

# TRAUMATIC BRAIN INJURY

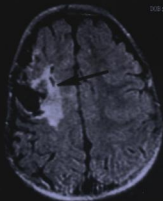
Methods for Clinical and Forensic  
Neuropsychiatric Assessment

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ROBERT P. GRANACHER

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# TRAUMATIC BRAIN INJURY

## Methods for Clinical and Forensic Neuropsychiatric Assessment

Numerous books exist on traumatic brain injury, yet none comprehensively cover evaluation from both clinical and forensic standpoints. *Traumatic Brain Injury: Methods for Clinical and Forensic Neuropsychiatric Assessment* is the first medical book to guide treatment practitioners not only in methods for evaluating traumatic brain injury in adults and children, but also in the important elements of forensic brain injury assessment.

From the clinical aspect, the book details neurobehavioral data analysis and describes how to apply it to treatment planning and pharmacotherapy following traumatic brain injury. From the forensic perspective, it provides methods for detecting deception at examination and emphasizes the important legal concepts of causation, damages, and impairment determination following traumatic brain injury.

The text provides multiple explanatory tables, structural and functional brain imaging figures, and liberal case examples of actual traumatic brain injury examinations and reports. The practical and pragmatic approach offered in *Traumatic Brain Injury: Methods for Clinical and Forensic Neuropsychiatric Assessment* will aid clinicians and forensic specialists in comprehensively evaluating the TBI patient and successfully presenting the evaluation in the courtroom.

### FEATURES

- Provides a step-by-step approach to neuropsychiatric evaluation of traumatic brain injury
- Presents the only comprehensive evaluation process for assessing a TBI patient
- Covers the differences between adults and children
- Provides measurement techniques for determining cognitive and behavioral deficits
- Assists the forensic practitioner in the preparation of a TBI case for trial
- Translates complex brain injury concepts into understandable language for a jury

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Neuropsychiatric Assessment

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ROBERT P. GRANACHER



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# Preface

Approximately 2 million traumatic head injuries occur in the U.S. yearly. These in turn produce more than 50,000 deaths annually. There is a biphasic distribution of brain injury, with the highest incidence found among young people 15 to 24 years of age and a second group of citizens greater than 75 years of age. Almost 25% of head injuries require hospitalization, and nearly 100,000 persons yearly are left with some level of chronic brain impairment.

This text has a specific focus. It provides not only methods for clinical examination but also the forensic evaluation of traumatically brain-injured persons. The reader can be selective in using this book. If he or she is interested only in clinical assessment, treatment planning, and neuropsychiatric treatment, the first eight chapters of the book will suffice. On the other hand, for the physician performing a forensic neuropsychiatric examination, the entire book should be useful. If the clinician is already highly skilled in the clinical evaluation of traumatic brain injury but wishes to learn further forensic issues, he or she may focus only on the last four chapters of this text.

There is a simple logic to the book. It follows traditional medical evaluation concepts with a neuropsychiatric focus. It demarcates differences in the adult evaluation vs. the child evaluation. [Chapter 8](#) integrates the clinical section of this text, whereas [Chapter 11](#) integrates the forensic section of the text. The seven preceding chapters in the clinical section of the book proceed logically to a culmination of data analysis and case studies in [Chapter 8](#). The same format applies to the forensic section, [Chapters 9 to 12](#). [Chapters 9 to 11](#) provide the forensic analysis database, and [Chapter 12](#) offers the forensic expert guidance for the writing of neuropsychiatric traumatic brain injury reports and the providing of neuropsychiatric testimony.

This text is not intended to provide complete information regarding the multiple advances within the entire field of traumatic brain injury. For instance, it provides only a limited focus on management of acute traumatic brain injury. This is better left to neurosurgeons and trauma physicians. Its primary intent is to provide the physician, at some time well after the brain injury, with a clinically tested schema for either evaluating and treating a patient or examining a plaintiff or defendant. The genesis for this text comes from the author's database of almost 3000 traumatically brain-injured persons, or those alleging a traumatic brain injury, examined by extensive historical, physical, imaging, neuropsychological, and laboratory procedures. It is hoped that the reader will find this to be a practical text providing pragmatic information either for evaluation and treatment of one's patient or for providing a state-of-the-art forensic examination of an alleged traumatic brain injury.

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# Author

**Robert P. Granacher Jr., M.D., D.F.A.P.A.,** is president and executive director of the Lexington Forensic Institute in Lexington, Kentucky. For more than 25 years he has taught at the University of Kentucky College of Medicine within the Department of Psychiatry, and he currently functions as clinical professor of psychiatry in that division. He has a full-time private practice as a treating psychiatrist and as a forensic psychiatrist. He is board-certified by the American Board of Psychiatry and Neurology in general psychiatry, with added qualifications in geriatric psychiatry and forensic psychiatry. He is also board-certified in forensic psychiatry by the American Board of Forensic Psychiatry, Inc. He is a diplomate and board-certified in sleep medicine by the American Board of Sleep Medicine, and he is certified in psychopharmacology by the American Society of Clinical Psychopharmacology. He serves on the board of directors of St. Joseph Hospital, a 630-bed medical–surgical tertiary care facility, and from 1999 to 2002, he served as chairman of the board. He is a director of C.B.A. Pharma, an oncology and research pharmaceutical company.

Dr. Granacher received his bachelor's degree in chemistry from the University of Louisville and his doctor of medicine degree from the University of Kentucky, Lexington. He served as resident and chief resident in psychiatric medicine at the University of Kentucky Hospital, and later as resident and fellow at the Harvard Medical School and the Massachusetts General Hospital, Boston. He has specialized in the areas of treatment and evaluation of traumatic brain injury, perinatal birth injury, and toxic brain injury for more than 25 years. He has personally evaluated by complex neuropsychiatric assessment almost 3000 traumatic brain injury cases.

Dr. Granacher is a member of numerous professional associations, including the American Medical Association, American Psychiatric Association, American Neuropsychiatric Association, American Academy of Sleep Medicine, American Society of Clinical Psychopharmacology, Kentucky Medical Association, Kentucky Psychiatric Association, and Fayette County (Kentucky) Medical Society. He received the Exemplary Psychiatrist Award from the National Alliance for the Mentally Ill in 1996. He is a distinguished fellow of the American Psychiatric Association.

This text is Dr. Granacher's second book. He and Aaron Mason, M.D. previously published *The Clinical Handbook of Antipsychotic Drug Therapy*. In addition to books, Dr. Granacher has published widely in the medical literature, including book chapters and scientific articles. He currently serves on two important committees of the American Academy of Psychiatry and the Law: the Private Practice Committee and the Forensic Neuropsychiatry Committee. He also serves on the Core Forensic Committee of the American Society of Clinical Psychopharmacology. His forensic psychiatry practice is national in scope, and he consults to plaintiff lawyers, defense lawyers, courts, the U.S. Department of Justice, corporations, and school systems.

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# 1 The Epidemiology and Pathophysiology of Traumatic Brain Injury

## INTRODUCTION

It has been questioned whether traumatic brain injury forms a model of acquired psychiatric illness.<sup>1</sup> Neurosurgical care in the U.S. has markedly progressed in the last 25 years. The good news from this progress is that the survival rate of traumatically brain-injured persons has increased dramatically. The bad news is that improved survival rates have led to a dramatic increase in the number of cognitively and behaviorally impaired persons with long-term neuropsychiatric disorders as a consequence of traumatic brain injury. Roughly 2 million cases of head trauma occur in the U.S. each year.<sup>2</sup> Traumatic brain injury results principally from vehicular accidents, falls, acts of violence, and sports injuries and is twice as likely to occur in men than women. The estimated incidence is 100 per 100,000 persons, with 52,000 annual deaths. The highest incidence is among persons aged 15 to 24 years and those older than 75 years, with a less striking peak at age 5 years. Prevalence is 2.5 to 6.5 million persons.<sup>11</sup> Of these injuries, almost 25% require hospitalization and 80,000 to 90,000 persons are left with some level of chronic disability.

In terms of neuropathology of head injuries, structural and functional abnormalities develop progressively after brain trauma, which suggests that the resulting brain injury is a dynamic process of events rather than a single event. Numerous types of neuropathologies can occur in the same brain, in the same individual, from the same injury. Neuropathological damage can occur by direct damage caused by excitotoxic-mediated calcium influx into cells, free radical-mediated damage, receptor-mediated damage, and inflammatory processes.<sup>3</sup> Furthermore, the direct consequences of trauma may be complicated by secondary injuries occurring after head trauma. These include intracranial hypertension, vascular failure, ischemia, endogenous brain defenses, axonal injury, and neuronal injury.<sup>5</sup>

Head injury classification has no universally accepted system. Many classification schemes have been proposed. All existing classification systems have limitations.<sup>6</sup> The types of brain trauma are straightforward, and these include damage from skull fractures. Focal brain damage is a result of contusions, hemorrhage, hematoma, or tissue tears. Diffuse brain damage may be the result of diffuse axonal injury, ischemic injury, or the complications of brain edema.<sup>7</sup> Lastly, there is the apparent relationship between traumatic brain injury and late-appearing neurodegeneration of the Alzheimer's type.<sup>8</sup>

This chapter focuses on the key concepts of the epidemiology of brain injury and the pathophysiology of traumatic brain injury. Various classification systems for categorizing the severity of brain injury are expressed. The serious neurosurgical consequences of acute brain injury are demonstrated, and their relationships to cellular and neuronal injury are exemplified. The current apparent relationship between traumatic brain injury and the later expression of Alzheimer's-like neurodegeneration is explored.

## EPIDEMIOLOGY OF TRAUMATIC BRAIN INJURY

The stated estimates of the occurrence of brain injury appear to be a moving target in the U.S. There is tremendous variability in reported incidence rates of traumatic brain injury depending upon the population studied, the age and sex of the individuals, the race or ethnicity of the victims, and the socioeconomic status of the injured persons.<sup>9</sup> The death rate is somewhat dramatic. Brain injury is the leading cause of death for persons under 45 years of age in the U.S. In 1990, approximately 140,000 persons died of acute traumatic injury, accounting for about 8% of all deaths in the U.S. Approximately 50% of these deaths were due to brain trauma. The National Health Interview Survey for 1985 to 1987 was extrapolated to the 1990 U.S. Census population of about 249 million residents. This survey reported that about 1,975,000 head injuries occur per year in the U.S.<sup>10</sup> Recent epidemiological reports cite approximately 2 million head injuries each year in the U.S. that produce a brain injury rate of 175 to 200 per 100,000 population and cause as many as 56,000 deaths per year<sup>11</sup> (see [Table 1.1](#)). The more recent National Institutes of Health (NIH) Consensus Development Panel on Rehabilitation of Persons with Traumatic Brain Injury noted that traumatic brain injury is of major public health concern.<sup>12</sup>

When the incidence of traumatic brain injury is examined regionally in the U.S., many variations are seen. In the Commonwealth of Virginia, persons aged 40 years and younger represented almost 80% of all head injuries presenting to Virginia emergency rooms for 1988 to 1993. Age-adjusted incidence rates were greatest for children under 6 years (237 of 100,000 person-years) and least for persons 40 to 69 years (56 of 100,000 person-years). Head injuries occurred 1.4 times more frequently in males than females, and the male mortality rates were 1.6 times greater than the female rates. Falls exceeded motor vehicle accidents as the most common cause of head trauma after fiscal year 1989, followed by assaults and sports- and recreation-related injuries.<sup>13</sup> In Colorado, cases of traumatic brain injury were surveyed from 1991 to 1992. Traumatic brain injury age-adjusted rates varied significantly from 98 per 100,000 population for the most urban group to 172 per 100,000 population for residents of rural or remote counties. Total mortality ranged from 18 per 100,000 population among urban residents to 34 per 100,000 population among rural residents.<sup>14</sup> In Alaska, from 1996 through 1998, the average incidence of brain injury was 105 per 100,000 population.<sup>15</sup>

When one examines international or worldwide rates of brain injury, in most developed nations, the brain injury rates are comparable to those of the U.S. For instance, in a university hospital in Norway during 1993, the annual incidence of hospital-referred head injury was 229 per 100,000 population, with a male preponderance of 1.7:1.0.<sup>16</sup> In south Australia, a higher than expected incidence of traumatic brain injury was discovered. The rate of 322 brain injuries per 100,000 population annually exceeded the average rates reported for the U.S. and Europe. The elevated rates were seen mostly in young males living in the country working in manual trades.<sup>17</sup> Estimated incidence rates in France have been reported recently to be between 150 and 300 per 100,000 inhabitants. The annual incidence of severe head injury was estimated to be approximately 25 per 100,000 inhabitants for cerebral trauma, with intracranial injuries around 9 per 100,000 for the most severe level of head injury with coma.<sup>18</sup>

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**TABLE 1.1**  
**Epidemiology of Traumatic Brain Injury**

- 2 million per year in U.S.
  - 175–200 per 100,000 population
  - 50,000–55,000 deaths per year
  - Rates comparable in industrialized countries
-

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**TABLE 1.2**  
**Glasgow Coma Scale (GCS)**

Eye opening (E):	
Spontaneous	4
To voice	3*
To pain	2
No response	1
Verbal response (V):	
Oriented conversation	5
Confused, disoriented	4
Inappropriate words	3*
Incomprehensible sounds	2
No response	1
Best motor response (M):	
Obeys commands	6
Localizes	5
Withdraws (flexion)	4*
Abnormal flexion (posturing)	3
Extension (posturing)	2
No response	1

Note: GCS = 10: E = 3, V = 3, and M = 4 (as marked by the asterisks).

