #6:* This 28-year-old male employed in computer sales and attempting to complete a college degree was involved in a motor vehicle accident 3.5 years previous to testing. He exhibited amnesia for the event, but GCS was 15 at the scene, and brain imaging normal. He returned to school and work full-time, but claimed symptoms interfered with ability to function, although there was no change in grades from pre- to postinjury and no poor work evaluations post-injury.

On neurocognitive examination, the patient failed one of five dedicated PVTs; specifically, he failed only the b Test (E-score=1,157) [78, 88], while passing the Warrington Recognition Memory Test (Words=46) [60], Rey Word Recognition Test (9) [79, 80], Dot Counting Test

(E-score = 14) [65], and Rey 15-item plus recognition (29) [81].

However, the patient also exhibited noncredible performance on three of seven standard cognitive tests sensitive to feigned performance, including Finger Agnosia errors (8) [92], the RO/RAVLT discriminant function (-0.798) [64], and Picture Completion Most Discrepant Index (2) [84], but passed the RAVLT Effort Equation (13) [63], Finger Tapping dominant hand (52) [85], Digit Symbol recognition (138) [62], RO effort equation (59) [61, 82], and Digit Span variables (ACSS=13, RDS=13, mean time to recite 3 digits=1") [69].

On standard neurocognitive tests, average scores or better were documented in basic attention, constructional skill, visual memory, executive skills, language, and right hand motor dexterity, while low average/average scores were noted in processing speed, low average performance was found in left hand dexterity, and impaired scores were observed in verbal memory.

What did the embedded PVTs contribute? Like case #5, failure on a single free-standing PVT would be associated with a false-positive identification rate of 41 %, but failure on four total indicators increases specificity to 100 %. The single PVT failure occurred on a measure of processing speed/overlearned information, but the additional failed embedded PVTs also showed that the patient was underperforming in visual perception, verbal memory, and sensory function.

In cases 4 through 6, extra information was obtained, and specificity was increased, at no "extra cost" in terms of test administration time. The use of multiple PVTs provides more confidence in conclusions, and rather than complicating test interpretation, data from many PVTs bring results into "sharp focus." Data from multiple PVTs best protect credible patients from being labeled as noncredible, particularly when  $\geq$ 3 PVT failures are required.

Ideally, it would be preferable if each cognitive measure included an embedded performance validity indicator so that data regarding credibility could be gathered in "real time." The explosion of literature on embedded PVTs [99–101] shows that we are on well on the way to achieving this goal. *Personality Inventories (MMPI-2-RF, PAI, MCM-III).* In addition to PVTs requiring cognitive performance, personality inventories can provide information regarding cognitive symptom overreport in individuals presenting with mTBI. In particular, literature exists on the Minnesota Multiphasic Personality Inventory-2-Restructured Form (MMPI-2-RF) [102], the Personality Assessment Inventory (PAI) [103], and the Millon Clinical Multiaxial Inventory, Third Edition (MCMI-III) [104].

The MMPI-2-RF [102], the recently published and psychometrically advanced version of the MMPI-2 [105], appears to show particular promise in identifying noncredible symptom report in mTBI. A recent study conducted by Youngjohn and colleagues [106] examined litigants with claimed mTBI (n=55), complicated mTBI (n=13), and moderate/severe TBI (n=14). Thirtyfour of the entire sample failed at least one cognitive PVT (noncredible group), and 48 failed none (credible group). The authors found that FBS-r (Symptom Validity scale) successfully discriminated credible from noncredible litigants of all TBI severity levels, but also that the Neurologic Complaints (NUC), Head Pain Complaints (HPC), Gastrointestinal Complaints (GIC), and Malaise (MLS) scales predicted PVT failure, with the most variance in neurocognitive PVT performance accounted for by MLS. Interestingly, mTBI litigants were more likely to produce higher scores on HPC than were their complicated mTBI and moderate/severe TBI counterparts.

Jones and Ingram [107] examined several of the validity scales on the MMPI-2 [105] and MMPI-2-RF [102] in a mixed sample of 288 active duty military personnel, approximately 90 % of whom had sustained mTBI. A subset of 117 participants was judged to be engaging in response bias. The authors reported that that FBS-r, HHI (Henry-Heilbronner Index) [108], and RBS (Response Bias Index) [109] were better at discriminating between credible and noncredible participants than were the Fs (Infrequent Somatic Responses) scale and the F-family scales from the MMPI-2.

Several of the above-described cases illustrate the usefulness of the MMPI-2-RF [102] in mTBI evaluation. In case #3, scores on MMPI-2-RF validity scales indicated that the patient was overreporting psychiatric, physical, and cognitive symptoms in a noncredible manner (F-r=115T; Fs=115T; FBS-r=90T; RBS=114T; RC1=99T; MLS=87T; GIC=80T; HPC=85T; NUC=91T; COG=96T) not explained by random responding or inability to comprehend test questions (VRIN-r=58T; TRIN-r=50T). Similarly, in case #4, MMPI-2-RF validity and clinical scales revealed physical, cognitive, and psychiatric symptom overreport (FBS-r=108T; RBS=101T; F-r=106T; RC1=95T; MLS=87T; GIC=96T; HPC=78T; NUC=91T; COG=80T), and of interest, the low score on VRIN-r (42T), indicated that he was more careful/consistent in completing the protocol than the typical test taker; his extreme carefulness in completing the inventory would not likely be possible if his low cognitive scores were accurate. In case #5, MMPI-2-RF validity scales did not indicate over-report, but the fact that the patient was able to complete the MMPI-2-RF in 1 h with a low VRIN-r score (39T) would argue that cognitive function was likely intact. In case #6, physical/cognitive, and psychiatric symptom over-report was indicated (FBS-r=89T; RBS=90T; F-r=83T; RC1=74T; MLS=87T; GIC=72T; HPC=65T; NUC=86T; COG=86T). Thus, in most of the cases, MMPI-2-RF data provided critical information regarding veracity of patient symptom report.

A search of the literature revealed two studies investigating usefulness of the MCMI-III [104] in measuring credibility of symptom report in mTBI. Aguerrevere and colleagues [110] examined the response patterns of mild (n=76; 71 %) of sample) and moderate-severe TBI patients (n=31) on the MCMI-III; the groups were collapsed after analyses showed that injury severity was not associated with differences in MCMI-III scale elevations. Most of the sample had secondary gain, and approximately half met Slick et al. [111] criteria for malingered neurocognitive dysfunction (presence of external incentive, PVT failure, and performances upon evaluation are markedly discrepant from functional abilities). The authors found that all three validity scales (Disclosure, Desirability, and Debasement) accurately discriminated between malingerers and patients. Using the credible Disclosure, Desirability, and Debasement scales in combination (cut-offs of  $\geq$ 67BR,  $\leq$ 54BR, and  $\geq$ 71BR, respectively), specificity was 96 % with sensitivity reaching 64 %. Less promising results were observed in an earlier study of compensationseeking individuals, 94 % of whom were claiming TBI of unreported severity [112]. Thirty to 34 percent produced PVT failure (TOMM or RDS), but MCMI–III sensitivity was only 1.9 % for the Desirability Scale (cut-off of <24), 2.9 % for the Disclosure Scale (cut-off of  $\geq 85$ ), and 9.5 % for the Debasement Scale (cut-off of  $\geq$ 85).

Whiteside et al. investigated the PAI [103] validity scales and cognitive PVTs in a mixed clinical sample (n=222) [113], but mTBI patients were not examined separately and it unclear how many mTBI patients were included. Additionally, it is unclear how many of the mTBI patients were evaluated in the context of secondary gain, although the authors report that approximately 9 % of their entire sample had external incentive and approximately 11 % failed the TOMM [67, 114]. The authors examined the relationships between validity scales and TOMM performance and found that the Negative Impression Management (NIM) scale and the Infrequency (INF) scale were significantly related to poor performance on the TOMM. In a subsequent study by this group employing the same sample [114], clinical scale elevations and TOMM Trial 2 performance were examined. Only the Somatic Complaints (SOM) scale was significantly related to TOMM Trial 2 performance. Among subscales on the SOM scale, the Conversion subscale (SOM-C) was most associated with PVT performance. A T score of >87 on the SOM scale detected 76 % of those patients exhibiting response bias on the TOMM, at 93 % specificity. In fact, cases #3 and #6 above obtained scores of 92T and 93T, respectively, on the Somatic Complaints scale.

In conclusion, emerging data indicate an important role of personality inventories in documentation of symptom invalidity in the evaluation of mTBI.

## Other Methods to Detect Feigned Cognitive Performance in mTBI

Nonplausible Changes Sequential Across Testings. In addition to the foregoing methods for detecting noncredible performance, one should also be cognizant of atypical patterns of neuropsychological test scores that are inconsistent with the expected course of mTBI. In particular, as shown previously, it is well documented that the cognitive sequelae of mTBI remit relatively quickly [4, 34, 35, 37–40]. Therefore, when an individual produces a normal neuropsychological profile upon evaluation 3 months post-mTBI but then produces a protocol characterized by multiple areas of impairment 1 year post-injury, the data can be used to fortify conclusions regarding noncredible performance [10]. Similarly, "labile" test scores (i.e., those that vacillate over repeated evaluations) are likely indicators of response bias [115].

In case #1, on neurological examination 7 years post-injury, the patient was unable to recall any of three words following a short delay, but on subsequent exam less than 1 month later, he obtained a perfect score (30/30) on the Mini Mental Status Exam (MMSE) [116]. Case #4 had been tested on several occasions prior to the current examination, and test scores had "hopscotched" around in a nonplausible manner; for example, on initial testing 10 months post-injury, IQ scores had been documented in the mentally retarded range, but by 8 months later had risen to average. Case #6 above had been tested 1<sup>1</sup>/<sub>2</sub> years prior to the current examination and at that time all neuropsychological scores had been average or higher, in contrast to the impaired scores observed in verbal memory, and the low average scores documented in processing speed and left hand dexterity, on subsequent testing.

*Implausible Symptom Report.* It is preferable to obtain report of symptoms in an open-ended question format, rather than having patients complete symptom checklists. The latter educate patients as to what symptoms to report. On the other hand, if patients are requested to spontane-

ously report symptoms, at times unusual and implausible reports will be encountered. The genesis of the b Test [78] stemmed from mTBI litigants, who, when asked regarding postaccident cognitive symptoms, reported dyslexia (seeing letters "upside down and backwards"), a symptom we have never encountered in credible moderate to severe TBI patients. Additionally, we have observed some patients, when asked to report problems in thinking skills they attribute to the accident, to respond, "Like what?"; if they have to be provided with examples as to what types of cognitive symptoms one might experience, that would suggest that cognitive abnormalities are not truly a salient problem.

In case #6 above, when asked to provide an example of his claimed attention difficulties, he stated that he will have a fork in his hand and "can't get it to work"; use of utensils is an overlearned type of procedural memory unrelated to attentional abilities. Similarly, some patients, when asked to provide examples of "memory" difficulties, will report that they "forget to eat." Both cases #3 and #6 had claimed strange "fainting" spells/seizures that interestingly did not keep them from driving, and were judged to be syncopal or psychogenic. Cases #2 and #6 reported nonplausible language deficits ("foreign accent" and delayed onset word-retrieval and dysarthria in the former, and "stuttering" in the latter); neither of these symptoms have been reported in the empirical literature to be sequelae of mTBI. Case #6 also claimed to a previous examiner that he had been unconscious for several hours, had several weeks of post-traumatic amnesia, and had been paralyzed for 4 months, none of which were accurate.

Inconsistencies between Test Scores and Spontaneous Behaviors. In addition to datadriven evidence from test performances, behavioral observations can yield critical information regarding symptom validity. For example, if a patient performs within the impaired range on measures of visual memory but is able to independently navigate a complicated maze of hallways in a hospital in order to arrive at the neuropsychologist's office, one has good reason to suspect noncredible test performance. We once evaluated a mTBI patient who, when being administered a trial of a forced-choice PVT in which no feedback regarding accuracy of responses was given, exclaimed that she knew she was doing very poorly because she had already gotten 12 items wrong (an accurate observation, and one that revealed that she had known the correct answers but provided incorrect responses). In case #1 above, the patient scored at chance on verbal recognition memory, yet was able to provide detailed information regarding his daily life activities which included reading (with the read information well-retained), driving (including grocery shopping and running errands), and completing Sudoku puzzles. Many patients perform very poorly on memory testing years remote from the injury, but yet are able to provide rich detail for events surrounding the accident (information seen, smelled, and heard, including exact conversations, etc.). If an individual can recall substantial information from immediately following the injury, when the brain is arguably most dysfunctional, there would be no explanation, absent an intervening neurological event, to account for poor performance on memory testing remote from the injury.

In case #4 above, on finger Agnosia [92] testing, when the middle finger of the patient's left hand was touched, he paused for a lengthy period, and then commented, "That finger is numb" (if so, how did he know, with his eyes closed, that that finger had been touched?). Of note, when queried as to physical symptoms that he associated with the motor vehicle accident, he did not report finger numbness; if one's finger was completely numb, that would seem to be a highly salient symptom that would be unlikely to be omitted from a symptom report. Case #5 scored in the impaired range on finger dexterity, but he was observed to be dexterous in spontaneous motor movements; for example, while holding a piece of paper in his right hand, he used his left hand to hold and punch buttons to text on his cell phone. Some patients will exhibit odd "fast then slow then fast, etc." (e.g., three fast taps, then two slow taps, three fast taps, etc.) rhythms on finger tapping [85] that do not correspond to known patterns of motor dysfunction.

Case #4 was noted to perform extremely slowly on some tasks (time score for Warrington Words [60]; Digit Symbol [62]), but performed quickly on other timed measures (e.g., b Test [78]); true deficits in processing speed should be relatively consistent.

In conclusion, the detection of malingering in mTBI is a multi-factorial enterprise, which involves evidence from various types of PVTs and personality inventories, as well as atypical patterns/inconsistencies between test performances, self-reported symptoms, evidence as to how the person actually functions in spontaneous behaviors, and medical records. It is important to note that there may be factors contributing to responses bias aside from intentional feigning. In particular, patients with somatoform disorders may be more likely to fail PVTs than those without these disorders [117, 118] due to "unconscious" motives to feign illness [119, 120]. It is helpful for the clinician to conceptualize feigning along a continuum, running from intentional, "conscious" feigning on one end (malingering) and unintentional, "unconscious" feigning at the other extreme (somatoform conditions). Because it can sometimes be difficult to determine whether a patient is intentionally vs. nonconsciously engaging in response bias, it is best to use the phrase "noncredible performance" when describing PVT failures. Whether symptom feigning is conscious or nonconscious, standard test scores are uninterpretable, as they likely reflect an underestimate of the patient's actual cognitive capabilities [101].

Should the clinician encounter an individual who appears to have true cognitive deficits and who fails performance validity indicators, further investigation may be necessary. For example, Dean, Victor, Boone, and Arnold [121] examined PVT performance in a heterogeneous neuropsychological clinic sample with no motive to feign and found that patients within lower IQ bands failed more PVTs. Specifically, all patients with IQ<70 failed at least one PVT, and more PVTs were failed as IQ bands decreased, such that patients with IQs ranging from 60 to 69 failed a mean of 44 % of PVTs, and those with IQs ranging from 70 to 79 failed 17 % of PVTs. Likewise,

literature shows that credible patients with dementia also fail PVTs at an excessively high rate (30–70 % specificity at established cut-offs) [122]. Some evidence exists that other credible patient groups may also be susceptible to PVT failure at established cut-offs, in particular patients with moderate to severe TBI [123, 124] (although this may in fact be secondary to low IQ). However, cut-offs can often be adjusted to maintain adequate specificity although with some sacrifice to sensitivity [125, 126].

Further, individuals with actual substantial cognitive deficits may also engage in negative response bias, [127, 128] and a determination that a performance is noncredible does not preclude the presence of true cognitive dysfunction. In these cases, one can only maintain that the patient may have true cognitive deficits, based on the nature of the injury, but no conclusions regarding the extent of these deficits can be made, given noncredible performance on PVTs.

### References

- Bigler, E. D. (2008). Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. *Journal of the International Neuropsychological Society*, 14(1), 1–22.
- McCrea, M., Iverson, G. L., McAllister, T. W., Hammeke, T. A., Powell, M. R., Barr, W. B., et al. (2009). An integrated review of recovery after mild traumatic brain injury (mTBI): Implications for clinical management. *Clinical Neuropsychologist*, 23(8), 1368–1390.
- Ruff, R. M. (2011). Mild traumatic brain injury and neural recovery: Rethinking the debate. *NeuroRehabilitation*, 28(3), 167–180.
- 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 metaanalysis. *Journal of the International Neuropsychological Society*, 11(3), 215–227.
- Larrabee, G. J. (2003). Detection of malingering using atypical performance patterns on standard neuropsychological tests. *Clinical Neuropsychologist*, 17(3), 410–425.
- Mittenberg, W., Patton, C., Canyock, E. M., & Condit, D. C. (2002). Base rates of malingering and symptom exaggeration. *Journal of Clinical and Experimental Neuropsychologist*, 24(8), 1094–1102.
- Armistead-Jehle, P. (2010). Symptom validity test performance in U.S. veterans referred for evaluation

of mild TBI. Applied Neuropsychologist, 17(1), 52–59.

- Larson, E. B., Kondiles, B. R., Starr, C. R., & Zollman, F. S. (2013). Postconcussive complaints, cognition, symptom attribution and effort among veterans. *Journal of the International Neuropsychological Society*, 19(1), 88–95.
- Bush, S. S., Ruff, R. M., Troster, A. I., Barth, J. T., Koffler, S. P., Pliskin, N. H., et al. (2005). Symptom validity assessment: Practice issues and medical necessity: NAN policy and planning committee. *Archives of Clinical Neuropsychologist*, 20(4), 419–426.
- Heilbronner, R. L., Sweet, J. J., Morgan, J. E., Larrabee, G. J., Millis, S. R., & Conference Participants. (2009). American Academy of Clinical Neuropsychologist consensus conference statement on the neuropsychological assessment of effort, response bias, and malingering. *Clinical Neuropsychologist*, 23(7), 1093–1129.
- Greiffenstein, M. F. (2009). Clinical myths of forensic Neuropsychologist. *Clinical Neuropsychologist*, 23(2), 286–296.
- Alexander, M. P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology*, 45(7), 1253–1260.
- Rutherford, W. H., Merrett, J. D., & McDonald, J. R. (1979). Symptoms at one year following concussion from minor head injuries. *Injury*, 10(3), 225–230.
- McLean, A., Temkin, N. R., Dikmen, S., & Wyler, A. R. (1983). The behavioral sequelae of head injury. *Journal of Clinical Neuropsychologist*, 5(4), 361–376.
- Brenner, L. A., Terrio, H., Homaifar, B. Y., Gutierrez, P. M., Staves, P. J., Harwood, J. E., et al. (2010). Neuropsychological test performance in soldiers with blast-related mild TBI. *Neuropsychologist*, 24(2), 160–167.
- Cicerone, K. D., & Kalmar, K. (1995). Persistent postconcussion syndrome: The structure of subjective complaints after mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation*, 10(3), 1–17.
- 17. Stulemeijer, M., Vos, P. E., Bleijenberg, G., & van der Werf, S. P. (2007). Cognitive complaints after mild truamatic brain injury: Things are not always what they seem. *Journal of Psychosomatic Research*, 63(6), 637–645.
- Roe, C., Sveen, U., Alvsaker, K., & Bautz-Holter, E. (2009). Post-concussion symptoms after mild traumatic brain injury: Influence of demographic factors and injury severity in a 1-year cohort study. *Disability and Rehabilitation*, 31(15), 1235–1243.
- Villemure, R., Nolin, P., & Le Sage, N. (2011). Selfreported symptoms during post-mild traumatic brain injury in acute phase: Influence of interviewing method. *Brain Injury*, 25(1), 53–64.
- Nolin, P., Villemure, R., & Heroux, L. (2006). Determining long-term symptoms following mild traumatic brain injury: Method of interview affects self-report. *Brain Injury*, 20(11), 1147–1154.

- Hanna-Pladdy, B., Berry, Z. M., Bennett, T., Phillips, H. L., & Gouvier, W. D. (2001). Stress as a diagnostic challenge for postconcussive symptoms: Sequelae of mild traumatic brain injury or physiological stress response. *Clinical Neuropsychologist*, 15(3), 289–304.
- Whelan, B. M., Murdoch, B. E., & Bellamy, N. (2007). Delineating communication impairments associated with mild traumatic brain injury: A case report. *The Journal of Head Trauma Rehabilitation*, 22(3), 192–197.
- Mathias, J. L., & Coats, J. L. (1999). Emotional and cognitive sequelae to mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychologist*, 21(2), 200–215.
- McHugh, T., Laforce, R., Jr., Gallagher, P., Quinn, S., Diggle, P., & Buchanan, L. (2006). Natural history of the long-term cognitive, affective, and physical sequelae of mild traumatic brain injury. *Brain* and Cognition, 60(2), 209–211.
- Tucker, F. M., & Hanlon, R. E. (1998). Effects of mild traumatic brain injury on narrative discourse production. *Brain Injury*, *12*(9), 783–792.
- Cicerone, K. D. (1996). Attention deficits and dual task demands after mild traumatic brain injury. *Brain Injury*, 10(2), 79–89.
- Johansson, B., Berglund, P., & Ronnback, L. (2009). Mental fatigue and impaired information processing after mild and moderate traumatic brain injury. *Brain Injury*, 23(13–14), 1027–1040.
- 28. Drake, A., Gray, N., Yoder, S., Pramuka, M., & Llewellyn, M. (2000). Factors predicting return to work following mild traumatic brain injury: A discriminant analysis. *The Journal of Head Trauma Rehabilitation*, 15(5), 1103–1112.
- Cicerone, K. D., & Azulay, J. (2002). Diagnostic utility of attention measures in postconcussion syndrome. *Clinical Neuropsychologist*, 16(3), 280–289.
- Carroll, L. J., Cassidy, J. D., Holm, L., Kraus, J., & Coronado, V. G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: The WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine*, 43(Suppl), 113–125.
- Suhr, J. A., & Gunstad, J. (2002). "Diagnosis threat": The negative expectations on cognitive performance in head injury. *Journal of Clinical and Experimental Neuropsychologist*, 24(4), 448–457.
- 32. Suhr, J. A., & Gunstad, J. (2005). Further exploration of the effect of "diagnosis threat" on cognitive performance in individuals with mild head injury. *Journal of the International Neuropsychological Society*, 11(1), 23–29.
- Nolin, P., & Heroux, L. (2006). Relations among sociodemographic, neurologic, clinical, and neuropsychologic variables, and vocational status following mild traumatic brain injury: A follow-up study. *The Journal of Head Trauma Rehabilitation*, 21(6), 514–526.
- Belanger, H. G., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concus-

sion: A meta-analysis. *Journal of the International Neuropsychological Society*, 11(4), 345–357.

- Binder, L. M., Rohling, M. L., & Larrabee, G. J. (1997). A review of mild head trauma. part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical* and Experimental Neuropsychologist, 19(3), 421–431.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, A., et al. (2004). Prognosis for mild traumatic brain injury: Results of the WHO collaboration centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine*, 43(Suppl), 84–105.
- Frencham, K. A. R., Fox, A. M., & Mayberry, M. T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychologist*, 27(3), 334–351.
- Rohling, M. L., Binder, L. M., Demakis, G. J., Larrabee, G. J., Ploetz, D. M., & Langhinrichsen-Rohling, J. (2011). A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analysis and reconsiderations of Binder et al. (1997), Frencham et al. (2005), and Pertab et al. (2009). *Clinical Neuropsychologist*, 25(4), 603–623.
- Schretlen, D. J., & Shapiro, A. M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15(4), 341–349.
- McCrea, M. A. (2008). Mild traumatic brain injury and postconcussion syndrome: The new evidence base for diagnosis and treatment. New York: Oxford University Press.
- Bianchini, K. J., Curtis, K. L., & Greve, K. W. (2006). Compensation and malingering in traumatic brain injury: A dose-response relationship? *Clinical Neuropsychologist*, 20(4), 831–847.
- Kashluba, S., Paniak, C., & Casey, J. E. (2008). Persistent symptoms associated with factors identified by the WHO task force on mild traumatic brain injury. *Clinical Neuropsychologist*, 22(2), 195–208.
- 43. Ettenhofer, M. L., & Abeles, N. (2009). The significance of mild traumatic brain injury to cognition and self-reported symptoms in long-term recovery from injury. *Journal of Clinical and Experimental Neuropsychologist*, *31*(3), 363–372.
- 44. Stulemeijer, M., Andriessen, T. M., Brauer, J. M., Vos Pe, E., & van der Werf, S. (2007). Cognitive performance after mild traumatic brain injury: The impact of poor effort on test results and its relation to distress, personality, and litigation. *Brain Injury*, 21(3), 309–318.
- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *New England Journal of Medicine*, 358, 453–463.
- 46. Rapaport, M. J., McCullagh, S., Shammi, P., & Feinstein, A. (2005). Cognitive impairment associated with major depression following mild and moderate traumatic brain injury. *Journal of Neuropsychiatry* and Clinical Neurosciences, 17(1), 61–65.

- 47. Chamelian, L., & Feinstein, A. (2006). The effect of major depression on subjective and objective cognitive deficits in mild to moderate traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 18(1), 33–38.
- Fann, J. R., Uomoto, J. M., & Katon, W. J. (2001). Cognitive improvement with treatment of depression following mild traumatic brain injury. *Psychosomatics*, 42(1), 48–54.
- 49. Donnell, A. J., Kim, M. S., Silva, M. A., & Vanderploeg, R. D. (2012). Incidence of postconcussive symptoms in psychiatric diagnostic groups, mild traumatic brain injury, and comorbid conditions. *Clinical Neuropsychologist*, 26(7), 1092–1101.
- Verfaellie, M., Lafleche, G., Spiro, A., III, Tun, C., & Bousquet, K. (2013). Chronic postconcussion symptoms and functional outcomes in OEF/OIF veterans with self-report of blast exposure. *Journal of the International Neuropsychological Society*, 19(1), 1–10.
- Raskin, S. A. (1997). The relationship between sexual abuse and mild traumatic brain injury. *Brain Injury*, 11(8), 587–604.
- Pavawalla, S. P., Salazar, R., Cimino, C., Belanger, H. G., & Vanderploeg, R. D. (2013). An exploration of diagnosis threat and group identification following concussion injury. *Journal of the International Neuropsychological Society*, *19*(3), 305–313.
- 53. Lange, R. T., Iverson, G. L., Brooks, B. L., & Rennison, V. L. (2010). Influence of poor effort on self-reported symptoms and neurocognitive test performance following mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychologist*, 32(9), 961–972.
- 54. Cooper, D. B., Mercado-Couch, J. M., Critchfield, E., Kennedy, J., Vanderploeg, R. D., DeVillibis, C., et al. (2010). Factors influencing cognitive functioning following mild traumatic brain injury in OIF/ OEF burn patients. *NeuroRehabilitation*, 26(3), 233–238.
- 55. Landre, N., Poppe, C. J., Davis, N., Schmaus, B., & Hobbs, S. E. (2006). Cognitive functioning and postconcussive symptoms in trauma patients with and without mild TBI. Archives of Clinical Neuropsychologist, 21(4), 255–273.
- 56. Garden, N., & Sullivan, K. A. (2010). An examination of the base rates of post-concussion symptoms: The influence of demographics and depression. *Applied Neuropsychologist*, 17(1), 1–7.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation.
- Iverson, G. L., Zasler, N. D., & Lange, R. T. (2007). Post-concussive disorder. In N. D. Zasler, D. I. Katz, & R. D. Zafonte (Eds.), *Brain injury medicine: Principles and practice* (pp. 373–403). New York: Demos.
- Greve, K. W., & Bianchini, K. J. (2004). Setting empirical cut-offs on psychometric indicators of negative response bias: A methodological commentary with recommendations. *Archives of Clinical Neuropsychologist*, 19(4), 533–541.

- 60. Kim, M. S., Boone, K. B., Victor, T. L., Marion, S. D., Amano, S., Cottingham, M. E., et al. (2010). The Warrington Recognition Memory Test for Words as a measure of response bias: Total score and response time cutoffs developed on "real world" and noncred-ible subjects. *Archives of Clinical Neuropsychologist*, 25(1), 60–70.
- Reedy, S., Boone, K., Cottingham, M., Glaser, D., Lu, P., Victor, T., et al. (2013). Cross-validation of the Lu et al. (2003) Rey-Osterrieth Complex Figure effort equation in a large known groups sample. *Archives of Clinical Neuropsychologist*, 28(1), 30–37.
- 62. Kim, N., Boone, K. B., Victor, T., Lu, P., Keatinge, C., & Mitchell, C. (2010). Sensitivity and specificity of a Digit Symbol recognition trial in the identification of response bias. *Archives of Clinical Neuropsychologist*, 25(5), 420–428.
- Boone, K. B., Lu, P., & Wen, J. (2005). Comparison of various RAVLT scores in the detection of noncredible memory performance. *Archives of Clinical Neuropsychologist*, 20(3), 301–319.
- 64. Sherman, D. S., Boone, K. B., Lu, P., & Razani, J. (2002). Re-examination of a Rey Auditory Verbal Learning Test/Rey Complex Figure discriminant function to detect suspect effort. *Clinical Neuropsychologist*, 16(3), 242–250.
- Boone, K., Lu, P., & Herzberg, D. S. (2002). *The Dot Counting Test manual*. Los Angeles: Western Psychological Services.
- 66. Boone, K. B. (2009). The need for continuous and comprehensive sampling of effort/response bias during neuropsychological evaluation. *Clinical Neuropsychologist*, 23(4), 729–741.
- 67. Tombaugh, T. N. (1996). *Test of Memory Malingering*. Toronto, ON: Multi-Health Systems.
- Delis, D. C., Kramer, H. H., Kaplan, E., & Ober, B. A. (2000). *California Verbal Learning Test* (2nd ed.). San Antonio, TX: The Psychological Corporation.
- Babikian, T., Boone, K. B., Lu, P., & Arnold, G. (2006). Sensitivity and specificity of various Digit Span scores in the detection of suspect effort. *Clinical Neuropsychologist*, 20(1), 145–159.
- Slick, D., Hopp, G., Strauss, E., & Thompson, G. (1996). *The Victoria Symptom Validity Test*. Lutz, FL: Psychological Assessment Resources.
- Greve, K. W., Binder, L. M., & Bianchini, K. J. (2009). Rates of below-chance performance in forced-choice symptom validity tests. *Clinical Neuropsychologist*, 23(3), 534–544.
- 72. Slick, D., Hopp, G., Strauss, E., Hunter, M., & Pinch, D. (1994). Detecting dissimulation: Profiles of simulated malingerers, traumatic brain injury patients, and normal controls on a revised version of Hiscock and Hiscock's forced-choice memory test. *Journal of Clinical and Experimental Neuropsychologist*, 16(3), 472–481.
- Tombaugh, T. N. (1997). The Test of Memory Malingering (TOMM): Normative data from cognitively intact and cognitively impaired individuals. *Psychological Assessment*, 9(3), 260–268.

- 74. Greve, K. W., Ord, J., Curtis, K. L., Bianchini, K. J., & Brennan, A. (2008). Detecting malingering in traumatic brain injury and chronic pain: A comparison of three forced-choice symptom validity tests. *Clinical Neuropsychologist*, 22(5), 896–918.
- Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA division 40 members. *Archives of Clinical Neuropsychologist*, 20(1), 33–65.
- Sharland, M. J., & Gfeller, J. D. (2007). A survey of neuropsychologists' beliefs and practices with respect to the assessment of effort. *Archives of Clinical Neuropsychologist*, 22(2), 213–223.
- 77. Slick, D. J., Tan, J. E., Strauss, E., & Hultsch, D. F. (2004). Detecting malingering: A survey of experts' practices. Archives of Clinical Neuropsychologist, 19(4), 465–473.
- Boone, K., Lu, P., & Herzberg, D. S. (2002). *The b Test manual*. Los Angeles: Western Psychological Services.
- Nitch, N., Boone, K. B., Wen, J., Arnold, G., & Alfano, K. (2006). The utility of the Rey Word Recognition Test in the detection of suspect effort. *Clinical Neuropsychologist*, 20(4), 873–887.
- Bell-Sprinkel, T., Boone, K. B., Miora, D., Cottingham, M., Victor, T., Ziegler, M., et al. (2013). Cross-validation of the Rey Word Recognition symptom validity test. *Clinical Neuropsychologist*, 27, 516–527.
- Boone, K. B., Salazar, X., Lu, P., Warner-Chacon, K., & Razani, J. (2002). The Rey 15-Item recognition trial: A technique to enhance sensitivity of the Rey 15-Item memorization test. *Journal of Clinical* and *Experimental Neuropsychologist*, 24(5), 561–573.
- Lu, P. H., Boone, K. B., Cozolino, L., & Mitchell, C. (2003). Effectiveness of the Rey-Osterrieth Complex Figure Test and the Meyers and Meyers recognition trial in the detection of suspect effort. *Clinical Neuropsychologist*, *17*(3), 426–440.
- Schroeder, R. W., & Marshall, P. S. (2010). Validation of the Sentence Repetition Test as a measure of suspect effort. *Clinical Neuropsychologist*, 24, 326–343.
- 84. Solomon, R. E., Boone, K. B., Miora, D., Skidmore, S., Cottingham, M., Victor, T., et al. (2010). Use of the WAIS-III Picture Completion subtest as an embedded measure of response bias. *Clinical Neuropsychologist*, 24(7), 1243–1256.
- Arnold, G., Boone, K. B., Lu, P., Dean, A., Wen, J., Nitch, S., et al. (2005). Sensitivity and specificity of Finger Tapping test scores for the detection of suspect effort. *Clinical Neuropsychologist*, 19(1), 105–120.
- Binder, L. M. (2002). The Portland Digit Recognition Test: A review of validation data and clinical use. *Journal of Forensic Neuropsychologist*, 2(3–4), 27–41.
- Root, J. C., Robbins, R. N., Chang, L., & van Gorp, W. (2006). Detection of inadequate effort on the

California Verbal Learning Test-Second Edition: Forced choice recognition and critical item analysis. *Journal of the International Neuropsychological Society*, 12(5), 688–696.

- Roberson, C. J., Boone, K. B., Goldberg, H., Miora, D., Cottingham, M., Victor, T., et al. (2013). Crossvalidation of the b Test in a large known groups sample. *Clinical Neuropsychologist.* 27, 495–508.
- 89. Taylor, T. L. (2011). Sensitivity of finger agnosia as a measure of response bias. Doctoral dissertation, California School of Forensic Studies, Alliant International University, Los Angeles. http://gradworks.umi.com/34/57/3457631.html.
- Bortnik, K. E., Boone, K. B., Marion, S. D., Amano, S., Ziegler, E., Cottingham, M. E., et al. (2010). Examination of various WMS-III Logical Memory scores in the assessment of response bias. *Clinical Neuropsychologist*, 24(2), 344–357.
- Cottingham, M. E., & Boone, K. B. (2010). Noncredible language deficits following mild traumatic brain injury. *Clinical Neuropsychologist*, 24(6), 1006–1025.
- Trueblood, W., & Schmidt, M. (1993). Malingering and other validity considerations in the neuropsychological evaluation of mild head injury. *Journal of Clinical and Experimental Neuropsychologist*, 15(4), 578–590.
- Arnold, G., & Boone, K. B. (2007). Use of motor and sensory tests as measures of effort. In K. B. Boone (Ed.), Assessment of feigned cognitive impairment: A neuropsychological approach (pp. 178–209). New York: Guilford.
- Larrabee, G. J. (2008). Aggregation across multiple indicators improves the detection of malingering: Relationship to likelihood ratios. *Clinical Neuropsychologist*, 22(4), 666–679.
- Victor, T. L., Boone, K. B., Serpa, J. G., Buehler, J., & Ziegler, E. A. (2009). Interpreting the meaning of multiple symptom validity test failure. *Clinical Neuropsychologist*, 23(2), 297–313.
- Meyers, J. E., & Volbrecht, M. E. (2003). A validation of multiple malingering detection methods in a large clinical sample. *Archives of Clinical Neuropsychologist*, 18(3), 261–276.
- Suhr, J., Tranel, D., Wefel, J., & Barrash, J. (1997). Memory performance after head injury: Contributions of malingering, litigation status, psychological factors, and medication use. *Journal of Clinical and Experimental Neuropsychologist*, 19(4), 500–514.
- Greiffenstein, M. F., & Baker, W. J. (1994). Validation of malingered amnesia measures with a large clinical sample. *Psychological Assessment*, 6(3), 218–224.
- 99. Boone, K. B. (Ed.). (2007). Assessment of feigned cognitive impairment: A neuropsychological approach. New York: Guilford.
- Larrabee, G. J. (Ed.). (2007). Assessment of malingered neuropsychological deficits. New York: Oxford.
- 101. Boone, K. B. (2013). *Clinical practice of forensic Neuropsychologist*. New York: Guilford.