Reprinted with permission from Elsevier Science, Teasdale, G. and Jennett, B., *The Lancet*, 1, 81, 1974.

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## CLASSIFICATION OF HEAD INJURY

Multiple classifications of head injury are available to the reader: classification by level of severity, level of consciousness, mental status following head injury, or location of body injury.<sup>6</sup> The *Abbreviated Injury Scale* is primarily an anatomical system, but it also scores for severity and is based on the relative seriousness of the lesion and its effect upon mental state.<sup>19</sup> A seven-digit code number is assigned that reflects the location of the lesion and its size and severity. The final digit of this code is related to severity and is scored on a scale of 1 to 6. The *Glasgow Coma Scale* (GCS) was introduced to modern medicine by Teasdale and Jennett.<sup>20</sup> This system is the most widely used scoring procedure for mental and neurological status following head injury in the U.S. and most English-speaking countries. Its score is based on the sum of three components: eye opening, verbal response, and best motor response. For instance, if an individual at the accident scene opened eyes to voice, used inappropriate words, and demonstrated a flexion response to motor stimulation, the scoring would be  $E + V + M = 3 + 3 + 4 = 10$  (see [Table 1.2](#)). This in turn produces a graded score in the moderate severity range. The GCS can be further subdivided into mild injury (GCS = 13 to 15), moderate injury (GCS = 9 to 12), and severe injury (GCS = 3 to 8). The clinical features of mild injury are loss of consciousness for 20 min, no focal neurological signs, no intracranial mass lesion, and no intracranial surgery. Regardless of mental state, a focal CT lesion places the patient into the moderate category. A coma duration of at least 6 h places the patient into the severe category, regardless of mental state.

In terms of outcome, the most commonly used current scales are the Glasgow Outcome Scale<sup>21</sup> ([Table 1.3](#)) and the Rancho Los Amigos Level of Cognitive Functioning Scale<sup>23,156</sup> ([Table 1.4](#)). The Rancho Scale is widely used by rehabilitation facilities after the patient leaves the neurosurgical intensive care unit or neurosurgical floor for postacute care. Generally, a final grading using the

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**TABLE 1.3**  
**Glasgow Outcome Scale**

Categories	Clinical Features
Death	
Vegetative state	Absence of cognitive function with total abolition of communication
Severe disability	Conscious but dependent patient
Moderate disability	Independent but disabled
Good recovery	Independent patient who may return to work or premorbid activity; mild cognitive or neurological deficits may persist

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**TABLE 1.4**  
**Rancho Los Amigos Level of Cognitive Functioning**

Levels	Clinical Signs
I. No response	Unresponsive to any stimulus
II. Generalized response	Nonpurposeful responses, usually to pain only
III. Localized responses	Purposeful; may follow simple commands
IV. Confused, agitated	Confused, disoriented, aggressive; unable to perform self-care
V. Confused, inappropriate	Nonagitated; appears alert; responds to commands; verbally inappropriate; does not learn
VI. Confused, appropriate	Can relearn old skills; serious memory defects; some awareness of self and others
VII. Automatic, appropriate	Oriented; robot-like in daily activities; minimal confusion; lacks insight or planning ability
VIII. Purposeful, appropriate	Alert and oriented; independent in living skills; capable of driving; defects may remain in judgment, stress tolerance and abstract reasoning may not be at preinjury cognitive ability

Used with permission from the Rancho Los Amigos National Rehabilitation Center, Downey, California.

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Rancho Scale is made prior to the patient's discharge from a brain injury rehabilitation unit if such is required.<sup>156</sup>

## NEUROPATHOLOGY OF TRAUMATIC BRAIN INJURY

### BIOMECHANIC MECHANISMS IN TRAUMATIC BRAIN INJURY

The two major kinds of mechanical loading to the head that produce brain injury are static loading and dynamic loading.<sup>3,4</sup> Static loading occurs when forces are applied gradually to the head, such as in a squeezing injury due to compression by a large object, a head injury sustained in an earthquake or landslide, or a head injury sustained by a person at work under an automobile that falls from the jacks, crushing the head. The head is squeezed slowly, and usually the compression requires more than 200 msec to develop. The most common mechanical loading to the head seen by psychiatrists following head injury is dynamic. In this case, the forces acting on the head require less than 20 msec to develop. Thus, the duration of the mechanical load is a critical factor in determining the type of brain injury (e.g., motor vehicle trauma). Skull fracture<sup>22</sup> depends on whether the skull strikes a hard, unyielding surface or a soft, yielding surface. For a hard surface, like a steel plate, it takes approximately 33.3 to 75 ft-lb of energy to produce a linear fracture. This energy is absorbed in 0.0012 sec. The first 0.0006 sec deforms and compresses the scalp tissue, while the residual 0.0006 sec deforms the bone. Only a slight increase in energy is required to produce a stellate fracture or multiple linear fractures. A free fall backward from 6 ft for a head

weighing 10 lb gives an available energy of 60 ft-lb. The velocity of the head is approximately 20 ft/sec or 13.5 mi/h at impact.

Dynamic loading is further subdivided into two types: impulsive and impact. Impulsive loading is uncommon and occurs when the head is set in motion and then the moving head is stopped abruptly without it being either directly struck or impacted. This could occur, for instance, in a person violently struck in the thorax or the face, which sets the head violently in motion.<sup>4,23</sup> On the other hand, impact is the most common cause of injury to the brain, such as seen following a moving head within an automobile accident striking the support pillar or windshield.<sup>24</sup>

In acceleration–deceleration injury, there are two major types of acceleration: translational acceleration and rotational acceleration. In translational acceleration, all particles within the brain move simultaneously in the same direction. In other words, this is a linear acceleration, and most head injuries wherein injury occurs are distal to the point of impact; the acceleration is translational. On the other hand, the brain is damaged frequently by what has been called diffuse axonal injury. This is more likely to be caused by rotational or angular acceleration producing shear injury due to the differential densities of the gray vs. white matter and the shearing that may occur at the interface of these two masses.<sup>25</sup> Coup injuries are more common when the head is accelerated. This causes contusions beneath the site of impact. Contrecoup injuries (across from the blow) are more common with head deceleration.<sup>4,25</sup> The frequently occurring contusions of the frontal and temporal poles are almost always contrecoup, regardless of the site of head impact. Thus, contrecoup lesions by definition may be those that are not under the point of impact.<sup>4,26</sup> Strain is the proximate cause of tissue injury, whether it is induced by inertia or contact. Three types of strain affect brain tissue: compression, tension, and shear.<sup>4,27</sup> Biological tissues are usually elastic and thus deform slowly rather than quickly. The three principal tissues affected in a closed-head injury are bone, blood vessels, and brain, and they vary considerably in their tolerances to deformation.<sup>3</sup> Brain is virtually incompressible *in vivo*, but it has a very low tolerance to tensile or shear strain. The latter two types of strain are the usual causes of brain damage, as compression injury is rare, and the same holds for vascular tissue injury as well (Table 1.5).

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**TABLE 1.5**  
**Biomechanical Mechanisms of Traumatic Brain Injury**

Mechanism	Features
Static loading	$\geq 200$ msec to develop <sup>3,4</sup>
Skull bending	
Skull volume change	
Dynamic loading	$\leq 20$ msec to develop; impulsive or impact <sup>3,4,24,25</sup>
Impact	
Impulsive	
Acceleration	
Translational	All particles move simultaneously in same direction, linear <sup>3,4,27</sup>
Rotational	Particles move angular to others; shear forces common; causes diffuse axonal injury <sup>3,4,27</sup>
Angular	
Coup lesions	Predominate if the head is accelerated <sup>4</sup>
Contrecoup lesions	Predominate if the head is decelerated <sup>4</sup>
Strain	Compression, tension or shear <sup>3,4,27</sup>
Skull fracture	Requires 33.3–75 ft-lb of energy; a 6-ft person with a 10-lb head falling backwards will produce available energy of 60 ft-lb, causing the head to strike at 13.5 mi/h <sup>22</sup>

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The internal structure of the skull dictates the most probable location of traumatic injuries of the brain in most cases of closed-head injury. The skull surfaces above the eyes are quite rough, and the most anterior frontal vault of the skull is limited in size. As the brain accelerates forward or rotates into the frontal areas of the skull, the infraorbital frontal lobes are often contused or impacted by the rough prominences above the orbits. At the same time, the sphenoid ridges of the skull provide a significant structural impediment to the temporal poles, which in turn produces an accordion-like compression of the temporal tips. The temporal lobes contain numerous structures, such as the amygdalae, hippocampi, and limbic structures, which may account for disturbances of memory, mood, or complex emotions due to the temporal lobe deformation, while frontal lobe injury may result in specific frontal lobe syndromes<sup>27</sup> (Chapter 2). Recent study has led to mathematical models that enable biomechanical engineers to study head injury mechanisms and the forces at play within the skull during trauma.<sup>28–33,163</sup>

## **PATHOPHYSIOLOGY OF TRAUMATIC BRAIN INJURY**

The initial events of brain trauma involve mechanical distortion of the brain within the head. Contemporary knowledge teaches us that primary mechanical disruption of axons and subsequent instantaneous cell death are not common initial events following traumatic brain injury.<sup>3</sup> The most probable initial cellular abnormality following traumatic brain injury is focal impairment of axonal transport. This may create a traumatic defect in the cell membrane that occurs as the lipid bilayer is transiently separated from inclusions within the membrane, such as receptors or ligand- or voltage-gated channels. There is a differential tensile strength of the lipid bilayer relative to the stiffer membranes found in receptors or ion channels. Axonal transport injury occurs fundamentally and produces diffuse axonal injury primarily in the subcortical white matter, and recent work suggests that this process takes several hours to complete.<sup>34–37</sup>

The initial injury to the brain produces a series of cellular events contributing to a neurochemical and neurometabolic cascade. Presence of alcohol may influence the primary injury.<sup>38,39</sup> The primary injury in turn produces excitotoxic neuronal damage.<sup>158</sup> This cascade is defined by the release of neurotransmitters resulting in massive ionic flux, which consequently produces an increase in glycolysis. The increase in glycolysis is followed by metabolic derangement.<sup>40</sup> This cascade is set off initially, at least in part, by focal disruption of axonal transport. The ionic influxes activate genes and oxygen radicals, and then cell membrane lipid peroxidation occurs very early after injury. In turn, free intracellular calcium is increased and phospholipases are activated, as are calpains. These further damage the membrane and cytoskeleton and block axoplasmic transport. This can result in delayed cell death or trigger apoptosis.<sup>41,160</sup> Excess quantities of glutamate in the extracellular space may lead to uncontrolled shifts of sodium, potassium, and calcium, which in turn disrupt ionic homeostasis. This may lead to severe cell swelling and subsequent cellular death.<sup>42</sup> Moreover, approximately 60 min following traumatic brain injury, there is a significantly increased level of oxidative stress in the brain. This may be reflected by the formation of free radicals, which causes oxidative damage to neurovascular structures.<sup>43,157</sup>

When focal axonal transport disruption occurs, it may produce a cellular microdefect that is open for only a relatively brief period of time. It is then closed either passively by a flow in the lipid bilayer or more actively by generation of lysolecithin, patching the membrane by fusion.<sup>44</sup> Other mechanisms have been proposed as well.<sup>45</sup> Intracellular calcium increases and tends to parallel the amount of energy delivered to the cell membrane. Changes in calcium-mediated cellular signaling may contribute to the pathology that is observed after traumatic brain injury. Calcium influx elevates intracellular free calcium with subsequent activation of degradative enzymes.<sup>46</sup>

Mitochondrial oxidative stress activates mechanisms that impose a significant burden to the antioxidant reserve and free radical scavenging systems.<sup>157</sup> This may result in a neutrophil-mediated inflammation that also causes secondary damage. Oxidative stress may also induce

**TABLE 1.6**  
**Pathophysiology of Traumatic Brain Injury**

Process	Features
Acetylcholine binding	Hippocampus displays decreased cholinergic binding <sup>149,150</sup>
Altered membrane potential	Glutamate drives hyperglycolysis; intracellular calcium increases, causing cell death <sup>36,37,40–43</sup>
Apoptosis	Programmed cellular death; immature brain is the most vulnerable <sup>69,70,160</sup>
Arachidonic acid cascade	Free radicals released from mitochondria <sup>62,159</sup>
Focal impairment of axonal transport	Occurs at about 60 min postinjury; axotomy apparent at about 6–12 h postinjury; thereafter, the proximal segment swells at about 24–72 h postinjury, while the distal segment undergoes rapid degeneration <sup>4</sup>
Oxidative stress	Induces genes <i>C-Fos</i> , <i>C-Jun</i> , <i>Jun B</i> ; alters regulation of nerve growth factor, amyloid precursor protein, and opioid precursor protein <sup>47,48,157</sup>
PMN leukocyte accumulation	Cytokines and chemokines accumulate; macrophages secrete inflammatory chemicals <sup>65–67</sup>

gene and heat shock proteins. The immediate early genes, *C-Fos*, *C-Jun*, and *Jun B*, are transcription factors regulating the expression of target genes, which include neuronal growth factor, cytoskeletal proteins, and metabolic enzymes.<sup>47,48</sup> The protein *fos* forms a chemical complex with *jun* and regulates the expression of target genes, which include nerve growth factor, amyloid precursor protein, and opioid precursor protein. All these genes appear to be upregulated after traumatic brain injury. In addition, the expression of these genes has been associated with programmed cell death, termed *apoptosis*.<sup>49–51,160</sup> Table 1.6 describes brain trauma-induced pathophysiologic changes. The diversity of causes and multifactorial events involved in traumatic brain injury should not be overlooked. The delayed consequences of the primary injury remain incompletely understood.