- 102. Ben-Porath, Y. S., & Tellegen, A. (2008). *Minnesota Multiphasic Personality Inventory-2, Restructured Form.* San Antonio, TX: Pearson.
- Morey, L. C. (1991). *The Personality Assessment Inventory*. Odessa, FL: Psychological Assessment Resources.
- 104. Millon, T., Davis, R., & Millon, C. (1997). Millon Clinical Multiaxial Inventory-III: Manual. Minneapolis, MN: National Computer Systems.
- 105. Butcher, J. N., Graham, J. R., Ben-Porath, Y. S., Tellegen, A., Dahlstrom, W. G., & Kaemmer, B. (2001). *MMPI-2: Manual for administration, scoring, and interpretation* (Revised ed.). Minneapolis: University of Minnesota Press.
- 106. Youngjohn, J. R., Wershba, R., Stevenson, M., Sturgeon, J., & Thomas, M. L. (2011). Independent validation of the MMPI-2-RF somatic/cognitive and validity scales in TBI litigants tested for effort. *Clinical Neuropsychologist*, 25(3), 463–476.
- 107. Jones, A., & Ingraham, M. W. (2011). A comparison of selected MMPI-2 and MMPI-2-RF validity scales in assessing effort on cognitive tests in a military sample. *Clinical Neuropsychologist*, 25(7), 1207–1227.
- 108. Henry, G. K., Heilbronner, R. L., Mittenberg, W., & Enders, C. (2006). The Henry-Heilbronner Index: A 15-item empirically-derived MMPI-2 subscale for identifying probable malingering in personal injury litigants and disability claimants. *Clinical Neuropsychologist*, 20(4), 786–797.
- 109. Gervais, R. O., Ben-Porath, Y. S., Wygant, D. B., & Green, P. (2007). Development and validation of a Response Bias Scale (RBS) for the MMPI-2. *Assessment*, 14(2), 196–208.
- 110. Aguerrevere, L. E., Greve, K. W., Bianchini, K. J., & Ord, J. S. (2011). Classification accuracy of the Millon Clinical Multiaxial Inventory-III modifier indices in the detection of malingering in traumatic brain injury. *Journal of Clinical and Experimental Neuropsychologist*, 33(5), 497–504.
- 111. Slick, D. J., Sherman, E. M. S., & Iverson, G. L. (1999). Diagnostic criteria for malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *Clinical Neuropsychologist*, 13(4), 545–561.
- 112. Ruocco, A. C., Swirsky-Sacchetti, T., Chute, D. L., Mandel, S., Platek, S. M., & Zillmer, E. A. (2008). Distinguishing between neuropsychological malingering and exaggerated psychiatric symptoms in a neuropsychological setting. *Clinical Neuropsychologist*, 22(3), 547–564.
- 113. Whiteside, D. M., Dunbar-Mayer, P., & Waters, D. P. (2009). Relationship between TOMM performance and PAI validity scales in a mixed clinical sample. *Clinical Neuropsychologist*, 23(3), 523–533.
- 114. Whiteside, D., Clinton, C., Diamonti, C., Stroemel, J., White, C., Zimberoff, A., et al. (2010). Relationship between suboptimal cognitive effort and the clinical scales of the Personality Assessment Inventory. *Clinical Neuropsychologist*, 24(2), 315–325.

- Vanderploeg, R. D., & Curtiss, G. (2001). Malingering assessment: Evaluation of validity of performance. *NeuroRehabilitation*, 16(4), 245–251.
- 116. 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.
- 117. Boone, K. B., & Lu, P. H. (1999). Impact of somatoform symptomatology on credibility of cognitive performance. *Clinical Neuropsychologist*, 13(4), 414–419.
- 118. Stone, D. C., Boone, K. B., Back-Madruga, C., & Lesser, I. M. (2006). Has the rolling uterus finally gathered moss? Somatization and malingering of cognitive deficits in six cases of "toxic mold" exposure. *Clinical Neuropsychologist*, 20(4), 766–785.
- 119. Boone, K. B. (2007). Commentary on "Cogniform disorder and cogniform condition: Proposed diagnoses for excessive cognitive symptoms" by Dean C. Delis and Spencer R. Wetter. *Archives of Clinical Neuropsychologist*, 22(6), 675–679.
- Delis, D., & Wetter, S. R. (2007). Cogniform disorder and cogniform condition: Proposed diagnoses for excessive cognitive symptoms. *Archives of Clinical Neuropsychologist*, 22(5), 59–64.
- 121. Dean, A. C., Victor, T. L., Boone, K. B., & Arnold, G. (2008). The relationship of IQ to effort test performance. *Clinical Neuropsychologist*, 22(4), 705–722.
- 122. Dean, A. C., Victor, T. L., Boone, K. B., Philpott, L. M., & Hess, R. A. (2009). Dementia and effort test performance. *Clinical Neuropsychologist*, 23(1), 133–152.
- 123. Curtis, K. L., Greve, K. W., & Bianchini, K. J. (2009). The Wechsler Adult Intelligence Scale-III and malingering in traumatic brain injury: Classification accuracy in known groups. *Assessment*, 16(4), 401–414.
- 124. Ord, J. S., Greve, K. W., & Bianchini, K. J. (2008). Using the Wechsler Memory Scale-III to detect malingering in mild traumatic brain injury. *Clinical Neuropsychologist*, 22(4), 689–704.
- 125. Hacker, V. L., & Jones, C. (2009). Detecting feigned impairment with the word list recognition of the Wechsler Memory Scale-3<sup>rd</sup> edition. *Brain Injury*, 23(3), 243–249.
- 126. Schroeder, R. W., Twumasi-Ankrah, P., Baade, L. E., & Marshall, P.S. (2012). Reliable Digit Span: A systematic review and cross-validation study. *Assessment*, 19, 21–30.
- 127. Bianchini, K. J., Greve, K. W., & Love, J. M. (2003). Definite malingered neurocognitive dysfunction in moderate/severe traumatic brain injury. *Clinical Neuropsychologist*, 17(4), 574–580.
- 128. Bordini, E. J., Chaknis, M. M., Ekman-Turner, R. M., & Perna, R. B. (2002). Advances and issues in the diagnostic differential of malingering versus brain injury. *NeuroRehabilitation*, 17(2), 93–104.

# Special Issues with Mild TBI in Veterans and Active Duty Service Members

Heather G. Belanger, Alison J. Donnell, and Rodney D. Vanderploeg

## Abstract

Mild traumatic brain injury (TBI) or concussion is common in military deployment and combat contexts. As a result, identifying and treating mild TBI has been a high priority within both the Department of Defense (DoD) and Veterans Affairs (VA) since the onset of Operations Enduring Freedom (OEF), Iraqi Freedom (OIF), and more recently New Dawn (OND). Deployment-related personality and behavior changes are common, and are frequently accompanied by a variety of nonspecific symptoms that include sleep problems, irritability, headaches, other bodily aches and pains, concentration difficulties, and memory problems. Common diagnostic comorbidities include adjustment disorders, anxiety disorders including posttraumatic stress disorder (PTSD) and depressive disorders, all of which create TBI diagnostic and treatment challenges. These challenges and how to address them are the focus of this chapter.

H.G. Belanger, Ph.D., ABPP-Cn Department of Mental Health and Behavioral Sciences, James A. Haley VAMC, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA

Department of Psychology, University of South Florida, Tampa, FL, USA

Department of Psychiatry and Neurosciences, University of South Florida, Tampa, FL, USA

A.J. Donnell, Ph.D. Department of Mental Health and Behavioral Sciences, James A. Haley VAMC, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA The Henry M. Jackson Foundation for the Advancement of Military Medicine, Rockville, MD, USA

R.D. Vanderploeg, Ph.D., ABPP-Cn (🖂) Department of Mental Health and Behavioral Sciences, James A. Haley VAMC, Tampa, FL, USA

Defense and Veterans Brain Injury Center, Tampa, FL, USA

Department of Psychology, University of South Florida, Tampa, FL, USA

Department of Psychiatry and Neurosciences, University of South Florida, Tampa, FL, USA e-mail: Rodney.Vanderploeg@va.go

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7\_20, © Springer Science+Business Media, LLC 2014

#### H.G. Belanger et al.

#### Keywords

Brain concussion • Mild traumatic brain injury • Postconcussion symptoms

- Military service members United States Department of Veterans Affairs
- · Health Service Needs and Demands

## Introduction

Mild traumatic brain injury (TBI), also called concussion, may occur in as many as 20 % of combatants [1-3]. Mild TBI is an historical event associated with a physical force to the head with resulting alteration in consciousness. In this chapter, we will focus on those aspects of mild TBI presentation and recovery that may be unique to active duty and veteran populations. Identifying and treating mild TBI has been a high priority within both the Department of Defense (DoD) and Veterans Affairs (VA) since the onset of Operations Enduring Freedom (OEF), Iraqi Freedom (OIF), and more recently New Dawn (OND). Comorbidities, unique aspects of the DoD and VA medical environments, and treatment/evaluation challenges will be discussed.

## **Definition of Mild TBI/Concussion**

TBI is one of the most common and significant medical problems confronting military personnel and has been deemed the "signature injury" of combat in support of Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF). Across both civilian and military populations, approximately 75-90 % of TBIs are mild, although true incidence is difficult to estimate since many mild TBIs are unreported [4-6]. Prevalence rates of mild TBI range from 15 to 30 % of those engaged in active combat in Afghanistan and Iraqi theatres [1, 3, 7]. Assault utilizing explosive devices is a widespread method of warfare, accounts for a large percentage of combat-related injuries and casualties, and is believed to be responsible for 56-78 % of OEF/ OIF-related injuries [8, 9].

Diagnostically, to have sustained a TBI an individual must have experienced an external

physical force that resulted in a traumatically induced structural injury to the brain or a physiological disruption of brain function indicated by medical findings such as positive neuroimaging or an acute alteration in consciousness [10]. An alteration of consciousness may range from confusion disorientation initial and to unconsciousness. Whereas hospital records and/ or neuroimaging findings may assist with the historical diagnosis of more severe and/or acute injury, determining prior mild TBI is typically dependent on patient self-report, facilitated by structured or in-depth clinical interview [11]. Thus, a TBI is an injurious historical event.

An individual with a mild TBI will by definition have a Glasgow Coma Scale (GCS) score [12] of 13–15 within 30 min of injury, i.e., typically by the time they are initially assessed by emergency medical personnel. Thus, within 30 min of injury they obey commands, and at worst may be disoriented and consequently have confused or inappropriate speech. Also by definition, disorientation and mental confusion, if present, lasts less than 1 day. Mild TBI is believed to result when injury triggers a pathological neurochemical cascade, but is insufficient to produce widespread neuronal dysfunction or the axonal disruption that characterizes more severe brain injuries.

Many individuals with mild TBI experience cognitive deficits and postconcussive symptoms immediately after injury. Common symptom complaints include headaches, balance problems, dizziness, fatigue, depression, anxiety, irritability, and memory and attention difficulties, often without demonstrable structural changes to the brain or neuropsychological dysfunction [13, 14]. The majority of these patients makes excellent neurobehavioral recovery, but some report postconcussive symptoms months to years afterward and may experience significant functional impairment [15]. The prevalence of chronic symptoms varies across studies, ranging from 15 to 30 % [16, 17]. However, whether these are truly persistent symptoms associated with the TBI remains controversial. This issue is addressed in more detail below in the section entitled "Postconcussion Syndrome and Symptoms."

## **Common Comorbidities**

In addition to TBI, combat activities may contribute to a wide array of comorbid physical and psychological difficulties that extend well beyond one's time in combat. Following virtually every major war, a significant percentage of combatants return with changes in their personality and behavior patterns, and with a variety of nonspecific symptoms that include sleep problems, fatigue, irritability, headaches, other bodily aches and pains, concentration difficulties and memory problems. This diffuse symptom pattern is often accompanied by some social withdrawal, and a sense of emotional numbness or disconnectedness. A smaller subset meet criteria for a variety of psychiatric diagnoses including: adjustment disorders, anxiety disorders including posttraumatic stress disorder (PTSD), depressive disorders, conversion symptoms and at times classic hysteria, and psychotic reactions. Accompanying these symptoms and conditions are often various physical injuries (either minor or more significant).

Psychological responses to combat trauma can be acute or enduring, and vary across individuals. Symptoms reported post-exposure are often anxious or depressive in nature. Estimates of PTSD prevalence range from 8 to 15 % and rates of depression between 5 and 10 % across studies [2, 18, 19]. In a survey of 2,677 soldiers who had returned home from Iraq 1 month earlier, rates of probable PTSD and depression, as determined by cut scores on questionnaires, were 7 and 9 %, respectively [20].

Although TBI is often discussed as the signature injury of current conflicts, military personnel are sustaining other types of injuries including burns, maxillofacial and ocular injuries, amputations, and blast lung. Complaints of hearing difficulties in those exposed to explosives may well be due to tympanic membrane injury. In examination of 210 U.S. soldiers who underwent neuro-otological evaluation in the Air Force Theater Hospital in Iraq, Xydakis et al. [21] found a significant association between barotraumatic tympanic membrane perforations and concussive brain injury. Overall incidence of tympanic membrane perforation was 35 %, with those wearing ear protection having significantly reduced risk of perforation. Nageris et al. [22] found, in an examination of 73 patients who sustained physical trauma from an explosion, that only 7 % of patients with tinnitus at 3 months post-injury reported an improvement 1 year later.

# Challenges in Teasing Apart Comorbidities

Symptom complaints in those with mild TBI following military deployment have been an area of special interest. However, this has been a difficult area of study due to the presence of comorbid conditions that may present with similar symptoms. For example, Hoge et al. [1] reported that more than 40 % of soldiers who had symptoms associated with mild TBI with loss of consciousness (LOC) also met criteria for PTSD. After adjusting for PTSD and depression, mild TBI was no longer associated with postconcussion symptoms (except headache) or physical health outcomes. These authors suggest that the high rates of physical health problems reported by soldiers with mild TBI are mediated largely or entirely by PTSD or depression, rather than mild TBI. Indeed, other studies have similarly demonstrated that posttraumatic stress and postconcussion symptom reporting are highly interrelated [23-26].

However, a conceptual problem is present with virtually all of these studies. Given the overlap in postconcussion symptoms, PTSD symptoms, and depression in terms of both method of measuring them and associations with current emotional distress, it should not be surprising that such outcomes are more highly associated with each other than they are with possible deployment-related etiologies (e.g., concussion, other physical injuries, or combat) that occurred months or years earlier. Studies essentially confound past events (e.g., concussion and combat exposure) with current health outcomes (e.g., PTSD, depression, postconcussive symptoms, and other symptoms). That is, current PTSD and current depression are typically used as predictors of other current health outcomes and symptoms. Future work in this area will need to clearly distinguish possible deployment-related etiological mechanisms from temporally later post-deployment health outcomes.

In a VA sample, for those with a positive screen for OEF/OIF military deployment-related TBI, one study found a threefold increase in likelihood of having a PTSD diagnosis, and a twofold increase in likelihood of having depression and/or substance-related diagnosis compared to those who screened negative for TBI [27]. A weakness of this study was that groups were based on TBI screening data. Nationwide within the VA, only about 55 % of positive TBI screens are subsequently confirmed to have sustained a deployment-related TBI based on a full clinical evaluation and careful history [28]. Thus, it is possible that the initial TBI screens were positive because of exposure to blasts or other injurious trauma events with resultant psychological symptoms that overlapped with postconcussive symptoms.

## VHA and DoD Systems of Care

# VHA Polytrauma/TBI System of Care

An overview of the Polytrauma System of Care helps in understanding how and where mild TBI care falls within the Veterans Health Administration (VHA) system. This integrated nationwide system of care has been designed to provide access to lifelong rehabilitation services for veterans and active duty service members recovering from polytrauma and TBI. The Polytrauma System of Care has tiered levels of care with four overall components and is described in detail by Sigford [29].

Currently, the four polytrauma rehabilitation centers (PRCs) (Tampa, Florida; Minneapolis, Minnesota; Palo Alto, California; and Richmond, Virginia) are the first component of the VHA's Polytrauma System of Care. A fifth PRC is scheduled to open in San Antonio, Texas. These centers provide acute medical and rehabilitation care, research, and education related to polytrauma and TBI. Clinical care is provided by a dedicated interdisciplinary staff of rehabilitation specialists and medical consultants with expertise in the treatment of the physical, emotional, and psychosocial problems that accompany polytrauma and TBI. The inpatient rehabilitation programs at PRCs maintain accreditation by the Commission on Accreditation of Rehabilitation Facilities (CARF) for both TBI and comprehensive rehabilitation. The environment at PRCs is designed to reflect the demographic of patients served, with family lounges, Internet capability, and media resources appropriate for the age and interests of patients and their families. At the discretion of the military personnel on the unit, injured service members may participate in military formations, be recognized by rank, and wear their military uniforms.

The Polytrauma Network sites (PNSs), designated in December 2005, comprise the second component of care within the Polytrauma System of Care, with one PNS located within each of VHA's 21 regional veterans integrated service networks (VISNs). These PNS programs provide post-acute rehabilitation care for individuals with polytrauma/TBI, including, but not limited to inpatient and outpatient rehabilitation and vocational rehabilitation programs. They are responsible for coordinating access to VHA and non-VHA services across the VISNs to meet the needs of patients recovering from polytrauma and TBI. The PNS consults and collaborates with PRCs in transitioning care from the acute rehabilitation setting to community reintegration.

The Polytrauma System of Care network was expanded in March 2007 to include a new component of care: polytrauma support clinic teams (PSCTs). With their geographical distribution across the VHA, the PSCTs facilitate access to specialized rehabilitation services for veterans and active duty service members at locations close to their home communities. These interdisciplinary teams of rehabilitation specialists are responsible for managing the care of patients with treatment plans, providing regular follow-up visits, and responding to new treatment issues as they arise. The PSCT consults with the affiliated PNS or PRC when more specialized services are required. The remaining VHA medical centers have an identified polytrauma point of contact that is responsible for managing consultations for patients with polytrauma and TBI and referring these patients to appropriate programs capable of providing necessary services.

Patients identified through this system of care receive ongoing case management beginning at the military treatment facility and continuing through VHA hospitalization or outpatient care and on return home. This case management is conducted by both nurses for clinical case management and social workers for psychosocial case management. As patients move through the Polytrauma System of Care, case managers are responsible for a smooth "handoff" or transition to the next phase of care. This handoff includes personal communication with the receiving treatment team, often using videoconferencing. A polytrauma telehealth network allows PRCs and PNSs to communicate using high quality videoconferencing to facilitate discharge, coordination of care, and evaluation for treatment planning. Telehealth technology also links providers and patients at military treatment facilities with PRCs. This has been an invaluable tool in coordination of care and facilitating a smooth and successful transition from one facility to another. It gives providers, patients, and families the opportunity to "meet" one another, communicate, and anticipate future needs.

### DoD and VHA Screening

To enhance early identification and treatment of physical and mental health concerns associated with mild TBI, both the DoD and VHA have implemented screening procedures. The DoD instituted Post-Deployment Health Assessment (PDHA) and Reassessment (PDHRA) programs. The PDHA is scheduled with trained healthcare providers within 30 days after returning to home or to a military processing station. The purpose is

to review each service member's current health, mental health, psychosocial issues commonly associated with deployments, possible deployment-related occupational and environmental exposures (including TBI, which was added in 2008), and to discuss deployment-related health concerns. Positive responses require supplemental assessment and/or referrals for medical consultation. Similarly, the PDHRA is designed to identify and address health concerns, with specific emphasis on mental health, that have emerged over time since deployment. The PDHRA should be completed within the 3- to 6-month time period after return from deployment, ideally at the 3- to 4-month mark because many transitions occur between 90 and 120 days.

Similarly, in an effort to assure appropriate, effective, and timely care, the VHA developed a comprehensive and integrated system of care to treat returning service members. As one entry point, the VHA implemented a series of OEF/ OIF clinical reminders (i.e., mandated clinical questions to ask the veteran, prompted by the electronic medical record) including a TBI clinical reminder protocol that was incorporated into its computerized medical record system in April 2007. These clinical reminders are completed by any provider within the VHA system of care who first encounters that patient following deployment, most frequently a primary care provider. The TBI clinical reminder, which is completed only if the veteran served in Iraq or Afghanistan after September 11, 2001, and if he/she has not already been identified as having a TBI, consists of four questions: (1) Did you have any injury(ies) during your deployment from any of the following? (check all that apply: fragment, bullet, explosion, etc.), (2) Did any injury you received while deployed result in any of the following? (check all that apply: being dazed, confused or "seeing stars," not remembering the injury, losing consciousness, head injury, etc.), (3) Did any of these begin or get worse afterward? (check all that apply: dizziness, headaches, memory problems, balance problems, ringing in the ears, irritability, sleep problems), and (4) In the past week, have you had any of the above symptoms? (check all that apply: dizziness, memory problems, etc.).

A positive response to all four questions constitutes a positive screen. Positive screens automatically generate a consult to a TBI specialist or specialty clinic if the veteran agrees to further assessment or care. This specialist/clinic has 1 week to initiate contact with the patient to schedule a more detailed follow-up evaluation.

# VHA Follow-Up Evaluation and Treatment

Within VHA following a positive TBI clinical reminder, a consult is generated by the provider to obtain a follow-up comprehensive assessment and generation of a plan of care for the patient. This mandated follow-up evaluation, called the Comprehensive TBI Evaluation, consists of further evaluation of blast exposures and TBI events, targeted review of systems, and a physical examination conducted by a licensed medical practitioner with expertise in TBI, typically a physiatrist or a neurologist. Some VHA centers utilize an interdisciplinary team approach with multiple appointments scheduled over several hours to complete the Comprehensive TBI Evaluation. Across different approaches, the purposes of the follow-up evaluation are to: (1) confirm the diagnosis of TBI, even if the present symptoms are felt to be secondary to other factors such as PTSD, stress, depression, or chronic pain, and (2) institute an appropriate plan for follow-up care (e.g., other evaluations or diagnosis-based or symptom-based treatment). Diagnosis-based treatment might include referral to a PTSD program for comorbid PTSD treatment or a mental health clinic for treatment of a comorbid depressive disorder. Symptom-based treatment might consist of treating specific symptoms such as headaches, back pain, insomnia, fatigue, or memory problems, even if the symptom etiology is not clear (e.g., mild TBI, PTSD, post-deployment adjustment, etc.).

Embedded within the computer-based Comprehensive TBI Evaluation template that populates a report in the electronic medical chart is a 22-item postconcussive symptom questionnaire called the Neurobehavioral Symptom Inventory (NSI) [30]. These 22 symptoms are each rated on a scale of 1-5 (none, mild, moderate, severe, and very severe) with four different types of symptoms: affective/psychological/ stress, somatic/physical, neurosensory, and cognitive. The NSI can be useful to gauge patient distress and symptom complaints. A VA/DoD evidence-based mild TBI treatment guideline has been developed (http://www.healthquality. va.gov/management\_of\_concussion\_mtbi.asp). This guideline was designed to help the clinician develop a plan of care and treat the symptom complex identified through the comprehensive evaluation. So, for instance, if concentration problems are endorsed, a review of sleep hygiene is one of many recommended assessments, along with possible treatments.

# **Review of the Relevant Science**

### **Expected Mild TBI Recovery Course**

Most of what is known of the trajectory of cognitive recovery following mild TBI is from the civilian literature. For the overwhelming majority of individuals, a multitude of independent metaanalytic studies indicates a favorable prognosis, with recovery of function over the course of several days to no more than a few months [31–35].

In population-based studies of mild TBI there are acute difficulties (within the first month) with virtually all aspects of neuropsychological functioning adversely affected. Individuals with mild TBI perform about half a standard deviation (d=0.57) more poorly than demographic-matched controls [31]. However, the neuropsychological effect size associated with mild TBI in population-based studies is essentially zero by 3 months post-injury.

In contrast to this typical pattern of excellent recovery following mild TBI in the population at large (i.e., prospective or population-based studies), individuals with mild TBI who present to clinics in the chronic phase for medical or neuropsychological evaluation or who are in litigation (i.e., groups composed of individuals reporting ongoing symptoms and problems) represent a different subsample of patients. At least a portion of these individuals perform more poorly on neuropsychological tests, but in a manner not associated with any specific deficit pattern. In contrast to the expected recovery pattern, these individuals show worsening of neuropsychological performance across time [31]. Factors which may account for the chronic neuropsychological problems found in litigation and clinic-based samples include: self-expectations of difficulties [36], emotional reactions to an adverse event [37], poor coping styles [38, 39], concomitant psychiatric difficulties [40, 41], pain [42, 43] or psychosocial factors [44], malingering as well as subtle residual neurological impairments [45].

## Postconcussion Syndrome and Symptoms

Postconcussive syndrome (PCS) refers to a set of symptoms that can arise after mild TBI, often consisting of physical/somatic (e.g., headache, dizziness, photophobia, fatigue), cognitive (e.g., impaired memory, decreased concentration), and emotional (e.g., depression, irritability) symptoms [46]. Formal diagnostic criteria for PCS vary between the Diagnostic and Statistical Manual of Mental Disorders—4th Edition (DSM-IV) [47] and the International Statistical Classification of Disease and Related Health Problems-10th Edition (ICD-10) [48]. PCS is not an official diagnostic category in the DSM-IV. Rather, it is a set of proposed diagnostic criteria for investigation and includes not only the symptom complaints described earlier but also neuropsychological evidence of attention or memory difficulty. The ICD-10 criteria are not investigational and do not require evidence of neuropsychological dysfunction. In addition, the ICD-10 suggests that there may be hypochondriacal preoccupation, while the DSM-IV does not discuss this possible presentation. Despite the differences, the complex of symptom complaints is quite similar between the two classification systems.

As stated previously, the majority of individuals with mild TBI recover completely within 1–3 months in terms of both cognitive function and structural integrity. However, a significant minority complain of cognitive difficulty and other distressing symptoms months [49–51] or years post injury [52–54]. Some have suggested that these are persisting symptom complaints due to subtle neurological dysfunction allegedly beneath the detection threshold of routine diagnostic procedures such as CT, MRI, and EEG [55, 56]. As discussed in a review of recent neuroimaging literature [57], even more sensitive imaging modalities (such as fMRI) have yet to explain chronic symptom complaints in a convincing way.

Interestingly, so-called "postconcussion" symptoms are not unique to mild TBI and are frequently reported in other medical conditions such as chronic pain and depression [58–60]. As such, some authors argue that while neurological factors may play a role in acute postconcussive symptoms, psychological factors likely play a role in the ongoing maintenance of symptoms. Postconcussive symptoms may be the result of or exacerbated by psychological mechanisms such as poor coping styles [38], emotional reactions to an adverse event [37], or expectations of symptoms that may occur following a mild TBI [36]. Depression and anxiety correlate highly with initial symptoms. Regardless of the cause, these symptoms have the potential to be costly to the military and to the VA, both in terms of continued symptom complaints and healthcare utilization, as well as premature discharge from the military.

Complicating this issue further is whether, in this or other patient populations, these symptoms are simply related to stress or a variety of potential comorbidities. Of note, work by Brenner and colleagues [61] found that among soldiers with histories of physical injury, mild TBI (adjusted prevalence ratio = 4.03; 95 % CI: 2.67-6.07) and PTSD (adjusted prevalence ratio = 2.74; 95 % CI: 1.58-4.74) were independently associated with postconcussive symptom reporting. Similarly, Schneiderman, Braver, and Kang [62] presented data from a retrospective study which suggested that, in a veteran sample, mild TBI and PTSD are independently associated with endorsement of or more postconcussive symptoms. three Adjusted prevalence ratios for such symptoms in

those with mild TBI or PTSD were 1.50 and 3.71, respectively. Finally, results from a recent Florida National Guard survey [63] found that deployment-related mild TBI was a significant predictor of the set of PCS symptoms collectively (Odds = 2.47, CI = 1.53 - 3.98), but so was primary blast exposure (Odds = 1.66, CI = 1.00-2.76), medium (Odds=2.31, CI=1.08-4.93) and high levels of combat exposure (Odds = 3.01, CI=1.43-6.35), and other deployment-related injuries (Odds = 2.61, CI = 1.70-4.01), after controlling for demographic factors, pre-deployment issues, and other deployment-related exposures/ injuries. Deployment-related mild TBI was also a significant predictor for all specific postconcussive symptoms including: headaches, dizziness, balance problems, gait difficulties, fatigue, insomnia, memory problems, concentration difficulties, irritability, anxiety, and depression.

However, in the only prospective study of postconcussion symptoms with a trauma control group [64], during which civilian participants were assessed 5 days after mild TBI and again 3 months later, it was found that postconcussive symptoms waxed and waned over time. Further, postconcussive symptoms were actually unrelated to mild TBI altogether, but instead were associated with accompanying acute posttraumatic stress, and depressive or anxiety disorders.

# Evaluation and Treatment Challenges

# Context of Injury: Military Deployment and Combat Environment

Mild TBI is an historical event associated with an injury and subsequent alteration in consciousness. Being injured via an explosion or other event in a combat zone, often in the midst of a firefight, is an emotionally charged event. Given such circumstances, it is difficult to know whether any reported "alteration" in consciousness is due to a brain concussion, emotional trauma, adrenaline rush, pain related to other bodily injuries, or some other cause. A TBI-induced alteration of consciousness (i.e., confusion, disorientation, incoherency at the scene of the event) is different from a psychologically induced alteration of consciousness, although they may be difficult to distinguish, particularly if information is being obtained months later. Most individuals who are close to an unexpected explosion in which they hear and see the explosion, experience being physically shaken or feel the ground move, and may be struck by environmental debris will likely have an associated adrenalin rush and alteration of consciousness-fear, anxiety, alteration of attention, and/or change in environmental awareness. This is a typical psychological response. However, this type of alteration of consciousness would not mean that the person sustained a TBI. Careful clinical interviewing may be helpful in teasing apart these two types of alteration of consciousness. Simply accepting that a person had an alteration of consciousness as evidence of TBI will result in false-positive diagnoses. Frequently, the clinician's best option is to ask the patient to provide a detailed account of the event, thereby assessing for gaps in memory for the event, or asking what the person was told about his or her behavior or mental state by military colleagues who observed him or her at the site. Nonetheless, it is still difficult to definitively determine the cause (i.e., mild TBI, emotional shock, etc.), even when a likely "alteration" is reasonably established.

Complicating this somewhat is that postconcussion symptoms are not diagnostic of mild TBI and are indistinguishable from the diffuse nonspecific symptoms experienced by many combatants. Although it is clear that a number of individuals have symptoms months and even years following a concussion, it is not clear that these represent a persistent postconcussion syndrome. These symptoms may not be related to an individual's history of TBI as a significant percentage of combatants return with changes in their personality and behavior patterns, and with a variety of nonspecific symptoms that include sleep problems, fatigue, irritability, headaches, other bodily aches and pains, concentration difficulties and memory problems. Indeed, in a prospective study of postconcussion symptoms in

civilians, it was found that acute postconcussive symptoms were not specific to mild TBI and were inconsistently reported over time [64].

## Challenges Related to Reliance on Self-Report and Time Since Injury

Studies frequently utilize symptom inventories to investigate psychological and/or physical symptoms, but many of these inventories do not contain measures of response bias or validity. In addition, inventories typically do not allow for the determination of whether an individual meets full diagnostic criteria for the disorder in question. For example, one study may use a questionnaire that assesses severity of symptom reporting related to PTSD, while another may use a structured diagnostic interview to determine actual diagnosis.

Symptom-based questionnaires give us some idea of symptom type and severity, but have extensive overlap with multiple diagnoses and with other symptom-based measures of other constructs. Shared variance of self-report measures of different clinical entities (such as PTSD, depression, postconcussion syndrome, etc.) can lead to misinterpretation and potentially inflated estimates of correlations between these entities. In other words, multiple symptom measures may simply be alternative measures of the same underlying mental health or physical condition [65]. Also, there is a tendency in this literature to statistically "control for" emotional symptoms in the interest of studying the effects of TBI. This statistical control is controversial from a statistical standpoint, in part because of symptom overlap across multiple psychological conditions and with TBI sequelae [66].

Obtaining a careful history regarding details of potentially injurious events, any event-related loss or alteration of consciousness, and immediate and emerging symptoms over the subsequent hours and days is challenging. When the interview is conducted weeks, months, or years later, patients' memories have faded and merged with other deployment-related experiences. Veterans often describe multiple historical events in which a TBI may have occurred. Many of these events were potentially psychologically traumatizing and may be associated with recurrent intrusive thoughts or nightmares. Discussion of them may also be associated with increased anxiety and physiological arousal (i.e., PTSD symptomology). Furthermore, many Veterans describe chronic sleep disturbances and fatigue throughout their deployment, as well as headaches and back pain from physical activities and carrying heavy equipment. Most view these symptoms as part and parcel of their deployment, and consider them a shared experience with their peers. Asking them to attempt to ascribe onset or worsening of symptoms to particular events in time is problematic.

#### **Political/Social Context**

Given the current political climate and the emphasis on TBI as the "signature injury" of the war on terror, research regarding patient selfexpectation is particularly pertinent. Merely by screening individuals within the context of increased media attention on TBI, the stage is set for expectancies to exert an influence on the patient's belief system and to attribute many or all difficulties to TBI [36]. In addition, the work of Suhr and Gunstad [67, 68] has been particularly illustrative of the effects of the context of the evaluation on cognitive performance outcomes, a situation they refer to as "diagnosis threat." This is quite pertinent in the VA and DoD healthcare settings, with the aforementioned population-based screening and evaluation process. They have demonstrated that calling attention to one's brain injury diagnosis tends to create performance decrements on cognitive tests. Finally, there may be an inclination by the patient to attribute symptoms to TBI, rather than psychological diagnoses such as PTSD, given the perception that a physical brain injury is less a sign of personal or psychological weakness.

Given the denigration experienced by many returning Vietnam era veterans, there currently is a very strong desire to "do the right thing" for the OEF/OIF war-injured veteran. If there is *any* indication of exposure to or injury from blasts, or having sustained even a mild TBI, there is political pressure to assume that current symptoms and
complaints are valid and related to those deployment events. In that context, even if there is evidence for symptom exaggeration or overt malingering, there is pressure to base treatment decisions on patient preference in order to enhance customer satisfaction and avoid "bad press." Finally, even if the entire healthcare team believes that current symptoms are not related to a historic traumatic event, such as a mild TBI or other combat experience, the VHA system of care is nevertheless bound to work with the patient and his/her family to minimize subjective distress and symptoms, regardless of etiology (i.e., PTSD, depression, substance abuse, somatization, conversion, exaggeration, or malingering). As such, this practice environment is unique.

# Treatment Challenges: Lack of Evidence in Military-Relevant Settings

Fortunately, there is effective treatment available for patients having sustained a mild TBI and experiencing postconcussion symptoms or the full postconcussion syndrome. Acute brief psychological treatment significantly reduces the severity and duration of PCS symptoms following mild TBI. Several standardized, empirically supported treatment manuals are available [69, 70]. Even an early single session intervention can prevent the syndrome as effectively as traditional outpatient therapy [71]. In particular, a psychoeducational intervention, that included giving the patient a printed manual and having them meet with a therapist for 1 h prior to hospital discharge, resulted in significantly shorter symptom duration and significantly fewer symptoms at 6-month follow-up compared to a matched control group who received routine hospital care [70]. This 1 h meeting included: reviewing the nature and incidence of expected symptoms, providing the patient with a cognitive-behavioral model of symptom maintenance and treatment, giving the patient-specific techniques for reducing symptoms, and providing them with instructions for gradual resumption of premorbid activities.

Table 1 summarizes the studies published to date with regard to non-medication approaches to

the treatment of PCS symptoms following mild TBI. It is noteworthy that most literature has been developed in the civilian setting and it is therefore unknown the extent to which findings generalize to mild TBI sustained in combat. Certainly, providing the interventions is more challenging. As can be seen from the table, brief treatment is clearly effective in reducing PCS symptoms in patients admitted to the hospital following mild TBI. Numerous studies have found that single session psychoeducational intervention is an effective intervention [36, 72, 73].

The three notable exceptions [74–76] may not have been fair assessments of PCS-specific intervention, as they were not designed specifically to test the efficacy of education and reassurance about PCS symptoms. For example, Ghaffar et al. [75] conducted a treatment study comparing intensive, multidisciplinary rehabilitation treatment with no treatment. This study found no differences between the two groups on PCS symptoms at 6 months follow-up, though for those patients in the treatment group with a history of psychiatric problems, there were fewer depressive symptoms reported at follow-up. Given the present literature, it is unknown if patients seen more chronically (i.e., months or more post-injury) can benefit from this brief, psychoeducational treatment approach.

Only two published studies tested interventions in people with chronic PCS. In these studies, positive results were observed but the treatment was much more involved than the studies conducted on patients evaluated soon after injury [77, 78]. There is currently a gap in the literature with regard to treatment for patients with persisting PCS.