### NEUROCHEMICAL CHANGES FOLLOWING TRAUMATIC BRAIN INJURY

The mechanical forces during traumatic brain injury produce neurochemical changes that develop within the succeeding hours following injury.<sup>4</sup> These may be combined with secondary causes of injury such as cerebral hypotension, ischemia, edema, or changes in metabolism.<sup>5</sup> There is evidence that posttraumatic neurochemical changes are due to alterations in the synthesis or release of both endogenous neuroprotective agents and autodestructive agents, as described previously.<sup>3,9,52</sup> One of the compounds known to be altered is the neurotransmitter acetylcholine. There is an increase in the amount of acetylcholine found in the brains and cerebrospinal fluid of patients following head injury, and this is associated with decreased binding of acetylcholine at cholinergic receptors, particularly in the hippocampus. While Hayes et al. described these findings more than 20 years ago,<sup>53</sup> they have been replicated since, and it is now fairly well established that significant, adverse cholinergic changes follow traumatic brain injury.<sup>54,55</sup>

The most important early pathogenic mechanism in traumatic brain injury is alteration of the resting membrane potential of cells. This may be mediated by either voltage-dependent or agonist-dependent ion channels such as glutamate-dependent gates. Glutamate drives an increase in metabolism with secondary traumatic depolarization and hyperglycolysis.<sup>56,158</sup> Glutamate is an excitatory amino acid that induces a large calcium influx into the cells. The greater the strain on brain tissue and cells, the greater the peak intracellular free calcium concentration. The increased cellular concentration of calcium can lead to excitotoxic death.<sup>57,58</sup> Glutamate release not only affects calcium influx, but it increases hydroxyl radical production from cortical regions adjacent to the impact site. Glutamate seems to have a role in the pathogenesis of focal contusions.<sup>158</sup> There seems



to be an association between hydroxyl radical increase and glutamate release.<sup>59</sup> While calcium tends to increase intracellularly, there is a compensatory alteration in magnesium concentration within the cells. Intracellular free magnesium concentration shows a sustained decline that is apparent for about 4 days posttrauma, with eventual recovery to preinjury levels by day 6.<sup>60</sup>

## FREE RADICAL AND INFLAMMATORY CHANGES FOLLOWING TRAUMATIC BRAIN INJURY

The production of free radicals may be related to an association with glutamate release, as noted previously. However, the production of free radicals following traumatic brain injury also includes activation of the arachidonic acid cascade.<sup>61</sup> Thus, the activation of arachidonic acid and the increase in intracellular calcium induces the release of free radicals from mitochondria within the cell.<sup>62</sup> Free radicals are highly reactive molecules and they are thought to be activated by the mechanism known as oxidative stress. Central nervous system (CNS) tissue is particularly vulnerable to oxidative stress because of its high rate of metabolic activity, nonreplicating nature of neurons, and the high membrane-to-cytoplasm ratio.<sup>159</sup> Not only does oxidative stress activate free radicals, but it is also associated with other pathogenic mechanisms, such as glutamic acid excitotoxicity, intracellular calcium overload, mitochondrial cytochrome c release, caspase activation, and apoptosis of cells.<sup>63</sup> Cell death by either necrosis or apoptosis plays a role mediating tissue injury following brain trauma. Caspase-1 is also activated. Free radical production has been shown to be a downstream mediator of the caspase cell death cascade.<sup>64</sup>

Within 24 h of cellular changes following acute brain injury, polymorphonuclear leukocytes accumulate in damaged tissue.<sup>65</sup> Macrophages are commonly seen during the repair process, and they secrete soluble factors, including inflammatory chemicals. Both cytokines and chemokines have been implicated.<sup>66</sup> Interestingly, this type of cell activation following diffuse brain trauma strongly differs from that found after focal brain damage.<sup>67</sup> This form of injury may ultimately be repairable. Recent neural stem cell research in rats has been demonstrated to rescue hippocampal CA3 neurons when transplanted into the injured brain during the acute posttraumatic period.<sup>68</sup>

## APOPTOSIS FOLLOWING TRAUMATIC BRAIN INJURY

Simply put, apoptosis is programmed cell death. It can be triggered by excitatory amino acids, derangement of calcium homeostasis, free radicals, and death receptor–ligand binding. Cell death and survival and cell signaling are interrelated.<sup>160</sup> It is a phenomenon that is under intense investigation, and it has been found to occur following traumatic brain injury.<sup>69,70</sup> Some of the mechanisms discussed previously, such as increases in intracellular calcium and the production of free radicals, can cause cells to undergo apoptosis. There are other pathways involved in programmed cell death as well. Some of these seem to be related to a shift in the balance between proapoptotic protein factors and antiapoptotic protein factors.<sup>71</sup> With regard to children, however, apoptotic neurodegeneration has been shown to be an age-dependent neuropathological outcome after head trauma. The immature brain seems to be exceedingly vulnerable relative to the more mature brain. These results may help explain more unfavorable outcomes of very young pediatric head trauma patients when compared with their older counterparts.<sup>72</sup>

There is evidence that apoptosis can be suppressed genetically. The apoptosis-suppressor gene *bcl-2* is induced in brain tissue following injury, and it may serve to regulate neuronal death. It has been detected in infants and children and is thought to regulate cell death after traumatic brain injury in the pediatric age group. Increases in *bcl-2* have been found at higher levels in patients who survived than in patients who did not, and this finding is consistent with a protective role for this antiapoptotic protein.<sup>73,74</sup> Other studies have confirmed neuroprotection associated with *bcl-2* activity, and these findings have suggested that research focus on this gene may improve outcome after ischemia and trauma in youngsters and adults.<sup>75,76</sup>

## TYPOLOGY OF TRAUMATIC BRAIN DAMAGE

### SKULL FRACTURE

The presence of skull fracture indicates that impact to the head has occurred with force.<sup>22</sup> Interestingly, some patients with a skull fracture may have no evidence of brain damage and make an uneventful recovery. It is hypothesized that the energy producing the fracture is dissipated by the fracture itself, which in turn displaces the focus of energy into the skull bones rather than into the brain parenchyma. However, patients suffering a skull fracture due to head trauma have a much higher incidence of intracranial hematoma than those who do not sustain a fracture.<sup>77,78</sup> The type of fracture found following trauma is dictated in part by the shape of the object that makes contact with the head. Flat-shaped objects tend to produce fissure fractures, which can extend into the base of the skull, while angled or pointed objects produce a localized or stellate fracture.<sup>79</sup> Fractures at the base of the skull may give rise to infection. These fractures often pass through the petrous bone or the anterior cranial fossa (cribriform plate) and cause leakage of cerebrospinal fluid through the nose, mouth, or ear. Up to 30% of patients who have skull fractures producing leakages of cerebrospinal fluid develop tumor-like complications when the resulting cavity distends as a result of trapped air (aerocoele).

*Contrecoup* fractures (fractures located at a distance from the point of injury that are not direct extensions of a fracture originating at the point of injury) occur principally in the roofs of the orbits and the ethmoid plates after falls that cause trauma to the back of the skull<sup>4</sup> (the classic “slip and fall”). Skull fractures in infants and young children may give rise to subsequent complications due to the phenomenon of “growing fractures.” In youngsters, the dura is closely attached to the inner surface of the skull and thus is easily ruptured after a depressed skull fracture. This may cause the meninges or neuronal tissue to protrude outward between the fractured bones. This may delay or stop healing and leave a swollen mass of brain tissue or dural structures under the surface of the scalp.<sup>79</sup>

### FOCAL BRAIN DAMAGE

#### Contusions and Lacerations

A contusion is a type of focal brain damage caused mainly by contact between the surface of the brain and the bony protuberance of the base of the skull or by rapid acceleration–deceleration.<sup>4</sup> By definition, the pia-arachnoid membranes are intact over surface contusions, but they are torn following lacerations. Considered to be the hallmark of brain damage due to head injury, they have a very characteristic distribution generally affecting the frontal poles, the orbital gyri, the cortex above and below the sylvian fissures, the temporal poles, and the lateral and inferior aspects of the temporal lobes. Less frequently, the inferior surfaces of the cerebellar hemispheres are affected.<sup>80</sup> Contusions are not usually found in the parietal and occipital lobes unless there is a skull fracture in these areas<sup>79</sup> (Table 1.7). Neuropathological studies have demonstrated that the initial appearance

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**TABLE 1.7**  
**Characteristic Distribution of Brain Traumatic Contusions**

- Frontal poles
  - Orbital surfaces of the frontal lobes
  - Temporal poles
  - Lateral and inferior surfaces of the temporal lobes
  - Cortex adjacent to the sylvian fissures
-

of contusions evolves over time. Shortly following injury, a contusion is visible as microscopic regions of perivascular hemorrhage that follows the tracts of small vessels in the cortex, and it usually runs perpendicular to the cortical surface. This may occur almost instantaneously following injury. Over time, blood products seep into the adjacent cortex and neuronal structures in the immediate vicinity begin to degenerate. The destruction of neurons subsequently produces a glial scar. In some cases, the hemorrhage will extend into the white matter, causing demyelination of axons and loss of neuronal tracts. Necrotic tissue is removed by macrophages, and the contusion develops into a shrunken glial scar, which is apparent to the naked eye at autopsy.<sup>22</sup> The scar is often brownish as a result of residual hemosiderin filling the macrophages. In fact, magnetic resonance imaging (MRI) examination may detect hemosiderin deposits resulting from contusions at a time distant from the original injury (see [Chapter 5](#)). Old contusions can develop a pyramidal shape with the apex of the pyramid at the depths of the cortex and the base of the pyramid at the crest of a gyrus.<sup>79</sup>

It has been argued by some authors that contrecoup contusions are the most severe. However, neuropathological studies contrainituitively demonstrate that, in patients who receive frontal or occipital brain injuries, contusions are almost always more severe in the frontal lobes regardless of the point of injury. The use of contusion indexes has shed doubt on the concept of severity from contrecoup contusions.<sup>81,82</sup> With regard to lacerations, those in the frontal and temporal lobes are often associated with acute subdural and intracerebral hemorrhage. These lesions may be described by descriptive terms: “burst” frontal lobe and “burst” temporal lobe hemorrhages.<sup>4,80</sup>

Computed tomography (CT) head scan is the method of choice to detect acute intracranial hemorrhage, and it is the most likely brain imaging modality to be used in the acute care setting. Moreover, it often easily detects extradural or subdural hematoma. Cerebral contusions produce characteristic findings on CT of the head<sup>83</sup> (see [Chapter 5](#)). Recent autopsy cases have demonstrated that contusions may result in microthrombi throughout the brain. These are found to be much more dense in cerebral hemispheres containing contusions and potentially are involved in secondary brain damage after trauma.<sup>84</sup> As noted, the CT scan is the imaging method of choice in the acute phase of brain contusion. However, diffusion-weighted magnetic resonance imaging has been shown capable of detecting contusion injury as well. Diffusion-weighted imaging has been shown superior to T2-weighted MRI images to demonstrate cortical contusion injury.<sup>85</sup> Contusions from brain trauma have been studied for many years by neuropathologists. Numerous subdivisions have been defined.<sup>86-89</sup>

## Hemorrhage and Hematoma

Some patients will demonstrate a lucid interval after their head injuries. They then will show a deteriorating level of consciousness. The most common cause of this clinical deterioration is hemorrhage, and an apparently trivial head injury can turn into a life-threatening situation. Hemorrhages following head injury generally occur in three forms or areas of the brain ([Table 1.8](#)). Extradural, subdural, and intracerebral hematomas cause expanding intracranial lesions. These in turn produce a mass effect promoting increased intracranial pressure, and these compress the surface of the brain. Subarachnoid hemorrhage is often associated with the formation of contusions. Intraventricular hemorrhage is often seen in patients with diffuse axonal injury.<sup>79</sup>

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**TABLE 1.8**  
**Hemorrhages Occurring after Head Injury**

- Within the extradural, subdural, or subarachnoid spaces
  - Intraparenchymal
  - Into the ventricles
-

## **Extradural (Epidural) Hematoma**

Epidural hematoma occurs in approximately 2% of brain injuries in one series.<sup>88</sup> Epidural hemorrhage was present in 10% of the brain injury cases in the Glasgow, Scotland, database.<sup>7</sup> About 85% of epidural hematoma patients will also demonstrate a concurrent skull fracture. In young children, epidural hematomas can occur in the absence of such a fracture. The most common anatomical site for epidural hematoma is the temporal region, but in 20 to 30% of cases, these occur in other parts of the brain. The temporal bone is somewhat flexible, and with a direct impact, it will often deform inward, develop a fracture line, and transect or rupture an artery, or occasionally a vein, lying on the inner table of the skull. In cases where an artery is ruptured, the arterial pressure quickly forces blood into the potential space between the skull and the dura, producing an enlarging mass. It is this potential for quick enlargement that may produce a life-threatening situation due to pressure transfer throughout the brain, often resulting in downward herniation of the inferior brain and thus compromising brain stem structures. As the hematoma enlarges, it will gradually strip the dura from the skull and form an ovoid mass that progressively indents and flattens the adjacent brain.<sup>7</sup>

If the hematoma is small, surgical evacuation may not be required, but in many cases, in order to save the patient's life, open-head evacuation is the treatment of choice. Small hematomas may become completely organized over time. Large hematomas will undergo partial organization, and their centers will remain cystic, filled with dark viscous fluid.<sup>90</sup> The size of an extradural hematoma may increase up to 50% during the first 10 to 14 days after injury, and then the clot liquefies. Following the second week, the hematoma gradually shrinks and, in the majority of patients, may be completely resolved by the fourth to sixth week postinjury.

## **Subdural Hematoma**

Subdural hematomas are usually induced by rupture of the bridging veins, and there may be little other evidence of brain damage. A small number of subdural hematomas are arterial in origin, and the hemorrhage comes from a ruptured cortical artery.<sup>90</sup> Subdural hematoma has been reported to occur by whiplash injury where there has been no contact or physical injury to the head.<sup>91</sup> In acute fatal head injury, about 13% of subdural hematoma cases are pure and very little neuropathological evidence of other brain damage is present.<sup>7</sup> Since blood can spread freely throughout the subdural space, subdural hematomas tend to cover the entire hemisphere if bleeding is extensive, and they are almost always more extensive than extradural hematomas. However, most cases of subdural hematoma are associated with considerable brain damage, and the mortality and morbidity is greater in subdural hematomas than in extradural hematomas. In infants, subdural hematomas are the most common type of intracranial injury following child abuse.<sup>92</sup> These hematomas are usually associated with skeletal injuries, and they may contain a blood clot consisting of xanthochromic fluid. In these cases, they are referred to as subdural hygromas.

If a subdural hematoma is not surgically evacuated, the blood remains clotted for about 48 h, and at times several days. Subsequently, there remains a mixture of blood clot and fluidized blood. Generally, after about 3 weeks, the clot is absorbed. Interestingly, it has been observed by structural imaging that the gyral and sulcal patterns on the side of the hematoma is preserved. There is no flattening of the convolutions over the surface of the brain, although marked flattening of the convolutions over the opposite hemisphere is found. This occurs because the subdural blood is in direct contact with both the gyri and sulci and exerts uniform compression on the underlying brain tissue, which prevents flattening of its contiguous surface.<sup>7</sup> Unfortunately, in about 25% of patients who undergo a neurosurgical evacuation of an acute subdural hematoma, acute brain swelling occurs in the hemisphere directly beneath the clot, and this often carries a bad prognosis.<sup>93</sup>

Chronic subdural hematomas may present weeks or months after what appeared originally to be a trivial head injury. The hematoma becomes encapsulated in a membrane and increases its size

slowly. This is thought to be due to repeated small hemorrhaging into the structure of the hematoma. Eventually, it becomes large enough to distort and even herniate the brain downward. Chronic subdural hematomas are particularly common in elderly patients, as there is generally some cerebral atrophy present and the distance between the inner table of the skull and the brain surface is much greater than in younger individuals, allowing for greater brain excursion during falls or head trauma.

### **Subarachnoid Hemorrhage**

Generally, there is some degree of subarachnoid hemorrhage associated with contusions or intraventricular hemorrhage. It is also a frequent occurrence in patients who sustain diffuse axonal injury. It has been reported that detection of subarachnoid hemorrhage is difficult with MRI using standard T1- or T2-weighted images. However, traumatic subarachnoid hemorrhage can be confirmed with fluid-attenuated inversion recovery (FLAIR) imaging on MRI. In general, though, in the acute care setting, CT is the preferred method for demonstrating subarachnoid hemorrhage<sup>94</sup> (see Chapter 5). There have been reports of traumatic laceration of the intracranial vertebral artery causing fatal subarachnoid hemorrhage, but these persons generally do not survive.<sup>95</sup> Japanese neurosurgical studies have demonstrated that in closed-head trauma, those patients who exhibited subarachnoid blood on admission CT scans developed delayed ischemic symptoms between days 4 and 16 after head injury. There has been found a close correlation between the main site of the subarachnoid blood and the location of focal severe vasospasm in the same anatomic area.<sup>96</sup>

### **Intraparenchymal Hemorrhage**

In general, the definition of intracerebral hematomas are those that generally occur within the brain tissue and are not directly related to the surface of the brain. They are caused by the rupture of internal blood vessels within the brain that are often found deep in the cerebral hemispheres, particularly in the frontal and temporal regions following closed-head injury. Sequential CT scans have shown that these hemorrhages are often multiple, and their appearance on CT scan is often delayed and may become apparent only several hours or the following day after admission.<sup>97</sup>

In the Glasgow database, intracerebral bleeding or hematomas were found to be present in 16% of the cases.<sup>7</sup> While they are predisposed to the frontal and temporal lobes, they may also occur deep within the hemispheres and less commonly in the cerebellum. Patients with this type of bleeding have an increased incidence of diffuse axonal injury or what are known as gliding hematomas. Gliding contusions are usually bilateral, but often they are asymmetrical and sometimes restricted to the white matter. A more appropriate term is thought to be *parasagittal contusions*. Their presence is more related to diffuse than focal brain injury.<sup>3</sup>

If a solitary hematoma is found deep within the brain of a patient following head injury, the differential diagnosis includes either a hypertensive bleed or the rupture of a saccular aneurysm due to the head injury.<sup>7</sup> However, if the hemorrhage is in the subfrontal or temporal regions, it is more likely to be due to trauma than of spontaneous vascular origin.<sup>98</sup> Modern brain imaging has revealed that small hematomas or bleeding deeply seated in the brain is often found in the basal ganglia. In these patients, there is a reduced incidence of a lucid interval following injury and an increased incidence of gliding contusions and diffuse axonal injury. It has been suggested that patients found to have basal ganglia bleeding or hematomas shortly after head injury are likely to have sustained diffuse brain damage at the time of the injury.<sup>99,100</sup> CT observation reveals that a considerable proportion of intracerebral bleeding and hematomas are not detected until 48 h after injury.<sup>101</sup> Recent studies measuring glutamate, cytokines, and adhesion molecules have concluded that high levels of prior inflammatory molecules within 24 h of intracerebral hemorrhage are correlated with the magnitude of the subsequent perihematoma brain edema. Poor neurologic outcome and the volume of a residual cavity are related to increased plasma glutamate concentrations.<sup>164</sup>

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**TABLE 1.9**  
**Types of Diffuse Brain Injury**

- Diffuse axonal injury
  - Ischemic injury
  - Brain swelling
  - Vascular injury
- 

### **Intraventricular Hemorrhage**

Prior to the availability of CT scans, intraventricular hemorrhage often was not diagnosed. However, its presence is usually seen in patients with diffuse axonal injury. For many years, it was thought that the prognosis was poor in those patients who had sustained intraventricular hemorrhage. However, recent studies have challenged this assertion, and the death rate may be no higher in patients with intraventricular hemorrhage than in those without. Abraszko and others suggest that higher mortality is related to the other associated lesions seen with intraventricular hemorrhage rather than to bleeding into the ventricles alone.<sup>102</sup>

### **DIFFUSE BRAIN DAMAGE**

There are four principal types of diffuse brain damage, and three are seen frequently in patients who survive their injuries long enough to be admitted to the hospital: diffuse axonal injury, ischemic brain injury, and brain edema. The fourth principal type is diffuse vascular injury (Table 1.9). Patients who sustain this generally die very soon after their head injuries.<sup>3</sup>

### **Diffuse Axonal Injury**

Severe diffuse axonal injury not accompanied by an intracranial mass lesion occurs in almost 50% of patients with a severe head injury, causes 35% of all deaths after head injury, and is the most common cause of the chronic vegetative state and severe disability until death.<sup>7,103</sup> Severe cases of diffuse axonal injury have three distinctive features: (1) a focal lesion in the corpus callosum, which usually extends anterior and posterior along the axis and lies to one side of the midline, and is often associated with intraventricular hemorrhage; (2) focal lesions of the rostral brain stem adjacent to the superior cerebellar peduncles; and (3) microscopic evidence of widespread damage to axons. Damage to axons seems to be mostly involved in the corpus callosum and rostral brain stem lesions. Patients who sustain diffuse axonal injury often have associated gliding contusions, and hematomas in the basal ganglia and hippocampi. These injuries are particularly associated with acceleration–deceleration trauma in motor vehicle accidents, but they have also been described after assaults. Some patients who fall from considerable height will also sustain diffuse axonal injuries.<sup>104–107</sup>

Since it takes between 18 and 24 h for classic microscopic axonal bulbs to appear in the human brain following injury, it is likely that the incidence of diffuse axonal injury is probably higher than the published figures suggest.<sup>7</sup> Pathological histochemistry has demonstrated the presence of axonal swellings appearing 3 to 12 h after an injury.<sup>7,108</sup> Diffuse axonal injury should be suspected strongly if there are focal lesions in the corpus callosum and the appropriate areas of the brain stem noted by CT or MRI. If gliding contusions or hematomas are found in the basal ganglia on appropriate brain imaging, the likelihood is even greater that diffuse axonal injury has occurred.<sup>7</sup>

### **Ischemic Brain Injury**

Ischemic brain damage is common in patients dying as a result of nonmissile head injury. A detailed study of 151 cases reported in 1978 revealed an incidence of 91%.<sup>109</sup> Obviously, some of these

patients have survived due to improvement in the early management of head injury. The evidence is that ischemic brain damage occurs soon after injury.<sup>110</sup> However, the pathogenesis of ischemic brain damage is not fully understood. In years before neurosurgical techniques improved, it was more common in patients who sustained a known clinical episode of hypoxia following head injury (blood pressure less than 30 mmHg for 15 min). It has also been found to be more common in patients who experience high intracerebral pressure.<sup>111</sup> On the other hand, brain damage may occur without intracerebral pressure being high, and moreover, there is a statistically significant correlation between ischemic brain damage and the presence of cerebral arterial spasm.<sup>112,113</sup> Modern neurosurgical care has made us aware that some ischemic damage is avoidable by controlling factors such as obstruction of airway, providing appropriate control of epilepsy, relieving hypertension, and aggressively treating intracranial hematomas.<sup>7</sup>

### **Brain Swelling**

Brain swelling occurs frequently in association with head injury and may be localized or generalized. It can occur singly or in combination with other focal brain injuries. It may contribute to the elevation of intracerebral pressure by impeding hemodynamic corrections within the brain following trauma. Swelling of sufficient severity may cause morbidity or death by distant trauma to the brain stem. The causes of swelling are not always clear, but in many cases, they are due to an increase in the cerebral blood volume, which causes congestive brain swelling. Swelling may also result from increased water content of the brain tissue, producing cerebral edema. There are three principal types of brain swelling: (1) swelling adjacent to contusions, (2) diffuse swelling of one cerebral hemisphere, and (3) diffuse swelling of both cerebral hemispheres.<sup>7</sup>

Skull volume is finite. As mass lesions such as hematomas occur, intracranial pressure may increase. This increase is often contributed to by brain swelling. Swelling of one hemisphere is seen most often due to an acute subdural hematoma over that hemisphere.<sup>93</sup> Diffuse swelling of both hemispheres tends to appear in younger head injury victims. The pathogenesis of this type of brain swelling is not clear but may be related to loss of vasomotor tone and subsequent vasodilatation.<sup>114,115</sup>

## **SECONDARY INJURY AFTER HEAD TRAUMA**

The most obvious cause of brain injury is the acute physical insult or primary injury to the brain parenchyma itself. Secondary injury is the term reserved for the harmful subsequent effects on the brain. Secondary damage is most often associated with three issues: brain swelling, ischemia, and elevated intracranial pressure. Brain swelling has been discussed previously as a focal phenomenon. It was noted that swelling occurs adjacent to contusions or may be in one hemisphere or bilateral. Ischemia and elevated intracranial pressure are both associated with cerebral hypoperfusion and an alteration in the autoregulation of cerebral blood flow.<sup>116</sup>

Cerebral perfusion pressure is the difference between the mean arterial pressure and intracranial pressure. It may be reduced following head injury by either an increase in the intracranial pressure or a decrease in the arterial pressure bringing blood to the brain.<sup>117</sup> [Table 1.10](#) outlines the secondary mechanisms following traumatic brain injury that may lead to reduced cerebral perfusion pressure.