# Key Issues for Effective Clinical Practice and Helpful Tips

# Providing Accurate Information for Providers and Patients About Mild TBI

There appears to be a common misconception among soldiers/veterans, the news media, and some healthcare providers that exposure to a

Table 1 Treatment stuc	lies with mild TBI	patients					
First author	Year published	Design	Treatment(s) tested	Study sample	Control and Tx groups	Follow-up	Tx better than control?
Bell [79]	2008	RCT	Telephone counsel (4–5 calls over 12 weeks)	Within 48 h of injury	N = 159 N = 195	6 months	Yes
Relander [80]	1972	RCT	Activity encouraged; good prognosis emphasized	Within 36 h of hospitalization	N = 96 $N = 82$	1 year	Yes
Minderhoud [81]	1980	Retrospective comparison	Printed + verbal education + activity encouraged	Hospitalized after mild TBI	N = 352 N = 180	6 months	Yes
Gronwall [76]	1986	Not randomized	Printed education	Within 2 weeks of injury	N = 54 $N = 34$	3 months	No
Alves [49]	1993	RCT	<ol> <li>Education only</li> <li>Education + reassurance about recovery</li> </ol>	Hospitalized after mild TBI	N = 210 N = 176 N = 201	3, 6, 12 months	Yes <sup>a</sup>
Ferguson [82] as reported in Miller [83]	1995	Case description	12-Session manualized cognitive-behavioral treatment	Referrals to outpatient clinic	N=4	12 weeks	Yes
Mittenberg [70]	1996	RCT	Handout + 1-h session	Hospitalized after mild TBI	N=29 N=29	6 months	Yes
Wade [84]	1997	RCT	Printed and verbal education + continued support	7–10 days post-injury	N = 130 N = 184	6 months	Yes
Wade [84]	1998	RCT	Advice, support and information	7–10 days post-injury	N = 86 $N = 132$	6 months	Yes
Paniak [72, 85]	1998, 2000	RCT	Single session education	Hospital emergency room	N = 59 $N = 60$	3–4 months, 12 months	Yes <sup>b</sup>
Ponsford [73]	2002	Alternate assignment to group	Information booklet	Hospital emergency room	N = 123 $N = 79$	3 months	Yes
Tiersky [78]	2005	RCT with multiple baselines	CBT+cognitive treatment for 11 weeks	Average of 5 years post-injury	N=7 (milds) N=11	1 and 3 months	Yes
Ghaffar [75]	2006	RCT	Multidiscipline treatment	Within 1 week of injury	N = 94 $N = 97$	6 months	No
Elgmark [74]	2007	RCT	Information, support by multiple disciplines	Median of 3 weeks post-injury	N = 131 N = 264	12 months	No
Notes:							

Special Issues with Mild TBI in Veterans and Active Duty Service Members

400

blast event means that a person sustained a TBI. When working with patients and families, this misconception should be corrected. Exposure can mean multiple things. One soldier may see a Humvee three vehicles in front of his going over an exploding roadside bomb, but not experience any direct or indirect physical effect of the explosion. Another soldier could be in the first Humvee under which the bomb exploded and sustain a broken leg and ruptured tympanic membrane from the force of the blast, and be knocked unconscious for several minutes. While the first soldier did not experience a TBI, the second did.

Additionally, misattribution of symptoms to a residual TBI when such symptoms are secondary to stress, chronic sleep deprivation, PTSD or other mental health condition, could iatrogenically reinforce the misconception that these symptoms are permanent. Even if it is likely that a mild TBI did occur, reassurance and education about expected full recovery is crucial to minimizing any adverse iatrogenic effects. Clinicians, patients, and their families alike should be educated that symptoms frequently attributed to TBI may be due to factors other than TBI, particularly long after the TBI occurred.

Finally, with recent media attention on multiple concussions in the sports arena, and their purported association with dementia, suicide, and other adverse long-term outcomes, it is understandable that both patients and providers may have a more catastrophic reaction to a diagnosis of mild TBI than perhaps may be warranted. Providing education about typical recovery patterns following a mild TBI is crucial.

# Managing and Moderating Patient Expectations

There is a growing body of literature that demonstrates the role of expectation in both cognitive performance and rate of symptom complaint. Mittenberg et al. [36] demonstrated that control subjects asked to imagine symptoms following a head injury reported very similar symptom profiles to individuals who had experienced a mild TBI. Findings also indicated that those with mild TBI underestimated their premorbid problems while reporting post-injury problem severity at levels consistent with "expected post-mild TBI levels" reported by control subjects. If there is an expectation of post-injury problems, then in at least a minority of individuals, such symptoms are more likely to occur. In addition, since virtually everyone with a mild TBI experiences acute symptoms for minutes to hours (e.g., initial confusion, perhaps a new onset headache, dizziness, and difficulty with focused and sustained attention), the mere presence of these symptoms can reinforce preexisting expectations and beliefs. In contrast, if there is no expectation of ongoing symptoms, as is the general case in sports-related mild TBI, then there are few or no ongoing symptoms reported [86, 87]. Both brain dysfunction and expectation likely play independent and interactive causative roles [88].

As discussed above, early post-TBI psychoeducational intervention is important in reducing post-injury anxiety, setting appropriate and realistic expectations for recovery, and enhancing the normal recovery process. This psychoeducational intervention should include providing information regarding the nature and incidence of expected symptoms, normalizing these initial symptoms as an expected part of the recovery process, providing a cognitive-behavioral model for understanding symptoms (e.g., worse with stress or poor sleep, or if emotionally upset), offering specific techniques for reducing symptoms, and providing instructions for gradual resumption of premorbid activities. If patients "expect" to get better and "understand" that initial symptoms are normal and not indicative of a significant problem, then they have a better recovery. In contrast, if patients believe that a symptom is a sign of a worsening or significant neurological problem, then their outcome is far worse. Expectation management is key, utilizing appropriate support, education, early symptom management, and a clear and consistent positive message of recovery over time. The "Home Depot" home improvement stores slogan of "You can do it, we can help" provides a model. The message to patients in the *acute* setting should be:

Your body will recover with rest and time; and if you need help managing or minimizing your symptoms, we can help. Don't overdo it too quickly. An initial period of rest and time for recovery are key. You should be back to normal soon if you allow for healing.

As healthcare providers, we need to be very careful that we do not send a mixed or contradictory message to our patients. We cannot say on the one hand that individuals with a mild TBI/ concussion should have some initial symptoms but that they should gradually diminish over a few days and be back to normal within weeks, while on the other hand we refer them to multiple providers for numerous tests and procedures. A patient's rational internal response to such a situation might be: "If I should get better no matter what happens, then why are you sending me for all these appointments and tests? You must really think that something serious is wrong with me, despite your superficially reassuring statements."

### Interviewing for Chronologically Remote Mild TBI

Often possible mild TBI events are unwitnessed, and clinicians are left to rely on the self-report of patients. Patients typically cannot accurately report if they sustained a LOC. They are likely to erroneously attribute a period of posttraumatic amnesia (PTA) to LOC, but may also erroneously deny LOC because they have some memories for being at the scene. Assessment of amnesia can pose similar challenges. A patient's self-report of PTA, for instance, can be hampered by issues such as psychogenic amnesia, severe physical pain/injuries, intoxication at the time of injury, and medication administered by emergency medical personnel [89]. The criterion of alteration in mental status is particularly challenging to determine. This is especially true for OEF/OIF veteran patients, as their suspected concussions often occurred within the context of emotionally intense and adrenalin laden combat situations or following blast exposure. In such instances, it is challenging for the clinician to differentiate whether the veteran was disoriented or confused as a result of an intense emotional reaction, adrenalin surge, pain from musculoskeletal injuries, or an actual concussion [90].

One of the primary goals of the clinical interview is to recreate the injury event based upon all available information. Given that a diagnosis of concussion is made on the basis of acute injury characteristics, a detailed clinical interview (in conjunction with collateral interview sources and medical record review, if available) has been identified as the "gold standard" for mild TBI diagnosis [11, 90]. When assessing for LOC, the National Academy of Neuropsychology (NAN) Education Paper [89] recommends that clinicians ask patients whether anyone told them they were unconscious or saw them lose consciousness, as opposed to directly asking whether they experienced a LOC per se. However, patients may need to be given an "operational definition" of LOC-"Did any observer indicate that you were unresponsive at the scene, lying or sitting there with your eyes closed and not responding in any way?" For assessment of amnesia, it is important to distinguish between what patients have been told or pieced together about the event versus what they actually remember. Asking very specific questions, such as what is the last thing remembered before the event and what is the first memory after the event can help make this determination. Having patients describe the time periods surrounding the event (before, during, and after) in a free-flowing minute-by-minute manner should be encouraged. Confusion and disorientation can be assessed by establishing a timeline of the accident followed by a clinical determination of whether any confusion and disorientation was a direct result of the neurological insult. Specific queries about the patient's emotional response (e.g., "Were you scared?") can be helpful in differentiating psychological confusion versus physiological confusion.

The present authors, along with a number of other clinician investigators advocate for the use of a structured or semi-structured interview to assess for a concussion history. For example, Corrigan and Bogner [91] published the Ohio State University TBI Identification Method (OSU TBI-ID), which is a structured interview designed to retrospectively identify a history of TBI through self- or proxy-reports. The OSU TBI-ID first elicits recall of all injuries that received medical attention (or warranted medical attention) and then concentrates on those that involved trauma to the head or high-velocity forces. Next, these incidents are further probed for any alterations in consciousness. Lastly, the three most severe injuries are focused upon to assess the onset and course of any post-TBI symptoms and to evaluate for any functional consequences as a result of TBI. The OSU TBI-ID has been found to be a reliable and valid method of assessing lifetime history of TBI [91, 92].

In contrast to the OSU TBI-ID, which explicitly and directly asks about loss or alternation of consciousness, Vanderploeg, Groer, & Belanger [93] assembled a national panel of TBI experts to develop the "VA TBI Identification Clinical Interview" as a semi-structured interview approach that avoids "leading questions." This was designed specifically to assess deploymentrelated TBIs in the post-deployment VA healthcare system months to years following the potential TBI. To minimize reporting bias veterans are not told that the interview is attempting to determine whether or not they sustained a TBI. Rather, patients are first told that the examiner is interested in understanding the effect of "physical forces" on them. Then, they are asked about the "physically most powerful event" they experienced, such as motor vehicle accidents, getting knocked in the head or to the ground, feeling a shock wave, or being hit by debris during an explosion. The interview process is designed to obtain a patient's story while carefully paying attention for information that is consistent with the natural history of TBI. The interview consists of three parts: Part A: Series of open-ended questions (and follow-up probes) to facilitate the patient's freely-told, spontaneous description of the event and any new onset symptoms or problems; Part B: Form for recording the patient's spontaneously reported information from the Part A semi-structured interview; and Part C: Questions and recording form for confirming information acquired during Part A and recorded on the Part B form. Part C is the first time that direct and closed-ended questions are asked of the patient, but it simply confirms what was reported spontaneously. The interview process and recording form assists the interviewer determine: (a) whether or TBI occurred, (b) the severity of that TBI, (c) immediate symptoms, (d) course of symptoms, and (e) total number of TBIs that occurred. The accompanying Manual provides information on how to conduct the interview, and provides clear and unambiguous information regarding the nature and diagnostic criteria of TBI (particularly mild TBI), its severity, and its natural course. Preliminary work has demonstrated good inter-rater reliability of this interview tool [94].

### Distinguishing Between "Persistent" Versus "Current" Symptoms

The "VA TBI Identification Clinical Interview" [93] also assists the clinician in determining the onset and course of symptoms. Current symptoms reported months to years following a concussion in military theater or elsewhere can only be considered postconcussion symptoms (or persistent postconcussion symptoms) if they began at the time of the concussion (within the first 24–72 h) and continued without ever completely remitting to the present time. Evidence suggests that acutely experienced symptoms are inconsistently reported over time [64]. Even persistent symptoms should decrease over time and may disappear for a day or so, but if an increased level of physical or cognitive activity immediately re-activates the identical symptom it can still be considered a postconcussion symptom. However, if a symptom (e.g., headache or dizziness) remits and does not reemerge until a week or more later, that "reemerged symptom" should be thought of as a new symptom with a different etiology. It should not be considered a postconcussion symptom. Then the cause of this new, but similar symptom, must be determined and treated accordingly.

# Adapting Treatment Interventions to the Chronic Post-deployment Setting

The acute psychoeducational message as outlined above will not work without modification in the chronic setting where a service member or veteran presents with multiple symptoms (e.g., headache, insomnia, irritability, anxious or sad mood, concentration problems, and memory difficulties) many months or even a year or more following a deployment-related mild TBI. Providers cannot say "your symptoms should resolve within days to weeks" without losing all credibility. The symptoms are experienced as having lasted for months, and frequently are presumed by the service member/veteran to have been caused by the TBI event. The psychoeducational message must be modified accordingly, and put into terms and a context that the service member/veteran will understand and accept. Veterans should be assured that the ongoing course of symptoms is understandable in light of their having been exposed to a high stress environment for many months. It is not reasonable to expect one's stress response and symptoms to abate rapidly. Furthermore, common postdeployment stressors should be identified and moderated if possible, including help in readjustment to civilian life and re-negotiating relationships with family, friends, and partners. Veterans should be made aware that while it is true that they may have sustained a concussion in theater, most of the symptoms they continue to experience are common symptoms of general stress that are unlikely to be linked with remote concussion history. An initial message such as the following, presented in a calm and empathetic manner, may be an appropriate modification of the empirically supported acute intervention for patients in the more chronic stages:

Of course you are experiencing a variety of symptoms that don't seem to go away. You have been in a high stress environment for a long time. Many of the symptoms that occur following a concussion are the same type of symptoms associated with prolonged exposure to high levels of stress. You also are trying to re-adjust to civilian life. Your symptoms and experiences are normal reactions to abnormal conditions. Stress related to deployment (even with no physical injury to the brain at all) has been shown to adversely affect one's ability to pay attention and remember information. Whether your current symptoms are due to concussion, prolonged stress, re-adjustment issues, or a combination of these factors, they will improve as your stress levels go down and you re-adjust to postdeployment life. However, it took you awhile to get to this point - a year or longer. Your body is not going to re-adjust over night. It won't even happen in a week or two.

Let's monitor your symptoms, and see if they improve. There are things we can do if any particular symptom remains problematic. The important thing to do now is to re-engage in civilian life, try to get caught up on your sleep, and engage in enjoyable social activities with your family and friends. Don't overdo it too quickly, and don't expect improvement over night. This will take a little time. If you continue to experience problems, let us know what they are and we will work together to resolve them.

Current care should focus on a combination of diagnostic-based treatment and symptom reduction, with an emphasis on reducing functional disability and re-engagement in positive life experiences. Patient-provider interactions centered on determining specific etiologies for each of multiple symptoms should be de-emphasized. For those with co-occurring mild TBI and PTSD and/or depression, providers should determine the most appropriate sequence of treatment that needs to be implemented. An OEF/OIF veteran with severe symptoms of PTSD may not be appropriate for TBI rehabilitation services such as cognitive compensatory techniques, and hence may first benefit from stabilization of emotional problems secondary to PTSD or other mental health conditions.

To address residual symptoms, regardless of etiology, a sequential clinical approach has been suggested by Terrio and colleagues [3] and is encouraged by the current authors. The initial step of treatment is providing service members/ veterans and their family members with the expectation for recovery and simultaneously addressing any psychiatric symptoms (e.g., depression) first, regardless of origin. The next step includes attending to somatic complaints (e.g., headaches) and self-care routines (e.g., sleep). Interventions focused on cognitive symptoms (e.g., memory loss) are not generally initiated until the initial two steps are sufficiently addressed, if cognitive symptoms have not resolved by then. Educating service members/ veterans and their family members about the potential interplay between symptoms and the importance of monitoring symptoms and recommended interventions is inherent to the process.

This approach is consistent with recently released mild TBI clinical practice guidelines [95]. Ultimately, this strategy is aimed at helping military personnel and veterans maintain or return to their social roles, thereby facilitating resilience. This treatment approach supports recovery, promotes health behaviors, and enhances resiliency. Utilizing this orientation, struggles related to getting better, frequently referred to as "secondary gain," would be addressed in treatment as barriers to recovery.

#### Case Example: Mr. "Smith"

Mr. Smith, a 24-year-old African American male, was referred for a neuropsychological evaluation by the TBI outpatient clinic due to his complaints about cognitive difficulties.

#### **Background Injury Information**

Mr. Smith was exposed to an improvised explosive device (IED) blast while driving a large truck in Iraq which, as a result, reportedly flipped several times. As is typical in most of these combatrelated situations. no medical records were available from medics at the site. Also typical was that without an extensive period of coma, information regarding acute Glasgow Coma Score, length of loss or alternation of consciousness, and duration of post-event confusion were not available from medical records. Mr. Smith remembers driving and remembers bits and pieces of his medical evacuation. He was able to report details surrounding the roll-over of the truck. He recalled wearing a seatbelt but he struck his head against the side of the vehicle. He did not think that he lost consciousness initially but was unable to provide the same level of detail on events following the crash. He reported a distinct memory, once he was being evacuated, of having a strange feeling of not knowing his whereabouts, followed by severe pain and an awareness that he was bleeding. He remembered being put in a helicopter, as well as portions of his 3-day stay at Landstuhl Regional Medical Center in Germany

for acute medical care. He had bilateral internal fixation for his fractured hips at Walter Reed Army Medical Center (WRMC) and reported participating in physical rehabilitation there for 13 months. He also worked as an intern for the Department of Labor while residing at WRMC.

Comment and discussion: This report of events is typical of a deployment-related injury being evaluated within the VA. Given the lack of gold standard for determining a history of TBI, as discussed in this chapter, the current "gold standard" is to elicit history from the patient in an open-ended manner [93] and determine, to the extent possible, if there was a force to the head and if there were gaps in the patient's recollection that might be consistent with a loss or alteration of consciousness. In this case, it seems likely that the patient sustained a mild TBI given that he struck his head and given the apparent gaps in his ability to provide details following his injury. However, as is typical, this report may be confounded by both emotional and bodily injury. The stress and shock associated with such an event can potentially cause "gaps" in memory, as could significant blood loss. In this case, the fact that he was able to successfully work during his acute rehabilitation suggests that anything more severe than a mild TBI is quite unlikely.

# **Current Subjective Complaints**

Mr. Smith reported significant difficulty with concentration, both in the classroom and at his job. He also complained of frequent headaches and back pain, as well as difficulty sleeping, typically getting 3 or 4 h of sleep per night. He reported that his headaches and cognitive problems were getting worse.