### **VASCULAR FAILURE**

It has been observed consistently in neurosurgical units that there is a reduction in cerebral blood flow following traumatic injury almost immediately. This may last as long as 24 h.<sup>118</sup> Many mechanisms seem related to blood flow change, including vascular disruption, vasospasm, thrombosis, postspreading cerebral depression hypoperfusion, and compression of the microcirculation due to astrocyte swelling.<sup>119</sup> This is a high-risk setting for secondary damage because of flow–metab-

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**TABLE 1.10**  
**Potential Causes of Reduced Cerebral**  
**Perfusion Pressure**

- Arterial hypotension
    - Hypovolemia
    - Cardiodepressant drugs
    - Sepsis
  - Intracranial hypertension
    - Mass lesions such as hematoma
    - Vascular engorgement
    - Cerebral edema
    - Acute hydrocephalus
- 

olism mismatch. Two outcomes usually follow: the early low-flow phase may progress to a state of normal, or there may develop persistently reduced blood flow during the period of cerebral swelling that usually follows.<sup>5</sup>

### **INTRACRANIAL HYPERTENSION**

As the brain injury evolves, cerebral swelling and intracranial hypertension often develop. This is associated with an increased permeability of the blood–brain barrier, oxidant damage, and leukotriene formation. Neurotoxic edema develops during this period and is a key contributor to swelling. Ionic shifts occur early after injury, causing release of glutamate and potassium. Astrocytes take up these ions and sodium, and water follows, causing astrocyte swelling. MRI studies have demonstrated that cellular swelling is the most important contributor to secondary cerebral swelling.<sup>5,120</sup>

As cellular and interstitial edema increases, intracranial hypertension follows. As the intracranial blood pressure exceeds the incoming brain perfusion pressure, inadequate cerebral perfusion results, causing secondary ischemic insult to the already damaged brain. Increases in cerebral blood volume from hyperemia may add to the swelling, but this is not considered to be an important cause in most patients.<sup>121</sup>

### **BRAIN SHIFT AND HERNIATION**

If a hematoma continues to enlarge, or focal swelling of adjacent brain tissue increases, the brain is shifted away from the growing mass, and structures that normally lie in the midline may be displaced. The falx is a very tough and adherent tissue and tends to remain in the midline. As a result, the cingulate gyrus may herniate under the free edge of the falx and cause compression or distortion of the pericallosal arteries.<sup>122</sup> Because the foramen of Monro becomes occluded in this process of midline shift, the contralateral ventricle may become dilated while the ventricle on the side of the mass becomes compressed. This sign on CT scan is a reliable indication that intracranial pressure is increased.<sup>123</sup>

With a hematoma, compression of the supratentorial compartment may occur. This is usually lateral and compresses the posterior cerebral artery and the third cranial nerve on the same side as the mass, markedly enlarging the ipsilateral pupil. In bilateral or frontal lesions, the swelling may cause posterior herniation compressing the tectal plate, which results in bilateral pupil abnormalities and inability of the patient to look upward.<sup>124</sup> With infratentorial masses, or a further progression of a supratentorial mass, herniation eventually occurs with downward displacement of the cerebellar tonsils through the foramen magnum. This will compress the medulla, causing apnea followed by cardiac arrest and death.<sup>116</sup>



**TABLE 1.11**  
**Alzheimer's-Like Aftereffects of Brain Injury**

Process	Features
Beta amyloid deposition	Increased expression of amyloid precursor protein following trauma; greater deposition of amyloid beta peptide <sup>130-142</sup>
Apolipoprotein E production	Gene is located on chromosome 19 and has alleles E2, E3, and E4; presence of E4 allele in head-injured patient may increase risk of later-appearing neurodegeneration <sup>145-148,161</sup>
Cholinergic dysfunction	Trauma reduces cholinergic binding in hippocampus with formation of amyloid plaques <sup>5,151-153</sup>

## RELATIONSHIP OF TRAUMATIC BRAIN INJURY TO LATE-APPEARING NEURODEGENERATION

Survivors of closed-head injury often have long-lasting neurological aftereffects. These include the development of neurodegenerative disorders.<sup>4,5</sup> Traumatic brain injury is now thought to be a significant risk factor for Alzheimer's disease.<sup>125,126</sup> Studies in boxers have noted a relationship between the apolipoprotein genotype and the development of Alzheimer's-like dementia.<sup>127</sup> In contrast to many studies demonstrating a relationship between traumatic brain injury and later development of neurodegeneration, a recent study in Rotterdam did not concur with previous cross-sectional studies suggesting an interaction with the apolipoprotein genotype and increased risk for Alzheimer's-like dementia following traumatic brain injury,<sup>128</sup> but it is in the minority in this regard. [Table 1.11](#) categorizes Alzheimer's changes following brain injury.

### TRAUMA-INDUCED BETA AMYLOID DEPOSITION

A leading contemporary theory for the biological basis of Alzheimer's disease is the formation of beta amyloid within the brain. Amyloid is known to destroy cholinergic neurons in the nucleus basalis of Meynert, and as Alzheimer's disease progresses, this damage becomes more widespread. Alzheimer's disease may be essentially a problem with too much formation of beta amyloid or too little removal of it.<sup>129</sup> A major component of these plaques is the 42-43 amino acid amyloid beta peptide that is cleaved from the transmembrane portion of amyloid precursor protein. One condition that can alter amyloid precursor protein metabolism, and is considered to be a risk factor for Alzheimer's disease, is head trauma.<sup>5</sup> The exact mechanism by which head injury leads to Alzheimer's disease-like pathology is not known. However, experimental evidence in animal and human models shows an increased expression of amyloid precursor protein and deposition of amyloid beta peptide after head trauma.<sup>130-142</sup> These studies suggest that injury-induced alterations in amyloid precursor protein expression and processing may result in increased deposition of beta amyloid, which in turn initiates the development of Alzheimer's disease-like pathology. Moreover, brain trauma may accelerate this process and increase the risk of later developing an Alzheimer's-like dementia syndrome.

### THE GENETIC COMPONENT OF TRAUMATIC BRAIN INJURY

Apolipoprotein E is an important genetic risk factor for late-onset Alzheimer's disease (neurodegeneration beginning after age 60). This gene is located on chromosome 19 and has three alleles: E2, E3, and E4.<sup>5</sup> Apolipoprotein E is a lipoprotein produced by brain astrocytes and microglia. It apparently has a role in transporting lipids to injured neurons to help them heal. These lipids are

primarily cholesterol derived.<sup>143</sup> In classic Alzheimer's disease, individuals who possess one or both E4 alleles are at increased risk of Alzheimer's disease, with a twofold increased risk for one allele and a sixfold increased risk for two alleles when compared with other genotypes.<sup>144</sup> The presence of the apolipoprotein E4 allele, along with a history of head injury, has been reported to increase the risk of Alzheimer's-like neurodegeneration from twofold to tenfold.<sup>145</sup> A similar effect has been observed in professional boxers who possessed an E4 allele when compared with boxers who did not possess this genetic subtype.<sup>146</sup> These findings in humans have been replicated following trauma in animals.<sup>142,147,148</sup> These data suggest that in the future allele measurements for specific apolipoprotein factors may be significant in determining prognosis of traumatically brain-injured patients.<sup>161,162</sup>

## CHOLINERGIC MECHANISMS AND NEURODEGENERATION

Impairments of attention and memory have been well characterized in traumatic brain injury. These are likely related to disruption of cholinergic functioning in the hippocampus. Additionally, disturbances in this neurotransmitter system may account for dysfunction in sensory gating systems and discriminative attention ability in head injury victims. The encephalographic P50 waveform of the evoked response to paired auditory stimuli has recently been shown to be a physiological marker of impaired sensory gating among traumatic brain injury survivors. This electrical marker probably represents cholinergic dysfunction.<sup>149</sup> Traumatic brain injury has been demonstrated to reduce hippocampal alpha 7 nicotinic cholinergic receptor binding. This has been measured using bungarotoxin binding 48 h following injury, and the binding defect seems related to the high calcium permeability of the alpha 7 nicotinic system.<sup>150</sup>

Alzheimer's disease clinically is associated with loss of memory and neuropathologically reveals deposition of neurofibrillary tangles with formation of amyloid plaques. Biochemical studies in patients with Alzheimer's disease demonstrate loss of cholinergic activity, particularly in the choline acetyltransferase enzyme systems.<sup>151</sup> In traumatic brain injury, there is a loss of memory and cholinergic neurotransmission with increased deposition of amyloid bodies. This suggests a relationship between the cholinergic deficits of brain trauma and Alzheimer's disease. Intact cholinergic neurotransmission is important for cognitive function. However, it may also play a role in determining the development of Alzheimer's-like neurodegeneration by influencing amyloid precursor protein metabolism.<sup>5</sup> *In vitro* studies demonstrate that stimulation with cholinergic agents at the M1 receptor will shift the processing of amyloid precursor protein in favor of its N-terminal secreted form and, as a result, decrease the formation of amyloid bodies.<sup>152</sup> Animal studies have demonstrated that cholinergic memory deficits persist in severely injured rats more than 10 weeks following posttraumatic brain injury. There is an initial period when overt deficits are present and these can be observed clinically. Following recovery of the overt deficits, the memory defect can be detected by pharmacologic challenge with scopolamine. Covert deficits can persist long after the recovery of clinical evidence of injury, and like other neurological deficits, the rate of recovery is dependent on the magnitude of the brain injury.<sup>153</sup> Further studies have demonstrated that cognitive deficits due to chronic changes in cholinergic systems can be modulated and improved by neurotrophic factors such as nerve growth factor stimulation.<sup>154</sup>

Alterations of cholinergic mechanisms appear not only to be related to the possible development of Alzheimer's-like neurodegeneration later in life, but also to have a profound impact in children on ongoing development due to their adverse affects upon memory and learning. Careful analysis of children for memory deficit following brain injury must be made to ensure proper educational rehabilitation. A dose-response relationship between memory functions within the acute phase of recovery is not easily detectable in children. This develops over time, with greater memory impairments evident for children with more severe traumatic brain injury. However, it is not easily detected until 12 months or more postinjury.<sup>155</sup>

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# 2 Neuropsychiatric and Psychiatric Syndromes Following Traumatic Brain Injury

## NEUROPSYCHIATRIC SYNDROMES

### INTRODUCTION

Neuropsychiatric syndromes following traumatic brain injury are not well delineated from the classical psychiatric syndromes such as depression, psychosis, or anxiety. As a term of art, they refer to complex brain–behavior relationships that affect cognition or that may result in neurobehavioral syndromes such as posttraumatic epilepsy, central nervous system hypersomnolence, posttraumatic headache syndrome, or normal-pressure hydrocephalus. Thus, these disorders present with both features of altered behavior and a brain-based neurological disorder.

There are recognized risk factors for the development of neuropsychiatric disturbances after traumatic brain injury. These include increased age, atherosclerosis, and alcoholism. These interfere with or delay the restorative processes occurring in the central nervous system following brain injury.<sup>1</sup> There is not a good classification system for neuropsychiatric disorders seen following traumatic brain injury.<sup>2</sup> Moreover, the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV), classification of organic mental disorders leaves much to be desired. The clinician will experience great difficulty attempting to classify traumatic brain injury syndromes within the framework of the DSM-IV.<sup>124</sup> Except for “dementia due to head trauma,” all other applicable disorders found in the DSM-IV are classified as “disorder due to general medical condition.” Thus, there is no scientifically validated way to use the commonly accepted psychiatric classification system, other than in a descriptive sense, and apply its diagnostic structure to the neuropsychiatric sequelae of traumatic brain injury.

### ADULT COGNITIVE DISORDERS

#### Disorders of Attention

When assessing cognition in a patient suspected of having a neuropsychiatric syndrome, it is of paramount importance to first assess attention. If attention is significantly altered, the remainder of the cognitive examination will thereby be altered as well. Sensory information cannot be processed if the person cannot attend to the stimulus. For instance, about 9% of consecutively referred patients suffering severe head trauma have impairments in vigilance (the maintenance of attention over time), whereas 77% of remaining patients showed increased distractibility within the context of normal vigilance.<sup>3,4</sup>

Following a mild traumatic brain injury, performance on simple measures of attentional capacity, such as the Digit Span subtest of the *Wechsler Intellectual Scales*, may recover to relatively normal levels. Alterations in attention may not be uncovered unless more sophisticated neuropsychological measures are used, and then prominent deficits may be noted in these same patients. In addition, slowed information processing speed is a sensitive and well-documented cognitive sequelae of head trauma. During the face-to-face mental status examination, little may be noted by the clinician other than a perception that the patient is not thinking very quickly. When more sophisticated neuropsychological measures are applied, deficits in the application of divided attention under progressively increasing rates of information processing speed may be noted. In those patients with mild head injury only, reduction in mental processing speed tends to be restricted to the first 1 to 3 months after recovery, and thereafter in most patients, mental processing speed returns to near baseline levels.<sup>5</sup>

The appearance of attentional deficits may be dependent upon the cognitive load placed on the injured person. In other words, these may not appear until sufficient cognitive loading is placed as a demand on the brain of the individual. The more effort required for the person to pay attention, the more likely the attentional deficit will be detected. Moreover, patients may also demonstrate difficulty refocusing their attention after a period of delay from stimuli. If the task is short, such as commonly performed in a face-to-face mental status examination, the attentional deficit may not be detected. More sophisticated attentional tasks, such as presented by neuropsychological evaluation, will generally reveal these deficits. One form of cognitive loading is to provide the individual with a stimulus that distracts him while he attempts to focus his attention on a target or other stimulus. Responses may be omitted within this type of assessment. On the other hand, patients may have difficulty inhibiting responses when asked to do so. Sensitive executive tests such as the *Wisconsin Card Sorting Test* or the *Category Test* may detect these impairments that will otherwise not be revealed by ordinary mental status examination (see [Chapter 6](#)).

## Disorders of Memory

Of the many cognitive functions affected following head trauma, memory is usually the most severely affected ([Table 2.1](#)). This is due to the high concentration of lesions preferentially found in the frontal and anterior temporal brain structures following closed-head injury. These brain areas contain the hippocampi and other neuronal structures that are strongly implicated in the storage and retrieval of new memories. Damage to these areas occurs from blunt trauma due to the protrusions within the skull of the sphenoid ridges. These may catch the temporal lobe tips, while the ridges on the infraorbital frontal fossae may bruise the anterior-inferior frontal lobes.

The memory loss following traumatic brain injury follows Ribot's law in that the memories most susceptible to disruption by organic pathology are those that were formed the most recently.<sup>6</sup> Patients who have sustained traumatic brain injury perform significantly worse than controls on prospective memory tasks, indicating that traumatic brain injury affects not only retrospective, but also prospective memory.<sup>7</sup> Studies have also indicated that persons who sustain traumatic brain injury show less impairment on explicit (factual) memory tasks than implicit (procedural) memory tasks.<sup>8</sup>

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**TABLE 2.1**  
**Elements of Memory Disorders in the Traumatically Brain-Injured Patient**

- Memory is usually the most affected cognitive function.
  - Ribot's law: There is a gradient of memory loss: recent > remote.
  - Explicit memory is affected greater than implicit memory (factual > procedural).
  - Patients report greater memory loss than their relatives.
  - The duration of anterograde amnesia is almost always longer than the duration of retrograde amnesia.
-

Many authors assert that if the traumatic brain-injured person recovers from posttraumatic amnesia, this indicates that the person has regained a grossly normal level of orientation and awareness of ongoing events. However, this does not imply that the patient's memory has returned to normal. Levin has found that, among patients who recovered normal intellectual functioning (full-scale IQ greater than or equal to 85), disproportionately severe memory deficit was found in 16% of those recovered from moderate head trauma and 25% of those recovered from severe head trauma. Patients tend to report a lower rate of memory complaints than relatives do, and this probably reflects their lack of insight or organic denial affecting self-monitoring following head trauma.<sup>9</sup>

The cause of memory disorders following traumatic brain injury is likely secondary to the relatively predictable pattern of diffuse and focal neuropathology sustained by persons with head trauma. This in turn results in high concentrations of parenchymal and extraparenchymal lesions in the frontal and anterior temporal lobes, the areas most likely to subserve memory.<sup>10</sup> These areas contain the hippocampus and other neuronal structures that are purported to be anatomical areas involved in the storage and retrieval of newly formed memories.<sup>11,12</sup> The orbitofrontal lobes, as previously noted, are particularly sensitive to injury during closed-head trauma. Moreover, the lateral areas of the temporal poles are also very susceptible to contusions or bruises. Hippocampal damage may result from release of excitotoxic amino acids after the injury.<sup>13-15</sup>

When evaluating traumatic brain injury patients, it is often useful to ask what is the last event remembered before the traumatic impact, whether they remember the impact itself, and the first thing they remember following impact. These are crude historical markers for retrograde and anterograde posttraumatic memory deficit. Retrograde amnesia extends backward in time from the moment of the trauma, whereas anterograde amnesia extends forward in time from the moment of that trauma.<sup>16,17</sup> The classical studies by Russell and Nathan<sup>17</sup> found that, in patients who recovered from traumatic brain injury, the duration of their retrograde amnesia is almost always much shorter than the duration of the anterograde amnesia. Thus, the majority of patients who sustain a traumatic brain injury will report a residual retrograde amnesia of only seconds or minutes in duration, whereas the anterograde amnesia will almost always be much longer than this by their reports. It was Ribot<sup>6</sup> who first wrote of a large survey of patients reporting memory disorders following trauma. His work proposed a temporal gradient of retrograde memory loss for head trauma patients that was subsequently confirmed by Levin and others.<sup>18</sup> Posttraumatic amnesia can be correlated to the severity of injury (Table 2.2) and related to the estimated time before a patient is capable of resuming work.<sup>19</sup>

It has been reported that patients recover their orientation following head trauma within the concept of a shrinking retrograde amnesia.<sup>20</sup> Patients who have sustained head trauma typically misstate the date to be earlier than the true date, although as they recover their memories, their orientation errors typically move forward in time to approximate the current date. When measures of new learning memory are applied to those who have sustained traumatic brain injury, 10% of patients with good recovery will show a deficit, while 44% of patients with moderate disability

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**TABLE 2.2**  
**Posttraumatic Amnesia Duration Related to Severity**  
**of Injury**

Degree of Concussion	Length of Posttraumatic Amnesia	Estimated Time before Resuming Work
Slight	Less than 1 h	4-6 weeks
Moderate	1-24 h	6-8 weeks
Severe	1-7 days	2-4 months
Very severe	More than 7 days	4-8 months

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will demonstrate memory impairments, and 100% of patients with severe disability will have some deficits of new learning memory.<sup>21</sup> Studies have been extended into time; 7 years after severe head trauma, memory deficit is still the single most frequent symptom reported by patients (53%) and their relatives (79%).<sup>22</sup>

## **Disorders of Language**

About 2% of patients consecutively admitted to a neurosurgery head trauma service are aphasic. Approximately one-third of these patients manifest one of the classic aphasic disorders. Of these aphasic patients, 51% have fluent aphasias, 35% have nonfluent aphasias, and 14% demonstrate global aphasias. Approximately another third of the patients are nonaphasic but demonstrate dysarthria. All patients admitted to rehabilitation after head trauma in this study demonstrated language deficits on sophisticated psychometric testing. This has been termed subclinical aphasia in that the patient has adequate conversational language, yet shows a clear language deficit on more challenging language testing.<sup>23</sup>

Anomia is the most common language disturbance seen after head trauma with sparing of fluency, repetition, and comprehension.<sup>24,25</sup> However, the examiner may notice that the patient speaks by circumlocution, and she may demonstrate semantic paraphasias. Semantic paraphasia may present by switching one word term for another. During neuropsychological testing of patients who have aphasia-related head injury, the highest rate of defective performance will be seen in confrontational naming. Deficits in comprehension, writing praxis, and verbal associative fluency will be seen at lower rates of occurrence. The ability to repeat words remains relatively preserved on neuropsychological testing.<sup>24</sup>

If a patient sustains a language disorder as a result of traumatic brain injury, the prognosis is fairly good. One study demonstrated that, of patients suffering acute aphasia after head trauma, 43% had full recovery of language functioning. Another 28% remained globally aphasic, and a second 28% had resolution as to the specific language deficits, but an anomia remained. On physical examination, the examiner may notice neurological factors associated with aphasias after head trauma, primarily right hemiparesis associated with left hemisphere brain damage.<sup>22,26</sup>

The physician performing a neuropsychiatric examination following traumatic brain injury will generally see the patient long after the acute phase of recovery. Language deficits that are no longer evident at the time the neuropsychiatric examination is performed may have been quite prominent immediately following the brain injury. For instance, posttraumatic mutism is present acutely in approximately 3% of patients despite the recovery of consciousness and ability to communicate in the neurosurgical intensive care unit. These patients typically have lesions in the putamen and internal capsule subcortical structures, or they have had cortical lesions develop in the left hemisphere.<sup>27</sup>

If aphasic traumatic brain injury patients recover their basic language abilities, the conversational discourse of these patients is often characterized by deficits that are not easily related to standard language parameters of fluency, repetition, comprehension, and naming. For instance, it has been demonstrated that, in patients who have preferentially left prefrontal traumatic lesions, the communication is characterized by disorganized and impoverished narratives. In contrast to this, the communication of patients who have suffered right prefrontal injuries tends to be tangential and socially inappropriate.<sup>28,29</sup>

## **Visual-Perceptual Disorders**

Most individuals who suffer a closed traumatic brain injury display normal visual-perceptual abilities.<sup>30</sup> However, in patients who sustain brain contusions or hematomas, those who have right hemisphere bruising or bleeding are more likely to show a deficit of visual perception. Visual-spatial function remains relatively preserved in patients even following severe head trauma.<sup>31</sup> Constructional ability or drawing praxis is also generally preserved in these individuals.<sup>32</sup>

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**TABLE 2.3**  
**Visual-Perceptual Disorders Following Traumatic Brain Injury**

- Visual-perceptual disorders are usually absent following traumatic brain injury.
  - Hematomas or contusions may predispose to visual-perceptual impairment.
  - Left parietal lesions can cause confusion, simplification, and concrete handling of designs.
  - Right parietal lesions may cause distortions or misperceptions of the design.
  - Usually the anterior–posterior gradient of head trauma spares the more posterior visual-perceptual cortex.
- 

Patients who sustain lesions in the left parietal areas tend to show confusion, simplification, and concrete handling of visual designs. When they approach a visual task, they are likely to be orderly and typically work from left to right. On the other hand, patients with right-sided lesions may begin at the right of the design and work to the left. Their visual-perceptual deficits may show up as distortions of the design or misperceptions of the design. Some patients with severe visual-perceptual deficits will lose sight of the squaring of corners or be unable to appreciate self-contained formats of the design. Lesak<sup>33</sup> notes that the Block Design subtest from the Wechsler IQ Scales is an excellent measure of visual-spatial organization ability. Block Design scores tend to be lower in the presence of any kind of brain injury, and they are generally lowest when the traumatic lesion involves the posterior right brain.<sup>34</sup> Edith Kaplan has called our attention to the performance of brain-injured patients in that errors occurring more at the top than the bottom of the visual field are important, as the upper visual fields have temporal lobe components, while the lower visual fields have parietal lobe components. Thus, a pattern of errors clustering at the top or the bottom corners can also give some indication of the anatomical site and also the extent of the lesion.<sup>33</sup>

It is generally accepted that the reason visual-perceptual skills are relatively preserved after closed-head trauma is consistent with the knowledge that there is an anterior-posterior gradient of tissue destruction usually induced by closed-head trauma. There is a sparing of the visual-perceptual processing systems that are located in the posterior aspects of the brain, as the structural irregularity of the skull in the posterior area is less than the more rough, inner skull surfaces present in the anterior portions of the skull<sup>35</sup> (Table 2.3).

### **Executive Disorders**

Human executive functions can be conceptualized with four components: (1) volitional behavior, (2) planning for the future, (3) action with a purpose, and (4) monitoring or regulating one's behavior<sup>33,36</sup> (Table 2.4). Executive functions are a form of supraordinate neurobehavioral systems that both motivate self-initiated behavior and govern the efficiency and appropriateness of task performance. The standard psychiatric clinical interview or basic psychological evaluation often fails to detect the presence of significant executive deficits. However, head trauma patients with executive dysfunction may lack the initiative to get anything done once they leave the professional's office. The adaptive functioning of patients is often impaired because they lack the necessary flexibility of reasoning and problem solving to respond to a complex environment.