# Education, Psychosocial, Vocational, and Medical Background

Mr. Smith had a high school education with no history of learning or attention problems. He performed infantry duties in the military with no difficulties or disciplinary action, and he reported that he received good reviews in his current job as a counselor at a Vet Center despite his perception that he was not performing to his full potential. He had been working there for about a year. He was collecting a service connected disability for his hip injury. At the time of the evaluation he was taking two classes and earning A's in both courses. He had a 5-year-old son from a previous marriage. He reported that his girlfriend of 3 years just broke up with him the week of the evaluation. He reported feeling depressed with decreased appetite and anhedonia. He reported having suicidal and homicidal thoughts in the past, but denies any currently. He denied any significant medical problems prior to this injury. He had left wrist surgery as a child. At the time of the evaluation he was being prescribed hydrocodone, Tylenol and trazodone. A recent MRI of the brain was read as normal.

#### Assessment Findings

*Behavioral observations*: Mr. Smith arrived on time for his appointment, ambulating with a cane. He appeared very tired initially but aroused considerably as the session progressed. He was fully oriented, with fluent and articulate speech. He provided a detailed, logical, and coherent history that was consistent with existing medical records. He complained of concentration problems which he believed were due to his head injury sustained approximately 3 years earlier.

He walked slowly using a cane and complained of back pain. He described his mood as "down." His affect was flat and depressed, and he reported being quite tearful. When questioned about psychological symptoms in an open-ended manner, he reported multiple symptoms consistent with depression including sadness, feelings of emptiness, anhedonia, early morning awakening, social withdrawal, and fatigue.

*Premorbid estimation*: Mr. Smith denied any difficulty obtaining his high school diploma and reportedly earned A's and B's. His predicted Full Scale IQ score, based on word reading (WTAR) [96] and demographic data, was 102 (with a possible range of 84–120 based on a 95 % confidence interval).

Validity concerns: Mr. Smith's performance during the neuropsychological evaluation was generally inconsistent with his current level of functioning. Specifically, he scored in the impaired range within every cognitive domain despite his ability to function independently, successfully hold down a job, and earn A grades in coursework. Results of symptom validity tests (SVTs) are shown in Table 2. Mr. Smith recalled only 4 digits forward and 2 digits backward (Digit Span 1st percentile; RDS=5) [97]. Mr. Smith failed the Medical Symptom Validity Test (MSVT) [98] as well as the Rey 15 Item Test with Recognition [99]. Despite these failing scores on symptom validity measures, he scored within normal limits on several cognitive measures. Taken together, these findings were interpreted as reflecting variable effort or engagement in the evaluation process.

Neuropsychological profile: Mr. Smith performed in the severely impaired range on a visuospatial measure requiring matching of angles (Judgment of Line Orientation, <1st percentile) [100], but in the average range on a visuoconstruction measure (Rey Osterrieth Complex Figure) [101]. He performed in the average range (37th percentile) on measures of letter-based and category-based verbal fluency (DKEFS) [102]. Measures of processing speed were impaired (DKEFS Color Naming 3rd percentile; DKEFS Word Reading <1st percentile; Digit Symbol 3rd percentile) and characterized by numerous errors. Finally, on a test of visual memory (BVMT-R) [103], he learned 2, 2, and 4 pieces of information (<1st percentile) and recalled only 2 after a delay (<1st percentile). His performance did not improve with a recognition format. He endorsed three of six correct designs, and committed two false-positive errors. Once again, this performance is highly unusual and inconsistent with Mr. Smith's current level of functioning (i.e., working, school, etc.).

Test	Score	Score	Percentile	Description
Validity				
MSVT	Immed. recall	80	n.a.	Failed
	Delayed recall	70	n.a.	Failed
	Consistency	70	n.a.	Failed
	Paired assoc.	50	n.a.	Failed
	Free recall	60	n.a.	Failed
Rey 15+recogn.	Rey 15+recogn.	8/30	Failed	Failed
WAIS-III	Reliable digit span	5 Raw	Failed	Failed
Premorbid				
WTAR		44 Raw	56	Average
Cognitive				
WAIS-III	Digit span	3 ACSS	1	Impaired
	Forward span	4 Raw	<1	Impaired
	Backward span	2 Raw	1	Impaired
	Digit symbol	37 Raw	3	Impaired
BVMT-R	Trial 1	2 Raw	<1	Impaired
	Trial 2	2 Raw	<1	Impaired
	Trial 3	4 Raw	<1	Impaired
	Total 1–3	8 Raw	<1	Impaired
	Learning	2 Raw	16	Low average
	Delayed recall	2 Raw	<1	Impaired
	Recog. hits	3 Raw	<1	Impaired
	Discriminability	1 Raw	<1	Impaired
DKEFS	Letter fluency	33 Raw	37	Average
	Semantic fluency	35 Raw	37	Average
	Color naming	41 Raw	3	Impaired
	Word reading	35 Raw	<1	Impaired
Rey-Osterrieth	Drawing copy	66 Raw	37	Average
Judgment of line orientation		14 Raw	<1	Impaired
Psychological				
BDI-II	Total score	28 Raw	n.a.	Depression
NSI	Total score	67 Raw	n.a.	n.a.
PCL	Total score	40 Raw	n.a.	n.a.

Table 2 Neuropsychological Data for Case Example, "Mr. Smith"

*Notes: MSVT* Medical Symptom Validity Test; *WAIS-III* Wechsler Adult Intelligence Scale – Third Edition; *WTAR* Wechsler Test of Adult Reading; *BVMT-R* Brief Visuospatial Memory Test – Revised; *DKEFS* Delis-Kaplan Executive Function System; *BDI-II* Beck Depression Inventory – Second Edition; *NSI* Neurobehavioral Symptom Inventory; *PCL* PTSD Checklist

*Psychological profile*: During the interview, Mr. Smith endorsed numerous symptoms of depression, as summarized above. His score on the Beck Depression Inventory—II (BDI-II) [104] was consistent with a moderate degree of depressive symptomatology. His score on the Posttraumatic Checklist (PCL) [105], though elevated, was not indicative of PTSD, nor was interview data. Finally, he endorsed numerous "postconcussion" symptoms on the NSI [30] including reportedly severe difficulties with headaches, sleep, and concentration.

# **Discussion of Case**

Mr. Smith is representative of many patients being seen by neuropsychologists within the VA. At face value, the results of his neuropsychological evaluation are difficult to interpret. There are apparent inconsistencies between his current level of functioning and his performance on cognitive tests, which render the cognitive data suspect. It is not possible to assess the extent to which his cognitive symptoms may be due to current depression, a past history of likely mild TBI, or whether they are due to secondary gain or a somatoform process. We do know that his report of worsening cognitive problems is inconsistent with mild or even more severe TBI and far more likely reflect problems with depression and/or maladaptive coping to increasing levels of stress, external incentives, or some other unknown cause.

Despite his overall poor performance during this evaluation, it can be concluded that his visuoconstruction, language, and at least some executive functions are grossly intact, given his intact performance on measures within these cognitive domains. Beyond that, it is impossible to assess his overall level of neurocognitive functioning. Many neuropsychologists might conclude that he is malingering, share this feedback with him, and move on. However, from a VA healthcare systems perspective, this is inadequate. Regardless of the cause of his symptom complaints, the VA is responsible for managing and treating his symptoms. As there is solid evidence to suggest that the "cause" of his cognitive symptoms is not TBI, it is imperative that the patient be educated as such.

Arguably, the current system of care, with its focus on referring all patients who screen positive for TBI to undergo additional evaluations, may have created an iatrogenic effect in this individual. This may well have created or amplified a negative expectation that results in the patient thinking the media's and VA's attention on TBI must mean that a TBI equates to serious functional impairment. We cannot expect patients to know the difference between a mild TBI and a severe TBI, nor can we expect them to appreciate that a brain injury does not necessarily equate to a life sentence of disability. It is unknown whether or not Mr. Smith was pursuing disability associated with TBI. Potential iatrogenic effects, secondary gain, and a lack of medical records related to the injury in question make this case complicated from an etiological perspective but typical from a clinician's perspective within the VA.

Despite etiological uncertainty, there are a number of things the neuropsychologist can do to assist this patient. First, the patient should be educated about the uncertainty of making a TBI diagnosis but nonetheless provided with the clinician's best determination. In this case, it seems likely that the patient did sustain a mild TBI based on a likely period of PTA following impact of his head. Second, the patient should be provided with education regarding the typical recovery pattern of mild TBI and told that his current worsening of cognitive symptoms is inconsistent with what is known about mild TBI. Third, he should be presented with the inconsistencies in his test performance and asked, in an open-ended fashion, about these inconsistencies [106]. Fourth, the clinician should work collaboratively with the patient in discussing the potential impact that his current depression is having on his subjective cognitive functioning and his overall quality of life. This should be done in concert with normalizing post-deployment difficulties and responding to the patient's concerns and perspective in an interactive manner. Finally, recommendations to assist the patient in addressing the depression should be made, as well as tangible educational information provided on mild TBI recovery, symptom management (no matter the cause), and depression.

Treatment recommendations in a case such as this should include outpatient psychotherapy to assist in learning more effective coping strategies and to prevent further escalation of mood-associated symptomatology. Referral to a chronic pain program may be considered to help the patient learn more effective coping strategies and assist him with pain management. Finally, though the patient is currently prescribed an antidepressant, if we take his report at face value, this medication is not effectively alleviating his depressive symptoms. As such, a psychiatric consultation might be useful to evaluate his current medication regime and assist with treatment planning.

#### Resources

www.dvbic.org—provides information for providers, services members, and their families on management of mild TBI.

www.BrainLineMilitary.org—provides militaryspecific information and resources on TBI to veterans, service members in the Army, Navy, Air Force, Marines, National Guard, Reserve, and their families. Through video, webcasts, articles, personal stories, research briefs, and current news, those whose lives have been affected by TBI can learn more about brain injury symptoms and treatment, rehabilitation, and family issues associated with TBI care and recovery.

www.traumaticbraininjuryatoz.org—provides an informative and sensitive exploration of TBI, including information for patients, family members, and caregivers. Topics include types and symptoms of brain injury, TBI treatment and recovery, and helpful insights about the potential long-term effects of brain injury. Animation is used to help patients clearly understand the brain, and the results of injuries to different parts of the brain. Survivors and their caregivers share courageous stories about their own experiences, providing down-to-earth facts along with inspiration and hope.

www.afterdeployment.org—provides self-care solutions targeting post-traumatic stress, depression, and other behavioral health challenges commonly faced after a deployment.

www.biausa.org—Brain Injury Association of America.

# References

- Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *New England Journal of Medicine*, 358(5), 453–463.
- Tanielian, T., & Jaycox, L. H. (Eds.). (2008). Invisible wounds of war: Psychological and cogni-

tive injuries, their consequences, and services to assist recovery. Santa Monica, CA: RAND Corp.

- Terrio, H., Brenner, L. A., Ivins, B. J., Cho, J. M., Helmick, K., Schwab, K., et al. (2009). Traumatic brain injury screening: Preliminary findings in a US Army Brigade Combat Team. *The Journal of Head Trauma Rehabilitation*, 24(1), 14–23.
- Kraus, J., McArthur, J., Silberman, T., et al. (1996). Epidemiology of brain injury. In R. K. Narayan, J. E. Wilberger Jr., & J. T. Povlishock (Eds.), *Neurotrauma* (pp. 13–30). New York: McGraw-Hill.
- Thornhill, S., Teasdale, G. M., Murray, G. D., McEwen, J., Roy, C. W., & Penny, K. I. (2000). Disability in young people and adults one year after head injury: Prospective cohort study. *BMJ*, 320, 1631–1635.
- Defense and Veterans Brain Injury Center. DoD worldwide numbers for traumatic brain injury. Updated November 15, 2011; Retrieved January 19, 2012, from http://www.dvbic.org/TBI-Numbers. aspx
- Schell, T. L., & Marshall, G. N. (2008). Survey of individuals previously deployed for OEF/OIF. Santa Monica, CA: RAND Corp.
- Owens, B. D., Kragh, J. F., Jr., Wenke, J. C., Macaitis, J., Wade, C. E., & Holcomb, J. B. (2008). Combat wounds in operation Iraqi freedom and operation enduring freedom. *Journal of Trauma*, 64(2), 295–299.
- Sayer, N. A., Chiros, C. E., Sigford, B., Scott, S., Clothier, B., Pickett, T., et al. (2008). Characteristics and rehabilitation outcomes among patients with blast and other injuries sustained during the global war on terror. *Archives of Physical Medicine and Rehabilitation*, 89(1), 163–170.
- American Congress of Rehabilitation Medicine. (1993). Report of the mild traumatic brain injury committee of the head injury interdisciplinary special interest group. *The Journal of Head Trauma Rehabilitation*, 8, 86–87.
- Corrigan, J. D., & Bogner, J. (2007). Screening and identification of TBI. *The Journal of Head Trauma Rehabilitation*, 22, 315–317.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, 2(7872), 81–84.
- Levin, H. S., Wilde, E., Troyanskaya, M., Petersen, N. J., Scheibel, R., Newsome, M., et al. (2010). Diffusion tensor imaging of mild to moderate blastrelated traumatic brain injury and its sequelae. *Journal of Neurotrauma*, 27(4), 683–694.
- Lewine, J. D., Davis, J. T., Sloan, J. H., Kodituwakku, P. W., & Orrison, W. W., Jr. (1999). Neuromagnetic assessment of pathophysiologic brain activity induced by minor head trauma. *American Journal of Neuroradiology*, 20, 857–866.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., et al. (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(Suppl), 84–105.

- Rimel, R. W., Giordani, B., Barth, J. T., Boll, T. J., & Jane, J. A. (1981). Disability caused by minor head injury. *Neurosurgery*, 9(3), 221–228.
- Vanderploeg, R. D., Curtiss, G., Luis, C. A., & Salazar, A. M. (2007). Long-term morbidities following self-reported mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 29(6), 585–598.
- Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., & Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *New England Journal of Medicine*, 351(1), 13–22.
- Seal, K. H., Bertenthal, D., Miner, C. R., Sen, S., & Marmar, C. (2007). Bringing the war back home: Mental health disorders among 103,788 US veterans returning from Iraq and Afghanistan seen at Department of Veterans Affairs facilities. *Archives* of Internal Medicine, 167(5), 476–482.
- Ferrier-Auerbach, A. G., Erbes, C. R., Polusny, M. A., Rath, C. M., & Sponheim, S. R. (2010). Predictors of emotional distress reported by soldiers in the combat zone. *Journal of Psychiatric Research*, 44(7), 470–476.
- Xydakis, M. S., Bebarta, V. S., Harrison, C. D., Conner, J. C., Grant, G. A., & Robbins, A. S. (2007). Tympanic-membrane perforation as a marker of concussive brain injury in Iraq. *New England Journal of Medicine*, 357(8), 830–831.
- Nageris, B. I., Attias, J., & Shemesh, R. (2008). Otologic and audiologic lesions due to blast injury. *Journal of Basic and Clinical Physiology and Pharmacology*, 19(3–4), 185–191.
- 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 International Neuropsychological Society*, *16*, 194–199.
- Benge, J. F., Pastorek, N. J., & Thornton, G. M. (2009). Postconcussive symptoms in OEF-OIF veterans: Factor structure and impact of posttraumatic stress. *Rehabilitation Psychology*, 54, 270–278.
- 25. Fear, N. T., Jones, E., Groom, M., et al. (2009). Symptoms of post-concussional syndrome are nonspecifically related to mild traumatic brain injury in UK armed forces personnel on return from deployment in Iraq: An analysis of self-reported data. *Psychological Medicine*, 39, 1379–1387.
- Polusny, M. A., Kehle, S. M., Nelson, N. W., Erbes, C. R., Arbisi, P. A., & Thuras, P. (2011). Longitudinal effects of mild TBI and PTSD comorbidity on postdeployment outcomes in national guard soldiers deployed to Iraq. *Archives of General Psychiatry*, 68, 79–89.
- Carlson, K. F., Nelson, D., Orazem, R. J., Nugent, S., Cifu, D. X., & Sayer, N. A. (2010). Psychiatric diagnoses among Iraq and Afghanistan war veterans

screened for deployment-related traumatic brain injury. *Journal of Traumatic Stress*, 23, 17–24.

- Scholten, J. D., Sayer, N. A., Vanderploeg, R. D., Bidelspach, D. E., & Cifu, D. X. (2012). Analysis of U.S. Veterans Health Administration comprehensive evaluations for traumatic brain injury in Operation Enduring Freedom and Operation Iraqi Freedom Veterans. *Brain Injury*, 26, 1177–84.
- 29. Sigford, B. J. (2008). "To care for him who shall have borne the battle and for his widow and his orphan" (Abraham Lincoln): The Department of Veterans Affairs Polytrauma System Of Care. *Archives of Physical Medicine and Rehabilitation*, 89, 160–162.
- Cicerone, K., & Kalmar, K. (1995). Persistent postconcussive syndrome: Structure of subjective complaints after mild traumatic brain injury. *The Journal* of *Head Trauma Rehabilitation*, 10, 1–17.
- Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcome following mild traumatic brain injury: A meta-analysis. *Journal of International Neuropsychological Society*, 11, 215–227.
- 32. Binder, L. M., Rohling, M. L., & Larrabee, J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, 19, 421–431.
- Frencham, K. A., Fox, A. M., & Maybery, M. T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology*, 27, 334–351.
- 34. Rohling, M. L., Binder, L. M., Demakis, G. J., Larrabee, G. J., Ploetz, D. M., & Langhinrichesen-Rohling, J. (2011). A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analyses and reconsiderations of Binder et al., (1997), Frencham et al. (2005), and Pertab et al. (2009). *Clinical Neuropsychology*, 25, 608–623.
- Schretlen, D. J., & Shapiro, A. M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15, 341–349.
- Mittenberg, W., DiGiulio, D. V., Perrin, S., & Bass, A. E. (1992). Symptoms following mild head injury: Expectation as aetiology. *Journal of Neurology, Neurosurgery, and Psychiatry, 55*, 200–204.
- Bryant, R. A., & Harvey, A. G. (1999). Postconcussive symptoms and posttraumatic stress disorder after mild traumatic brain injury. *Journal of Nervous and Mental Disease*, 187, 302–305.
- Bohnen, L., Jolles, J., Twijnstra, A., Mellink, R., & Sulon, J. (1992). Coping styles, cortisol reactivity, and performance on vigilance tasks of patients with persistent postconcussive symptoms after mild head injury. *International Journal of Neuroscience*, 64, 97–105.

- Marsh, H. V., & Smith, M. D. (1995). Postconcussion syndrome and the coping hypothesis. *Brain Injury*, 9, 553–562.
- Fann, J. R., Katon, W. J., Uomoto, J. M., & Esselman, P. C. (1995). Psychiatric disorders and functional disability in outpatients with mild traumatic brain injuries. *The American Journal of Psychiatry*, 152, 1493–1499.
- 41. Trahan, D. E., Ross, C. E., & Trahan, S. L. (2001). Relationships among postconcussional-type symptoms, depression, and anxiety in neurologically normal young adults and victims of mild brain injury. *Archives of Clinical Neuropsychology*, 16, 435–445.
- Nicholson, K. (2000). Pain, cognition and traumatic brain injury. *NeuroRehabilitation*, 14, 95–103.
- Perlis, M. L., Artiola, L., & Giles, D. E. (1997). Sleep complaints in chronic postconcussion syndrome. *Perceptual and Motor Skills*, 84, 595–599.
- 44. Luis, C. A., Vanderploeg, R. D., & Curtiss, G. (2003). Predictors of postconcussion symptom complex in community dwelling male veterans. *Journal of International Neuropsychological Society*, 9, 1001–1015.
- Vanderploeg, R. D., Curtiss, G., & Belanger, H. G. (2005). Adverse long-term neuropsychological outcomes following mild traumatic brain injury. *Journal* of International Neuropsychological Society, 11, 228–236.
- Hall, R. C., Hall, R. C., & Chapman, M. J. (2005). Definition, diagnosis, and forensic implications of postconcussional syndrome. *Psychosomatics*, 46, 195–202.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Publishing.
- World Health Organization. (1992). International statistical classification of diseases and related health problems (10th ed.). Geneva, Switzerland: WHO.
- Alves, W., Macciocchi, S., & Barth, J. (1993). Postconcussive symptoms after uncomplicated mild head injury. *The Journal of Head Trauma Rehabilitation*, 8, 48–59.
- Dikmen, S., McLean, A., & Temkin, N. (1986). Neuropsychological and psychosocial consequences of minor head injury. *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 49, 1227–1232.
- Powell, T. J., Collin, C., & Sutton, K. (1996). A follow-up study of patients hospitalized after minor head injury. *Disability and Rehabilitation*, 18, 231–237.
- Alexander, M. P. (1992). Neuropsychiatric correlates of persistent postconcussive syndrome. *The Journal* of *Head Trauma Rehabilitation*, 8, 60–69.
- Deb, S., Lyons, I., & Koutzoukis, C. (1999). Neurobehavioural symptoms one year after a head injury. *British Journal of Psychiatry*, 174, 360–365.
- Hartlage, L. C., Durant-Wilson, D., & Patch, P. C. (2001). Persistent neurobehavioral problems follow-

ing mild traumatic brain injury. *Archives of Clinical Neuropsychology*, *16*(6), 561–570.

- Hayes, R. L., & Dixon, C. E. (1994). Neurochemical changes in mild head injury. *Seminars in Neurology*, 14(1), 25–31.
- 56. Povlishock, J. T., & Coburn, T. H. (1989). Morphopathological change associated with mild head injury. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Mild head injury* (pp. 37–53). New York: Oxford University Press.
- Belanger, H. G., Vanderploeg, R. D., Curtiss, G., & Warden, D. (2007). Recent neuroimaging techniques in mild traumatic brain injury: A critical review. *Journal of Neuropsychiatry and Clinical Neurosciences*, 19, 5–20.
- Gunstad, J., & Suhr, J. A. (2001). "Expectation as etiology" versus "the good old days": postconcussion syndrome symptom reporting in athletes, headache sufferers, and depressed individuals. *Journal of International Neuropsychological Society*, 7(3), 323–333.
- Gunstad, J., & Suhr, J. A. (2004). Cognitive factors in postconcussion syndrome symptom report. *Archives* of *Clinical Neuropsychology*, 19(3), 391–405.
- 60. Smith-Seemiller, L., Fow, N. R., Kant, R., & Franzen, M. D. (2003). Presence of post-concussion syndrome symptoms in patients with chronic pain vs mild traumatic brain injury. *Brain Injury*, 17, 199–206.
- 61. Brenner, L. A., Ivins, B. J., Schwab, K., et al. (2010). Traumatic brain injury, posttraumatic stress disorder, and postconcussive symptom reporting among troops returning from Iraq. *The Journal of Head Trauma Rehabilitation*, 25, 307–312.
- 62. Schneiderman, A. I., Braver, E. R., & Kang, H. K. (2008). Understanding sequelae of injury mechanisms and mild traumatic brain injury incurred during the conflicts in Iraq and Afghanistan: Persistent postconcussive symptoms and posttraumatic stress disorder. *American Journal of Epidemiology*, 167(12), 1446–1452.
- 63. Scott, S. G., Belanger, H. G., & Vanderploeg, R. D. (2011, June 13–15). Sorting through the contributions to health outcomes from the Florida National Guard Survey Study: Blast and TBI effects. Symposium presented at 3rd Federal Interagency Conference on TBI, Washington, DC.
- 64. Meares, S., Shores, E. A., Taylor, A. J., et al. (2011). The prospective course of postconcussion syndrome: The role of mild traumatic brain injury. *Neuropsychology*, 25(4), 454–465.
- O'Donnell, M. L., Creamer, M., & Pattison, P. (2004). Posttraumatic stress disorder and depression following trauma: Understanding comorbidity. *The American Journal of Psychiatry*, 161(8), 1390–1396.
- Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology*, 110(1), 40–48.
- 67. Suhr, J. A., & Gunstad, J. (2002). "Diagnosis threat": The effect of negative expectations on cognitive

performance in head injury. *Journal of Clinical and Experimental Neuropsychology*, 24, 448–457.

- Suhr, J. A., & Gunstad, J. (2005). Further exploration of the effect of "diagnosis threat" on cognitive performance in individuals with mild head injury. *Journal of International Neuropsychological Society*, 11, 23–29.
- Mittenberg, W., Zielinski, R., & Fichera, S. (1993). Recovery from mild head injury: A treatment manual for patients. *Psychotherapy in Private Practice*, *12*, 37–53.
- Mittenberg, W., Tremont, G., Zeilinski, R. E., Fichera, S., & Rayls, K. R. (1996). Cognitivebehavioural prevention of postconcussion syndrome. *Archives of Neurology*, 11, 139–145.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the postconcussion syndrome. *The Journal of Head Trauma Rehabilitation*, 15(2), 783–791.
- 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, 219–226.
- Ponsford, J., Willmott, C., Rothwell, A., et al. (2002). Impact of early intervention on outcome following mild head injury in adults. *Journal of Neurology, Neurosurgery, and Psychiatry*, 73(3), 330–332.
- 74. Elgmark, A. E., Emanuelson, I., Bjorklund, R., & Stalhammar, D. A. (2007). Mild traumatic brain injuries: The impact of early intervention on late sequelae. A randomized controlled trial. *Acta Neurochirurgica (Wien)*, 149(2), 151–159.
- Ghaffar, O., McCullagh, S., Ouchterlony, D., & Feinstein, A. (2006). Randomized treatment trial in mild traumatic brain injury. *Journal of Psychosomatic Research*, 61(2), 153–160.
- Gronwall, D. (1986). Rehabilitation programs for patients with mild head injury: Components, problems, and evaluation. *The Journal of Head Trauma Rehabilitation*, 1, 53–63.
- 77. Huckans, M., Pavawalla, S., Demadura, T., et al. (2010). A pilot study examining effects of groupbased cognitive strategy training treatment on selfreported cognitive problems, psychiatric symptoms, functioning, and compensatory strategy use in OIF/ OEF combat veterans with persistent mild cognitive disorder and history of traumatic brain injury. *Journal of Rehabilitation Research and Development*, 47(1), 43–60.
- Tiersky, L., Anselmi, V., Johnston, M., et al. (2005). A trial of neuropsychologic rehabilitation in mildspectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 86, 1565–1574.
- Bell, K. R., Hoffman, J. M., Temkin, N. R., et al. (2008). The effect of telephone counselling on reducing post-traumatic symptoms after mild traumatic brain injury: A randomised trial. *Journal* of Neurology, Neurosurgery, and Psychiatry, 79(11), 1275–1281.

- Relander, M., Troupp, H., & Bjorkesten, G. (1972). Controlled trial of treatment for cerebral concussion. *BMJ*, *4*, 777–779.
- Minderhoud, J. M., Boelens, M. E., Huizenga, J., & Saan, R. J. (1980). Treatment of minor head injuries. *Clinical Neurology and Neurosurgery*, 82(2), 127–140.
- 82. Ferguson, R. J., & Mittenberg, W. (1996). Cognitivebehavioral treatment of postconcussion syndrome: A therapist's manual. In V. B. Van Hassel & M. Hersen (Eds.), Sourcebook of psychological treatment manuals for adult disorders (pp. 615–655). New York: Plenum Press.
- Miller, L. J., & Mittenberg, W. (1998). Brief cognitive behavioral interventions in mild traumatic brain injury. *Applied Neuropsychology*, 5(4), 172–183.
- Wade, D. T., King, N. S., Wenden, F. J., Crawford, S., & Caldwell, F. E. (1998). Routine follow up after head injury: A second randomised controlled trial. *Journal of Neurology, Neurosurgery, and Psychiatry*, 65(2), 177–183.
- Paniak, C., Toller-Lobe, G., Durand, A., & Nagy, J. (1998). A randomized trial of two treatments for mild traumatic brain injury. *Brain Injury*, *12*(12), 1011–1023.
- Ferguson, R. J., Mittenberg, W., Barone, D. F., & Schneider, B. (1999). Postconcussion syndrome following sports-related head injury: Expectation as etiology. *Neuropsychology*, 13, 582–589.
- Mittenberg, W., Ferguson, R. J., & Miller, L. J. (1997). Postconcussion syndrome following sport related head injury: Expectation as etiology (abstract). *Journal of International Neuropsychological Society*, *3*, 13.
- Vanderploeg, R. D., Belanger, H. G., & Curtiss, G. (2006). Mild traumatic brain injury: Medical and legal causality considerations. In G. Young, A. Kane, & K. Nicholson (Eds.), *Psychological knowledge in court: PTSD, pain and TBI* (pp. 279–307). New York: Springer.
- Ruff, R. M., Iverson, G. L., Barth, J. T., Bush, S. S., Broshek, D. K., & NAN Policy Planning Committee. (2009). Recommendations for diagnosing a mild traumatic brain injury: A National Academy of Neuropsychology education paper. *Archives of Clinical Neuropsychology*, 24, 3–10.
- Belanger, H. G., Uomoto, J. M., & Vanderploeg, R. D. (2009). The Veterans Health Administration system of care for mild traumatic brain injury: Costs, benefits, and controversies. *The Journal of Head Trauma Rehabilitation*, 24, 4–13.
- Corrigan, J. D., & Bogner, J. (2007). Initial reliability and validity of the Ohio State University TBI identification method. *The Journal of Head Trauma Rehabilitation*, 22, 318–329.
- Corrigan, J. D., & Bogner, J. (2009). Reliability and predictive validity of the Ohio State University TBI identification method with prisoners. *The Journal of Head Trauma Rehabilitation*, 24, 279–291.

- Vanderploeg, R. D., Groer, S., Belanger, H. G. (2012). Initial development process of a VA semistructured clinical interview for TBI identification. *Journal of Rehabilitation Research and Development*, 49, 546–556.
- 94. White, K. T., Gutmann, J. M., Vanderploeg, R. D., & Groer, S. (2011). Inter-interviewer reliability of a new VA TBI identification clinical interview. Unpublished manuscript.
- 95. Department of Veterans Affairs and Department of Defense. (2009, April). VA/DOD clinical practice guideline for management of concussion/mild traumatic brain injury [Internet]. Retrieved January 19, 2012, from http://www.healthquality.va.gov/mtbi/ concussion\_mtbi\_full\_1\_0.pdf
- 96. Wechsler, D. (2001). *Wechsler test of adult reading* (*WTAR*). San Antonio, TX: Harcourt Assessment.
- Wechsler, D. (1997). Wechsler adult intelligence scale-III. San Antonio, TX: The Psychological Corporation.
- Green, P. (2004). Green's medical symptom validity test (MSVT) for windows: user's manual. Edmonton, Alberta, Canada: Green's Publishing.
- 99. Rey, A. (1964). *L'examen clinique en psychologie*. Paris: Presses Universitaires de France.
- Benton, A. L., Sivan, A. B., Hamsher, K. S., Varney, N. R., & Spreen, O. (1994). *Contributions to neuro-*

*psychological assessment* (2nd ed.). Orlando, FL: Psychological Assessment Resources.

- Rey, A. (1993). Psychological examination of traumatic encephalopathy [J. Corwin & F. W. Bylsma (trans.)]. *Clinical Neuropsychology*, 7, 3–21 [Original work published in 1941].
- 102. Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). Delis-Kaplan executive function system. San Antonio, TX: The Psychological Corporation.
- Benedict, R. H. B. (1997). Brief visuospatial memory test—revised. Odessa, FL: Psychological Assessment Resources.
- 104. Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck depression inventory-II. Manual. San Antonio, TX: Psychological Corporation.
- 105. Weathers, F. W., Litz, B. T., Herman, D. S., Huska, J. A., Keane, T. M. (1993, October). *The PTSD checklist: Reliability, validity, and diagnostic utility.* Paper presented at Annual Convention of the International Society for Traumatic Stress Studies, San Antonio, TX.
- 106. Carone, D. A., Iverson, G. L., & Bush, S. S. (2010). A model to approaching and providing feedback to patients regarding invalid test performance in clinical neuropsychological evaluations. *Clinical Neuropsychology*, 24, 759–778.

# Index

#### A

Alzheimer's disease, 336, 338-339 Anticholinergic Cognitive Burden (ACB) Scale, 334 Anticholinergic Drug Scale, 334 Apolipoprotein (APOE), 339 Assessment, Review, and Dismissal (ARD) committee, 319 Attention-executive functions direct training interventions APT-3, 198 BI-ISIG Cognitive Rehabilitation Task Force, 199-200 focused/sustained attention, 198 functional cerebral activation, 199 selective/alternating attention, 198 working memory/divided attention, 198 impairments alerting attention network, 193 anticipatory neural network, 194 attentional system, 194 cognitive impairments, 195-196 DFC model, 194-195 emotional and behavioral regulation, 196 endogenous and exogenous stimulus control, 194 executive attention network, 193 large-scale distributed neural networks, 193 metacognitive functions, 195 metacognitive impairments, 196-197 orienting attention network, 193 real-world and clinical implications, 196-197 revamped attentional model, 194 rostral and lateral system, 195 ventral system, 195 metacognitive knowledge, 193 metacognitive training intervention anticipate and plan, 203 awareness, 202-203 execute and self-monitor, 203-204 multifaceted approach, 200 patient history, 205-207 problem-solving deficits, 204 self-evaluation process, 204 Stuss' theoretical framework, 201 self-monitoring process, 193 Attention Process Training (APT) program, 198, 293

#### B

Beck Depression Inventory-II (BDI-II), 262, 374 Bedside evaluations CAP (see Confusion assessment protocol (CAP)) characteristics, 51 cognitive abilities, 50-51 cognitive assessment (see Cognitive assessment) CRS-R (see Coma recovery scale-revised (CRS-R)) DOC, 50 emotional status, 50-51 depression and anxiety, 51, 69 GAD-7, 70, 71 PHO-9, 70, 71 psychiatric disturbances, 69 recommendations, 70-71 scaling, 70 language abilities, 50 MAST (see Mississippi aphasia screening test (MAST)) neuropsychological assessment, 50 posttraumatic confusion, 50 Behavior Intervention Plan (BIP), 319 Behavior management acute vs. post-acute stages, 158 agitation amygdala functions, 159 cognitive impairment, 159 environmental stimulation, 160 external stimulation, 159 impulse control/emotional lability, 159 pharmacological interventions, 160 posttraumatic amnesia, 158 brain injury development, 160-162 functional analyses definition, 162, 163 hypothesis, 162 reinforcement and punishment techniques, 163 three-term contingency model, 162 hitting/striking of therapists, 165-167 implementation, 170 inappropriate sexual behaviors, 163-165 population specific groups, 170 uncooperative with vitals exam, 167-169 Behavior Rating Inventory of Executive Function-Preschool, 312

M. Sherer and A.M. Sander (eds.), *Handbook on the Neuropsychology of Traumatic Brain Injury*, Clinical Handbooks in Neuropsychology, DOI 10.1007/978-1-4939-0784-7, © Springer Science+Business Media, LLC 2014 Ben-Yishay/Prigatano model, 284
Brain concussion. See Mild traumatic brain injury (mTBI)
Brain Injury-Interdisciplinary Special Interest Group (BI-ISIG), 199–200
Brief Infant and Toddler Social Emotional Assessment, 312

#### С

Canadian CT Head Rule (CCHR), 112 CAP. See Confusion assessment protocol (CAP) CBIR. See Comprehensive brain injury rehabilitation (CBIR) Center for Epidemiological Studies-Depression Scale, 262 Child Behavior Checklist, 312 Children's Head injury Algorithm for the prediction of Important Clinical Events (CHALICE) rule, 113 Cognitive and behavioral outcomes mitigating factors APOE-4 allele, 37 caregivers and family functioning, 37 environmental factors, 38 ethnicity and minority status, 38 litigation and financial incentives, 39 penetrating head injuries, 36-37 moderate-severe TBI cognitive deficits, 29, 30 communication problems, 29 independent living, 32 life satisfaction, 32-33 neurobehavioral symptoms, 29 productivity outcomes, 30-32 PTA, 29 PTC, 29 social functioning and community integration, 32 vegetative and minimally conscious states, 28-29 mTBI, 26-27 complicated mTBI, 28 repeated mTBI, 27-28 psychological sequelae anxiety, 34-35 mood disorders, 33-34 personality disorders, 35 schizophrenia and psychosis, 35 substance abuse disorders, 36 Cognitive assessment assessment batteries, 67-68 auditory number search test, 68 cognitive impairment memory impairment, 66 post-discharge planning, 66 posttraumatic amnesia, 65 complex ideation material test, 68 disability rating scale, 68, 69 FIM score, 69 modified token test, 68 recommendations, 68 trail making test, 69 visual number search test, 68 Wechsler test, 69