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**TABLE 2.4**  
**Executive Dysfunction Due to Traumatic Brain Injury**

- Human executive function equals volitional behavior, planning for the future, purposeful action, and regulating one's behavior.
  - Impairment of executive function leads to disorders of emotional intelligence.
  - The standard psychiatric mental status examination may be inadequate to uncover executive disorders.
-

In the standard clinical examination, the psychiatrist usually asks leading questions and actively guides the interview while the patient passively provides what may well be habitual answers based on preinjury knowledge. Moreover, most psychological evaluations follow standard psychometric procedures, and the person is examined using highly structured tasks with explicit instructions. Often, situations are not open-ended enough to detect the executive dysfunction present following traumatic brain injury, as these do not adequately measure performance that requires self-initiation and active self-monitoring of performance. The adaptive function necessary to lead satisfactory lives is often termed *emotional intelligence*. It is probably more important to human efficiency and success than is test IQ. In the brain-injured patient, the individual may lack the necessary flexibility of reasoning and problem solving to respond to her environment as it challenges her with novel or complex situations.<sup>36</sup> The Wisconsin Card Sorting Test or the Category Test may help to delineate executive disorders.<sup>33</sup>

Intrinsic vocabulary levels are generally resistant to closed-head trauma, and no deficit may be noted in the person's general vocabulary abilities. However, if the brain-injured patient is asked to generate as many words as she can, starting with a specific letter of the alphabet, her performance is often below preinjury levels.<sup>37</sup> Following head trauma, patient vocabulary is generally well preserved. However, mental flexibility may be so lacking that the individual cannot access all of the words potentially available in her lexicon. In a similar vein, patients with executive dysfunction may well retain the ability to construct figures by drawing when imitating a model, but when they are asked to generate their own design independently, they may be unable to do so.<sup>33</sup>

## Intellectual Disorders

Piaget has said that intellect is what we use when we do not know what to do.<sup>38</sup> However, in traumatic brain injury, the untoward effects on intellectual functioning are often indirect rather than direct. In fact, intelligence testing, using instruments such as the *Wechsler Adult Intelligence Scale-III*, is poor in detecting brain injury, and full-scale intellectual changes measured by similar test instruments after head injury may not be significantly different from those of normal age-matched controls. On the other hand, certain subtest scores measured by the *Wechsler Memory Scale-III* are very likely to show diminishment following traumatic brain injury when compared with those of controls.<sup>39</sup> In fact, measurement of IQ alone is not an appropriate yardstick when determining cognitive changes following traumatic brain injury. The predominant reason that IQ testing alone is a poor choice for injury assessment is that intelligence testing does not tap into many of the critical areas of a person's cognitive functioning, such as personality regulation, short-term memory, attentional capacity, and executive function.<sup>40</sup> Cattalani et al. have shown in adults who were head-injured as children that changes in intellectual functioning are less important years after a traumatic brain injury than are prevailing problems of social maladjustment and poor quality of life, which seem related to behavioral and psychosocial disorders.<sup>41</sup>

The effects of brain injury upon intellectual functioning are often indirect rather than direct. The problems of capacity to concentrate, use language, abstract, calculate, reason, remember, plan, and process information are often affected in head injury. Many of these functions are poorly tapped by standardized intellectual test measurement. On the other hand, there are data to demonstrate that using the Glasgow Outcome Scale as a measure against median IQ scores, a correlation with a drop in intelligence following head injury can be shown.<sup>21</sup> In a study of 27 patients followed after severe head trauma, all patients with a good recovery demonstrated mean intelligence scores above 85; the IQ scores of patients with moderate disability ranged from 73 to 114, and the IQ scores of patients with severe disability ranged from 39 to 69. Dikman and his colleagues tested IQ scores at 1, 12, and 24 months after moderate to severe head trauma. Compared with the patients' postinjury baseline, they found a 17-point increase in verbal IQ and a 25-point increase in performance IQ at 12 months postinjury. At 24 months postinjury, verbal IQ had improved 4 more points, while performance IQ had improved 7 points. There is the possibility that this study measured

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**TABLE 2.5**  
**Unique Characteristics of Pediatric Traumatic Brain Injury**

- Traumatic brain injury affects a developing brain more so than a mature brain.
  - Children below age 5 are much more affected by traumatic brain injury than older children.
  - Brain plasticity does not benefit the very young child after traumatic injury.
  - Three-fourths of preschool brain-injured youngsters may not work as adults.
- 

practice effects at 24 months.<sup>42</sup> Thus, it is recommended that measurements of intelligence following brain injury be used to develop internal standards against which other neuropsychological tests may be compared. Use of intellectual testing as a single measure of cognitive change following brain injury is not recommended.

### **CHILD COGNITIVE DISORDERS**

There is a natural tendency to assume that because the plasticity of the developing brain is so dramatic, children suffering a traumatic brain injury will recover function, as they still have time for cerebral growth. In fact, there is evidence of dramatic recovery of function after focal and unilateral hemispheric damage in young children.<sup>43,44</sup> However, there is a caveat: the effects of diffuse insult produced by traumatic brain injury in children may ultimately result in greater cognitive impairment in a developing brain than in a mature brain. In fact, there is an inverse age-related gradient. Children below 10 years of age are more at risk for significant cognitive impairment following brain injury than are adolescents, whereas infants and toddlers are at greater risk for brain damage following trauma to the head than children of preschool and kindergarten age.<sup>45,46</sup> Children who sustain head injury are no different in their population distribution than adults. They do not represent a random sample of the population, and thus there is a higher rate of premorbid learning disability, academic dysfunction, and developmental disorders in children who sustain head trauma than in children who are not so injured.<sup>10</sup> Recent studies suggest that brain plasticity does not benefit outcome when diffuse cerebral pathology of the young child's brain is concerned. Nybo and Koskiniemi followed severe brain-injured preschool youngsters until age 21. Only 27% of youngsters worked full-time by age 21. There was a strong direct relationship between tests measuring speed, executive, and memory functions vs. vocational outcome. These results support the notion that the very young child's brain is much more susceptible to early trauma than the older child's brain<sup>55</sup> (Table 2.5).

### **Disorders of Attention**

Only a limited number of studies have investigated attention abilities following pediatric head injury. Research using objective measures of attention is relatively limited, and very few studies have provided a comprehensive assessment of attention based on current theoretical models.<sup>47,48</sup> Most studies have shown that severe traumatic brain injury causes greater deficits of sustained attention in children than in those who have had mild to moderate injuries, particularly in the acute stage and during recovery. In those children who develop attentional deficits following traumatic brain injury, the attentional symptomatology in the first 2 years after injury, in both children and adolescents, is significantly related to the severity of injury. Moreover, the overall symptomatology in that 2-year period is also significantly related to the level of family dysfunction.<sup>49</sup>

In an effort to clarify further the attentional deficits following traumatic brain injury and differentiate those factors from attention deficit hyperactivity disorder, Konrad et al.<sup>50</sup> evaluated 8- to 12-year-old children. Not only did they note a general slowing of information processing in those children suffering brain injury, but they noted that this did not correlate with the level of inhibition deficit in the children. They concluded that slowing of information processing speed is



a general consequence of traumatic brain injury in childhood, whereas slowing of inhibitory deficits is related to attention deficit, which appears postinjury. Those children who merely had attention deficit hyperactivity disorder unrelated to brain trauma did not display slowing of information processing. Studies of attention in brain-injured preschool children between 3 and 8 years indicate that youngsters recover many of the deficits of arousal and motivation over time, whereas focused attention, impulsivity, and hyperactivity often remain as prominent chronic features.<sup>51</sup> Attentional weaknesses among children with severe head injury are generally demonstrated by poor modulation of responses, especially in the presence of distracters. These deficits appear to be more pronounced among younger head-injured children than among their older counterparts.<sup>52</sup> Measurements indicate that the deficits are primarily in sustained and divided attention, whereas focused attention remains relatively intact.<sup>53</sup> A recent study demonstrated that in those children who develop significant attention deficit hyperactivity syndromes following traumatic brain injury, lesions can be noted in the right putamen on magnetic resonance imaging (MRI).<sup>54</sup>

### **Disorders of Memory**

Traumatic brain injury may have a profound impact on a child's ongoing development. The occurrence of memory disorders following childhood brain trauma is frequent, and the magnitude of the deficits is dependent upon injury severity.<sup>48</sup> Most studies have reported impairment of verbal memory, and few studies have examined nonverbal memory disorders. Verbal memory impairments include tests of recognition memory for words, word-list learning, paired-associates learning, and story recall.<sup>56-59</sup> The nonverbal studies have reported impairment in the recall of shapes from the *Tactual Performance Tests* and impairment in the reproduction of simple and complex geometric shapes.<sup>60,61</sup> Memory deficits in children occur in a variety of memory components, and they are not confined to a single entity. For instance, studies in children have demonstrated impairments in storage, retention, and retrieval.<sup>62</sup> Yeates et al. have found that children with severe injuries display poor learning, less retention over time, and better recognition than recall when compared to matched controls.<sup>57</sup>

If children have their memory measured during the acute phase of traumatic brain injury recovery, no reliable dose–response relationship between injury severity and memory function can be found. However, if children are measured 12 months or more following brain injury, this relationship is shown to develop over time. In other words, the more severe the injury, the greater the memory deficit measured at 12 months or more postinjury.<sup>63</sup> When memory is measured implicitly and explicitly, children show more impairment of explicit memory than they do implicit memory<sup>64</sup> (factual more than procedural).

### **Disorders of Language**

As noted previously with adults, language disorders in brain-injured children also are uncommon. However, given that, in the pediatric age group, children are more likely than their adult counterparts to have language difficulties following traumatic brain injury. Moreover, children display pronounced difficulties with the pragmatic aspects of language. Various research studies in children have noted deficits such as interpreting ambiguous sentences, making inferences, formulating sentences from individual words, and explaining figurative expressions. Deficits in these skills reflect a general impairment in discourse, otherwise noted as the ability to convey a message by communicating a series of ideas, usually in sentences. Studies of narrative discourse in children using story recall indicate that children with severe closed-head injuries use few words in sentences within their stories.<sup>48</sup> It has further been noted that injuries sustained at an earlier age consistently predict poor performance on language tasks in brain-injured children. This complicates the ongoing mental development of youngsters as they acquire communicative skills. In contrast, the severity of the injury does not predict language performance as strongly as youthful injury.<sup>65</sup>

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**TABLE 2.6****Language Disorders Following Pediatric Traumatic Brain Injury**

- Children are more likely than adults to develop disorders of language following traumatic brain injury.
  - Pragmatic aspects of language often are affected.
  - Problems are commonly seen with interpreting ambiguous sentences, making inferences, or explaining figurative expressions (abstract language).
  - Speaking rate, articulatory speed, and linguistic processing often are reduced.
  - Injury below age 5 often reduces ability for discourse.
- 

Brain-injured youngsters often show a reduction in speaking rate and impairment of articulatory speed and linguistic processing. These impairments seem related not only to a reduction in the speed of forming words, but an increased time between the expression of these words. Both reduced articulatory speed and linguistic processing contribute independently to slowed speaking rates in children more than a year following the injury.<sup>66</sup> This slowness in the expression of a story or narration produces a striking burden upon the listener.<sup>67</sup>

In children injured at younger than 5 years, a consistent pattern of poor discourse is found (Table 2.6). There appears to be no relationship between the reduced discourse ability and locus of the brain injury lesion. On the other hand, in children older than 5 years, the size and laterality of the lesion tend to produce language disorders similar to those seen in adults.<sup>68</sup> A microanalysis of discourse difficulty in very young children reveals that the impairment is most pronounced at the level of cognitive organization of the language text, whereas influence from the level of lexical sentential organization has less influence.<sup>69</sup> Furthermore, the emotional content of language may become lost as the child expresses his or her ideas. Following brain injury, children may understand emotional communication and the spontaneous externalization of emotion, but they do not express well affective signals to influence others.<sup>70</sup> These findings are consistent with the greater chance of damage to anterior brain structures rather than posterior brain structures during closed-head injury.

**Executive Disorders**

Many of the deficits among brain-injured children that arise within academic settings are related to emotional or social intelligence.<sup>71</sup> These complex human cognitive functions owe much of their underpinnings to frontal lobe function, often termed *executive function*. Deficits in executive functions occur frequently after childhood closed-head injuries. However, few studies in this area have been completed upon children. Levin and others have provided much of our current understanding about these matters. They have found that children with traumatic brain injuries display deficits on various tasks meant to assess executive functions. They used test instruments such as the *Tower of London*, which measures planning skills; *Controlled Oral Word Association*, which measures verbal fluency; and the *Wisconsin Card Sorting Test*, which measures concept formation and flexibility.<sup>83</sup>

**Intellectual Disorders**

Brain-injured children who recover from head trauma generally reveal postinjury deficits in intelligence as measured by the *WISC-R* or *WISC-III* (the Wechsler Intelligence Scales for Children). There are progressive increments in IQ improvements during recovery.<sup>10</sup> Chadwick et al.<sup>72</sup> demonstrated that children who suffer moderate to severe head trauma had mean verbal intelligence quotient (VIQ) deficits of 10 points and mean performance intelligence quotient (PIQ) deficits of 30 points when compared to matched controls. At 1-year follow-up, VIQ recovered to within 2 points of the controls. However, the PIQ remained at 11 points below controls. These youngsters were measured for 2½ years further with no noticeable improvement. As has been demonstrated

in adults, the persistent deficit in performance IQ is most likely related to task novelty and deficits in mental and motor speed.<sup>73</sup>

For severe traumatic brain injury, younger age at injury leads to minimal recovery in IQ, while recovery in older children is similar to that for adults. Children sustaining severe traumatic brain injury in early childhood may be particularly at risk for residual problems postinjury.<sup>74</sup> When one examines academic achievement scores rather than intelligence, brain-injured children show significant improvement from baseline 6 months after injury. Moreover, many children will produce average achievement test scores by 2 years after traumatic brain injury. However, 79% of severely injured children in one study had either failed a grade or required special education assistance. Thus, traditional achievement tests may be insensitive to posttraumatic academic deficits.<sup>75</sup> If children are examined for ability to solve social problems, traumatic brain-injured children show substantial deficits when compared with a comparison group of normal children.<sup>76</sup> Cattelani and colleagues<sup>77</sup> have found that while intellectual deficits and functional impairment are frequent in brain-injured children after they reach adulthood, the prevailing problems of brain-injured youngsters as adults seem to be social maladjustment and poor quality of life. College students with a history of mild, but not moderate or severe, traumatic brain injury in childhood or adolescence are intellectually unimpaired and approach their studying in a manner similar to that of their uninjured classmates. However, they are more likely to report severe distress in terms of their general personal and emotional functioning than their uninjured counterparts.<sup>79</sup> Kinsella and others have determined that one can predict which children will need special education following traumatic brain injury based on the severity of injury and by the child's neuropsychological performance measured 3 months after brain injury.<sup>80</sup>