Cognitive outcomes mild TBI complicated mTBI, 28 post-concussion syndrome (PCS), 26 repeated mTBI, 27 moderate-severe TBI, 28-30 Coma recovery scale-revised (CRS-R) administration and scoring AFP. 52 arousal subscale, 53 auditory subscale, 52 communication subscale, 53 motor subscale, 53 oromotor/verbal subscale, 53 progress tracking chart, 53, 54 Record Form, 53 visual subscale, 52-53 clinical applications, 53 differential diagnosis, 56 outcome assessment, 56-57 reference standard, 57-58 TMS, 58 treatment monitoring, 57 25-point scale, 52 psychometric properties, 52, 55-56 Rasch analysis, 52 research applications, 53 total score, 54 Community Integration Questionnaire (CIQ), 102 Community participation CHART and CIO, 102, 104 definition. 102 Mayo-Portland Participation Index, 102-103, 105 Participation Assessment with Recombined Tools-Objective (PART-O), 102, 105 Participation Objective, Participation Subjective (POPS), 102, 105 Comprehensive brain injury rehabilitation (CBIR) Ben-Yishay/Prigatano model, 284 characteristics of, 285 holistic milieu-oriented treatment, 284 interdisciplinary evaluation, 285-286 interdisciplinary team, 286 monitoring/measuring outcomes GAS, 300-301 MPAI-4, 301 multidisciplinary team, 286 team evaluation, 302-303 transdisciplinary team, 286 briefing, 286 celebration, 287 debriefing, 286 practice, 286 treatment (see Transdisciplinary treatment) treatment plan outline, 302-304 Comprehensive TBI Evaluation, 394 Concord Informant Dementia Scale (CIDS), 331 Confusion assessment protocol (CAP) administration and scoring, 59 cognitive impairment, 62

confused vs. non-confused patients, 60 degree of cooperation, 62 disordered consciousness, 58 fluctuation and cognitive impairment, 61 post-injury productivity outcome, 61 PTCS, 58-59 recommendations, 60 rehabilitation discharge, 61 scoring rules, 60 Spearman's coefficient, 62 Control and Prevention (CDC) survey, 331 Core metrics cognitive-linguistic function, 144 medical complications checklist (MCC), 145 motor function, 144 neurobehavioral status, 143-144 Craig Handicap Assessment and Reporting Technique (CHART), 102 CRS-R. See Coma recovery scale-revised (CRS-R) Cumulative Illness Rating Scale (CIRS), 333

#### D

Depression Anxiety Stress Scales, 262 Diffuse axonal injury (DAI), 27, 235 Disability rating scale (DRS), 38, 97, 144 Disorders of consciousness (DoC) Care Map, 142, 143 coma, 140 DoC compass arousal monitoring protocol, 145, 146 core metrics (see Core metrics) CRS-R profile, 150 eProfile, 145, 151-155 family education and support, 145-146, 149 global functional status, 150 minimally conscious state, 140, 141 posttraumatic confusional state, 140-141 reflexive behaviors, 141 SRN DoC Program, 141–142 sustained attention training protocol, 145, 148 trial and error approach, 141 vegetative state, 140, 141 Yes-No response consistency protocols, 145, 147 Dorsolateral frontal cortices (DRC), 194-195 DRS. See Disability rating scale

#### Е

Early childhood intervention (ECI) services, 313–314 Emotional distress anxiety, 260–261 cognitive-behavioral interventions, 263 cognitive rehabilitation techniques, 264 depression cognition, 259–260 depressive symptoms, 259 post-TBI factors, 258–259 prevalence of, 257–258 severity, 258

psychological treatment, 264 psychotherapeutic interventions, 264 self-report measures, 262-263 suicide, 261-262 Epidemiology classification, injury severity, 5-7 definition, TBI, 4 incidence rates, TBI, 7-8 military TBI blast related injury, 14 CONUS, 15 inpatient hospitalization rate, 15 PTSD, 15, 16 screening data, 13 prevalence and societal impact, 16-17 prevention, 17 risk factors, 8-10 sports concussion collegiate sports, 12 grading system, 11 high school level, 11, 12 monitoring and detection, 10 professional sports, 12-13 rates, 12 women's sports, 12 Epidural hematoma (EDH), 6, 12, 127 Extended Glasgow Outcome Scale (GOSE), 38

# F

Family caregivers emotional distress, 272 financial stress, 272 injury-related problems, 280-281 marital relationship, 272 needs of, 273 neuropsychological services, 273 neuropsychologists' roles inpatient/acute trauma setting, 274-275 inpatient rehabilitation setting, 275-276 post-acute rehabilitation setting, 276-279 private practice setting, 279-280 FIM. See Functional independence measure Fluid attenuated inversion recovery sequence (FLAIR), 115 Functional assessment measure (FAM), 97 Functional independence measure (FIM), 38, 100, 102 - 103

#### G

GAD. See Generalized anxiety disorder (GAD)
Galveston Orientation and Amnesia Test (GOAT), 59, 153
Generalized anxiety disorder (GAD), 34, 70, 260
Glasgow Coma Scale (GCS), 4–6, 26, 28, 31, 36, 37, 258
Glasgow Outcome Scale (GOS), 100, 101
Goal attainment scaling (GAS), 300–301
Goal management training (GMT), 294
GOSE. See Extended Glasgow outcome scale

#### H

Hospital Anxiety and Depression Scale, 262

#### I

Impaired self-awareness (ISA) clinical practice self-perception, 249-251 treatment compliance, 247-249 clinical rating method Awareness Interview, 241 borderline awareness, 240 deficits interview, 240 Denial of Disability (DD), 241 functional implications, 240 intraclass correlation coefficient (ICC), 241 pre-injury goals, 240-241 SADI, 240, 241 SRSI, 240, 241 community integration programs, 245 diffuse axonal injury, 235 discrepancy method Awareness Questionnaire (AQ), 238-239 informant's ratings, 237 Mayo-Portland Adaptability Index, 237 patient's self-ratings, 237, 239 PCRS, 237 research, 239 Sickness Impact Profile, 238 early recovery, 235 education, 246-247 feedback approaches, 242-243 functional outcome, 236 group programs, 246 incidence, 234 metacognitive strategy training, 242 neurobehavioral deficits, 235 occupation-based interventions, 244-245 on-line awareness, 242 predicted performance, 243-244 psychotherapy, 246 rehabilitation process, 236 social cognition, 235-236 Improving Pragmatic Skills in Persons with Head Injury, 221 Individuals with Disabilities Education Act (IDEA), 314 Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE), 331 Instrumental activities of daily living (IADL), 86 International classification functioning (ICF) model, 81-82 Interpersonal process recall (IPR) method, 217-218, 225

#### L

Loss of consciousness (LOC), 26, 28, 34

#### М

Mayo-Portland Adaptability Inventory-4 (MPAI-4), 102, 301 Mayo-Portland Participation Index (M2PI), 102–103, 105 Memory problems clinical manifestation, 175 compensatory memory strategy description of, 179, 180 follow-up support, 181 in-depth information, 179 internal strategy, 176 paper-based aids and electronic aids, 176-177 pill box, 180 simulated and real-world practice, 181 verbal reinforcement and encouragement, 181 memory strategy training caregiver, 177-178 clinical assessment, 181-182 ecologically valid tasks, 177 follow-up support, 184–185, 188 identify potential obstacles, 178 information gathering, 182-183, 185-186 strategy and assignment of practice, 184, 187-188 strategy development, 183-184, 186-187 restorative treatments, 175-176 types, 174-175 Metacognitive training intervention clinical application anticipate and plan, 203 awareness, 202-203 execute and self-monitor, 203-204 self-evaluation process, 204 multifaceted approach, 200 patient history, 205-207 problem-solving deficits, 204 Stuss' theoretical framework, 201 Mild cognitive impairment (MCI), 331 Mild traumatic brain injury (mTBI), 4-8, 11 blast related injury, 14 clinical practice chronic post-deployment setting, 402-404 chronologically remote mild TBI, 401-402 patient expectations, 400-401 persistent vs. current symptoms, 402 providing accurate information, 398, 400 cognitive difficulties, 404-407 cognitive impairment complicated mTBI, 28 repeated mTBI, 27 cognitive outcomes complicated mTBI, 28 post-concussion syndrome (PCS), 26 repeated mTBI, 27 common comorbidities, 391 CONUS, 15 definition, 348-349, 390-391 inpatient hospitalization rate, 15 interventions, 357-358 loss of consciousness (LOC), 26 malingering (see Performance validity tests (PVTs)) military deployment and combat environment, 396 - 397military-relevant settings, 398, 399 military setting, 361-362, 364-365

natural recovery, 351, 352

neuropsychological assessment clinical interview, 353 record review. 352 test battery (see Neuropsychological test battery) pathophysiology, 351 PCS, 395-396 personal injury, 360-361, 363-364 political/social context, 397-398 post-concussion syndrome (PCS), 26-27 PTSD, 15, 16 recovery course, 394-395 screening data, 13 self-report and time since injury, 397 sports setting, 359-360, 363 teasing apart comorbidities, 391-392 VHA and DoD systems (see Veterans Health Administration (VHA) system) Mississippi aphasia screening test (MAST) administration and scoring, 63-64 comprehensive battery, 63 language impairment, 63 memory impairment, 63 patients with stroke, 65 recommendations, 64-65 structured testing, 63 yes/no accuracy, 65, 66 Moderate-severe TBI cognitive deficits, 29, 30 communication problems, 29 independent living, 32 life satisfaction, 32-33 neurobehavioral symptoms, 29 productivity outcomes, 30-32 PTA, 29 PTC, 29 social functioning and community integration, 32 vegetative and minimally conscious states, 28-29 mTBI. See Mild traumatic brain injury (MTBI)

Multiphasic Personality Inventory (MMPI-2), 357

#### Ν

Neurobehavioral Functioning Inventory-Depression Scale, 262 Neurobehavioral Symptom Inventory (NSI), 394 Neurodegenerative disease, 27 Neuroimaging techniques CCHR. 112 CHALICE/PECARN rule, 113 computed tomography attenuation coefficient, 115 axial/transverse plane, 114 gross anatomical information, 113-114 Marshall classification, 126 vs. MRI, 114 Rotterdam CT classification, 126 sophisticated computer algorithms, 115 consciousness/posttraumatic amnesia, 113 future aspects, 132 limitations, 130-131 magnetic resonance imaging

balanced/proton density sequences, 115 vs. CT. 114 diffusion tensor imaging, 129 fluid attenuated inversion recovery sequence, 115 hemosiderin, 115 magnetic resonance spectroscopy, 129 multimodal imaging, 130 resting state fMRI, 130 signal intensity, 115 susceptibility-weighted imaging, 115 typical tissues appearance, 115, 116 visualizing brain anatomy, 114, 115 New Orleans criteria guidelines, 112 qualitative imaging analysis age-matched healthy control, 116, 124 frontal lobe, 124, 125 symmetric and similar appearance, 114, 124 time frame post-injury, 124 ventriculomegaly, 124 quantitative image analysis hemosiderin-identified lesions, 129 pixel, 128-129 **SDH/EDH. 127** ventricle-to-brain ratio, 127 volumetric analysis, 127, 128 standardization facilitates research, 132 traumatic pathology, 116-123 Neuropsychological assessment acute inpatient assessments, 78 car crash injury, 90-91 feedback session family and caregivers, 88 injury, 89 patients, 88 inpatient neuropsychological evaluations, 79 mood symptoms, 79-80 neuropsychological testing behavioral observations, 87 cognitive functioning, 87-88 objective measures, 87 symptom validity, 87 outpatient brain injury rehabilitation settings, 88-89 post-acute settings ICF model. 81-82 inpatient rehabilitation, 80 outpatient rehabilitation (see Outpatient assessment) posttraumatic amnesia (PTA), 78 Neuropsychological test battery chronic effects, 353 cognitive impairment, 353 forensic/disability setting, 353 formal assessment, 354 language and academic skills, 355 MMPI-2, 357 NYU Langone Medical Center, 354 Post-Concussion Scale (PCS-R), 356 Test of Memory Malingering (TOMM), 356 verbal list-learning measures, 355 WMS-IV Logical Memory subtest, 355 Word Memory Test (WMT), 356

#### 0

Obsessive compulsive disorder (OCD), 35 Older adults patient CDC survey, 331 cognitive stimulation., 340 diagnostic issues Alzheimer's disease, 336 depression, 337-338 Glasgow Coma Scale (GCS) scores, 335, 336 neurobehavioral recovery, 334-335 neurosurgical complications, 336-337 preinjury cognitive impairment, 335 encourage physical exercise, 340 external memory aids, 340 neurobehavioral outcome dementia, 331 medical comorbidities, 332-333 medication, 334 mild cognitive impairment (MCI), 331 prevent future falls, 339 reduce polypharmacy, 339 treat depression, 339-340 treat medical comorbidities, 339 Outcome assessment disability rating scale (DRS), 97 FAM/FIM, 97 impairments, body structures and function, 97 limitations and restrictions, 97 outcome measure selection, 98, 99 community participation (see Community participation) disability and activity measures, 100, 102–103 environmental factors, 106, 107 global outcome measures, 100, 101 patient characteristics/environmental variables, 97 prevalence of, 98 PROMIS methodology, 107-108 Outpatient assessment awareness and psychosocial functioning, 84, 85 clinical interview, 82 compensatory strategy, 86-87 components of, 82, 83 daily activity imitations, 86 premorbid and current functioning, 84, 85 psychiatric and substance abuse history, 83-84

#### P

Parkinson's disease, 27 Participation Assessment with Recombined Tools-Objective (PART-O), 102, 105 Patient Competency Rating Scale (PCRS), 237 Patient Health Questionnaire (PHQ-9), 70, 262, 338 Patient Reported Outcomes Measures Information System (PROMIS), 107 PCS. See Post-concussion syndrome (PCS) Pediatric emergency care applied research network (PECARN) rule, 113 Pediatric traumatic brain injury CDE, 312 epidemiological studies, 311 family environment, 323 infancy and early childhood academic deficits, 313 acquisition, 313 assessment of, 312 brain reserve, 312 cognitive and motor abilities, 312 cognitive reserve, 312 early childhood intervention (ECI) services, 313-314 falls and bicycle/pedestrian-motor vehicle collisions, 311 inflicted brain injury, 314-316 neuropsychological outcomes, 312 parenting style and family functioning, 313 reading tasks, 313 vocational outcomes, 313 re-entry to school ADHD, 320 audiology services, 319 BIP. 319 educational accommodations, 319 IDEA, 318 IEP. 319 incidence of, 318 medical and educational systems, 318 PBIS. 319 physical fatigue and confusion, 318 speech-language pathology, 319 school-age and adolescence academic outcome studies, 316 assessment of, 317-318 family functioning, 317 intervention, 313-314 metacognitive processes, 317 motor vehicle collision, 311, 321-323 pre-injury intellectual/learning disabilities, 317 premorbid academic ability, 317 recovery for, 323-324 social development and adaptive functioning, 317 verbal and visual learning, 316 Performance validity tests (PVTs) cognitive symptom feigning, 372 feigned cognitive performance, 382-384 neurocognitive functioning BDI-II, 374 cognitive abnormalities, 372, 373 comorbid psychiatric disorders, 374 compensation-seeking status, 373 diagnosis threat, 373 long-term cognitive deficits, 373 noninjury factors, 374 postconcussive symptoms, 373, 374

pre-existing psychiatric problems, 373 self-report, 372, 373 sexual abuse history, 374 TOMM, 374 noncredible cognitive symptoms embedded vs. free-standing, 375-376 feigned language impairment, 377-378 feigned memory/attention impairment, 377 feigned processing speed and sensory impairment, 378 forced choice vs. nonforced choice, 376 MCMI-III, 381 MMPI-2-RF, 380-381 motor vehicle accident, 378-380 statistical terminology, 374-375 Polytrauma Network sites (PNSs), 392 Polytrauma rehabilitation centers (PRCs), 392 Polytrauma telehealth network, 393 Positive behavior intervention and supports (PBIS), 319 Post-Concussion Scale (PCS-R), 356 Post-concussive syndrome (PCS), 26, 27, 395-396 Post-Deployment Health Assessment and Reassessment (PDHRA), 393 Posttraumatic amnesia (PTA), 4-6, 28 Posttraumatic confusion (PTC), 28 Posttraumatic confusional state (PTCS), 58-59 Post-traumatic stress disorder (PTSD), 15, 16, 34-35, 260-261.391 Pseudo-abnormalities, 39 Psychological sequelae anxiety GAD, 34 OCD, 35 panic disorder, 35 PTSD, 34-35 mood disorders bipolar disorder, 34 depression, 33-34 personality disorders, 35 schizophrenia and psychosis, 35 substance abuse disorders, 36 PTA. See Posttraumatic amnesia PTSD. See Posttraumatic stress disorder PVTs. See Performance validity tests (PVTs)

#### R

Resting state fMRI (rs-fMRI), 130

#### S

Self-Awareness of Deficits Interview (SADI), 240
Self-Regulation Skills Interview (SRSI), 240
Social communication interventions acquired brain injury (ABI), 216
ANOVA, 219
behavioral rating scales, 224
cognitive and behavioral changes, 214

cohort study, 221 communication partners, 223-224 conversational behaviors, 218 employment, 215-216 family burden, 216 feedback, 218 frontal contusion, 224 GAS approach, 227 goals for, 225 impaired social perception, 215 information-processing model, 214 interpersonal process recall method, 217-218, 225 job coach, 225 marital relationships, 216 problematic communication behaviors, 226 randomized clinical trials, 219-221 receptive communication skills, 221-223 recommendations, 228 research, 227-228 self-monitoring conditions, 218 skills training program, 219 social integration, 215 ventral frontal lobe injury, 215 verbal and nonverbal skills, 214 verbal learning and memory, 224 verbal praise, 219 Spaulding Rehabilitation Network (SRN) Disorders of Consciousness Program, 141-142 Sports concussion collegiate sports, 12 grading system, 11 high school level, 11, 12 monitoring and detection, 10 professional sports, 12-13 rates, 12 women's sports, 12 Subdural/epidural hematoma, 5, 6, 12 Subdural hematoma (SDH), 127 Substance abuse disorders, 36 Suicidal ideation (SI), 261

#### Т

Transcranial magnetic stimulation (TMS), 58 Transdisciplinary treatment accessibility modifications, 298 behavior management training, 294-295 CBIR, 294-295 cognitive rehabilitation APT. 293 evidence-based methods, 293 functional impact, 292 GMT. 294 memory notebook, 293 memory problems, 292 systematic problem-solving, 293-294 co-morbidities and prevention, 299-300 environmental assists, 298 group therapy, 288-289

Transdisciplinary treatment (*cont.*) impaired self-awareness disability, 290 education, 290–291 individual and group psychotherapy, 292 peer feedback, 292 pre-injury personality tendency, 290 structured repeated learning experiences, 291–292 involvement and intervention, 297–298 psychoactive mediations, 298–299 resource facilitation, 295, 296 social communication skills training, 294 therapeutic alliance, 289–290 therapeutic milieu, 288 vocational interventions, 295, 296

#### work and independent living trials, 296-297

#### V

Veterans Health Administration (VHA) system and DoD screening, 393–394 follow-up evaluation and treatment, 394 polytrauma/TBI system of care, 392–393 Voxel-based morphometry (VBM), 127–129

# W

Wide Range Assessment of Memory and Learning (WRAML), 321

Wisconsin Card Sorting Test (WCST),

259–260, 355

Woodcock-Johnson Tests of Achievement (WJ-III), 316