Youngsters who have been brain-injured sustain a greater impact upon mathematics performance than upon reading and spelling skills. This may be because mathematics skill requires more attentional input than do verbal skills.<sup>57</sup> Children with traumatic brain injuries require comprehensive, multidisciplinary evaluation during rehabilitation in order to facilitate a smooth transition to home and school. Significant communication is required among rehabilitation specialists, family members, and educators to optimize the child's reentry into the academic setting.<sup>81</sup> For the youngster with a learning disability prior to brain injury, further complications arise. Moderate to severe traumatic brain injury can cause a significant additional cognitive impairment in those youngsters who have a preinjury learning disorder (Table 2.7). Even greater modification of the academic curriculum may be required in these children after a brain injury.<sup>82</sup>

Most studies of neuropsychological and intellectual deficits of brain-injured children are reported in those who have sustained a closed-head injury. Thus, the child's brain was injured by dynamic loading at the time of impact. Neuropsychological and intellectual outcome after brain injury produced by static loading of the head is much more favorable in children than those who have sustained closed-head injuries. A recent study by Prasad et al. evaluated children ranging in age from 13 to 32 months who had sustained crush head injuries by static loading. These children demonstrated a better neuropsychological outcome after brain injury than did a comparison group of children who had sustained impact trauma closed-head injury.<sup>78</sup> Common sense tells us that as a

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**TABLE 2.7**  
**Intellectual Outcomes in Traumatically Brain-Injured Children**

- Performance IQ may be permanently reduced relative to verbal IQ due to task novelty demands and reduced mental and motor processing speed.
  - The younger the child at time of injury, the less IQ recovery will be.
  - Traditional achievement tests may be insensitive to IQ-driven academic deficits.
  - Mathematics performance sustains a greater negative impact than reading or spelling skills.
  - A child who is learning-disabled prior to brain injury will sustain an additional cognitive decrement.
-

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**TABLE 2.8**  
**Frontal Lobe Disorders Following Traumatic Brain Injury**

**Disinhibited (Orbitofrontal) Syndromes**

- Behavioral disinhibition
- “Acquired sociopathy”
- Impulsive, socially inappropriate behaviors
- Lack of affective modulation

**Disorganized (Dorsolateral) Syndromes**

- Inability to integrate sensation into a whole
- Inability to switch sets with alternating paradigms
- Inflexible, perseverative responses
- Poor self-monitoring ability

**Apathetic (Mediofrontal) Syndromes**

- Can cause akinetic mutism
  - Amotivational syndrome
  - Lack of intentional behavior
  - May cause severe environmental inattention
- 

result of the many neuropsychological deficits in children described previously, one would rationally expect that closed-head-injured children will often demonstrate declines in academic performance.<sup>48</sup>

## **FRONTAL LOBE SYNDROMES**

The term *frontal lobe syndrome* comprises a variable group of different clinical syndromes produced by focal lesions of the prefrontal cortex. These have been divided into disinhibited syndromes, observed following lesions of the orbitofrontal cortex; disorganized syndromes, caused by lesions of the dorsolateral prefrontal cortex and its connections; and an apathetic syndrome, following lesions affecting the functional relationship between the anterior cingulate gyri and the supplemental motor areas<sup>84</sup> (Table 2.8). These syndromes may lead to generalized, adverse behavioral effects following frontal traumatic brain injury. Patients may suffer changes in personality ranging from extreme disinhibition to marked apathy. However, even beneficial effects have been described, and in some rare cases, frontal lobe injury produces a *de facto* salutary frontal lobotomy.<sup>85</sup>

It is often very difficult to distinguish the neurobehavioral changes of frontal lobe damage from the cognitive changes. For instance, everyday planning difficulties may be impaired following frontal traumatic brain injury, and even one’s autobiographical (incidental) memory may become somewhat defective.<sup>86</sup> Working memory impairments are extremely common due to damage to the central executive system.<sup>87</sup> The child with brain injury often is described as having had a substantial personality change, and this is a frequent observation following severe traumatic brain injury in children and adolescents. It is much less commonly diagnosed following mild to moderate brain injury in youngsters. Max et al.<sup>88</sup> recently demonstrated that, in severe traumatically brain-injured children, persistent personality change was significantly associated with the severity of injury, particularly if consciousness remained impaired for more than 100 h following trauma.

Recent positron emission tomography (PET) imaging studies using 18-fluorodeoxyglucose (18-FDG) demonstrate abnormal local cerebral metabolic rates in the midtemporal, anterior cingulate, precuneus, anterior temporal, frontal white matter, and corpus callosum brain regions following brain injury. Even mild traumatic brain injury may result in continuing behavioral deficits consistent with focal hypometabolic areas found during PET scanning.<sup>89</sup> Other research has demonstrated a close link between cognitive and behavioral disorders and decreased cortical metabolism in these anatomical areas. Functional brain imaging results suggest a predominant role for prefrontal and

cingulate dysfunction in the cognitive and behavioral disorders of patients who have sustained significant traumatic brain injury, even in the absence of focal structural lesions of the brain on MRI or computed tomography (CT) scan.<sup>90</sup> These findings also are being explored by using oxygen-15 PET and functional MRI.<sup>91</sup>

Traumatic brain injury is characterized by an extremely unpredictable recovery course. The U.S. government has recently acknowledged this and has published new rules and regulations for evaluating mental impairments. It has added guidance to the adult neurological listings regarding the evaluation of traumatic brain injury in an effort to improve the adjudication of claims involving traumatic brain injuries in adults and children.<sup>92</sup>

### **Disinhibited (Orbitofrontal) Syndromes**

The disinhibited or orbitofrontal syndrome is characterized by personality changes, amnesia with confabulation, and failure to perform on neuropsychological tests that measure inhibition. These neurobehavioral outcomes may also be related to anosmia as the olfactory fibers are often severed when acceleration–deceleration momentum causes orbitofrontal brain areas to slide across the ethmoid plate above the orbits.<sup>93,94</sup> There are rich connections between the anterior-inferior frontal lobes and the hypothalamic areas, which may also play a role in these behaviors.<sup>95</sup> Traumatic lesions in these anatomical areas probably disrupt modulatory and inhibitory mechanisms, which thus result in impulsive and socially inappropriate behaviors.<sup>96</sup>

The disinhibited behavior expressed by persons with orbitofrontal syndromes may result in extremely outlandish behavior. Social responses can become impaired to the point that the person develops “acquired sociopathy.”<sup>97,98</sup> Increases in criminal activity and aggression have been reported following injury to the frontal lobes. Disinhibition can progress such that the patient becomes stimulus-bound. That is, a stimulus in the patient’s environment has such a “pull” on the person that he cannot resist its attractiveness. The stimulus-bound components cause the individual to be distracted by irrelevant stimuli and unable to maintain directed attention. It appears that damage to the right frontal cortex is more likely to produce orbitofrontal behaviors than injury to the left hemisphere.<sup>98</sup> Orbitofrontal lesions may produce such abnormal social conduct that it becomes impossible for those with severe injuries to live independently.<sup>99</sup>

### **Disorganized (Dorsolateral) Syndromes**

Lesions in the dorsolateral prefrontal cortex are more likely to cause dysfunction on neuropsychological tests measuring executive abilities than similar lesions in the orbitofrontal or medial frontal areas. The dorsolateral syndromes are characterized by an inability to integrate sensory elements into a coherent whole. Perseveration is a common feature of disorders resulting from lesions in the dorsolateral prefrontal cortex, and the afflicted person may be extremely inflexible in her behavior, lack self-monitoring ability, and may become unable to switch sets on tasks requiring alternating responses.<sup>100,101</sup> As a result of injuries to the dorsolateral frontal brain areas, the patient generally demonstrates significant difficulties when tested with psychological instruments such as the Wisconsin Card Sorting Test or the Category Test.

### **Apathetic and Akinetic (Mediofrontal) Syndromes**

Injuries to the anterior cingulate gyri and mediofrontal lobe structures result in syndromes much more neuropsychiatrically impairing to the whole person than either the orbitofrontal or dorsolateral syndromes. Brain damage in the mediofrontal areas can lead to akinetic mutism, wherein the injured person fails to respond to environmental stimuli and remains anergic and without spontaneous speech. Unilateral lesions generally result in transitory akinesia, whereas bilateral lesions often result in a persistent apathetic amotivational syndrome. Impairment can be so great that the individual loses the ability to move, and severe contractures of the limbs may result.<sup>96,102</sup>

Disorders of intention often are more frequently associated with left hemisphere dysfunction or bilateral hemispheric lesions. The frontal lobes play a central role in the human intentional network. It is thought that mediofrontal lobe lesions may interfere with limbic connections that provide the frontal lobe with motivational information, or they cause a disconnection from the inferior parietal lobe that may deprive the frontal lobe of stored semantic and spatial information. The exact neural circuitry of intention-related mediofrontal lobe disorders is not fully elucidated.<sup>103</sup>

Mediofrontal and anterior cingulate lesions may result in patients beginning a task correctly but then being unable to complete the task. Repeated prompts from the examiner may be required in order for the patient to persist. Memory function is often impaired, but it is not clear whether this is due to poor attention, motivation, or a specific defect of working memory. In general, patients are inattentive to their environment, and even if motivation is preserved, they may be unable to organize their impulses into directed behavior. Patients with mediofrontal lesions often are unable to plan or sequence. Affect is generally noticeably diminished, and a particular flatness of personality has been described. Social relations may become dysfunctional or strained due to the person's inability to initiate or maintain a friendship.<sup>96</sup>

## **POSTTRAUMATIC SEIZURE DISORDERS**

Posttraumatic seizures are divided into early- and late-onset seizures and usually occur in two forms: (1) focal seizures with or without secondary generalization, or (2) generalized tonic-clonic seizures.<sup>104</sup> Age is a major risk factor for early posttraumatic seizures. Young children have a higher incidence of posttrauma seizures than adults with the same severity of injury.<sup>105</sup> The severity of the brain trauma is the most potent risk factor for early or late posttraumatic seizures in either adults or children. Early posttraumatic seizures are rare after mild head injuries, causing only a brief disruption of consciousness. The exception to this rule is seen in young children, who usually have higher rates of early posttraumatic seizures than adults. The risk of early posttraumatic seizures increases with prolonged unconsciousness, hematomas, skull fractures, hemorrhagic brain contusions, or focal neurologic signs.<sup>106</sup>

Early seizures are more likely to be focal with or without secondary generalization and are seen in 60 to 80% of people with early posttraumatic seizures. The remainder consists of generalized tonic-clonic seizures. Focal seizures are more likely to be seen in children or in adults who have sustained missile wounds or gunshot wounds to the head. About 10% of adults develop status epilepticus following brain injury, and about 4% of children under age 5 may have prolonged seizures.<sup>107,108</sup>

Late closed-head injury seizures (those occurring more than 7 days postinjury) have a decreasing risk as time increases following injury. The Viet Nam Head Injury Study revealed that 18% of late-onset penetrating wound seizures developed within the first month and 57% began with the first year.<sup>109</sup> Jennett has earlier reported a similar distribution.<sup>105</sup> Adults are at a higher risk for late posttraumatic seizures than are children. Penetrating head wounds are more likely to cause late posttraumatic seizures than are closed-head injuries. Brain volume loss positively correlates with increasing risk of late seizures. In nonmissile wounds, the main risk factors for seizures in a civilian population of head-injured patients are hematomas, depressed skull fracture, early seizures, and focal neurological signs.<sup>104</sup> Unlike early posttraumatic seizures, 60 to 70% of late seizures are generalized convulsive seizures with or without focal onset<sup>106</sup> (Table 2.9).

Other long-term studies of posttraumatic seizures have recently been reported. Records of the Rochester Epidemiological Project followed traumatic brain injury cases in Olmsted County, Minnesota, from 1935 to 1984. Medical records were secured from the Mayo Clinic and the other medical facilities in Olmsted County, and these formed a database for the study. Incidence rates of seizures after traumatic brain injury were compared with the base rate of idiopathic epilepsy that previously had been determined for Olmsted County. The overall excess incidence rate was calculated and compared with the base rate for idiopathic epilepsy. The excess rate was found to

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**TABLE 2.9****Posttraumatic Seizure Disorders Following Traumatic Brain Injury**

- Seizure incidence is higher in children than adults.
  - Depressed skull fracture or hemorrhagic contusions predispose to seizures.
  - Early seizures (first 7 days postinjury) tend to be focal with or without secondary generalization.
  - Late seizures are more likely to occur in adults or following penetrating missile injury.
  - Late seizures are more likely to be generalized convulsions with or without focal onset.
  - Many early seizures are nonconvulsive and thus not detected.
- 

be very low in mild traumatic brain injury — only 0.3 cases per 1000 per year — but was higher in severe traumatic brain injury, with 10 per 1000 cases per year. Only 7.2% of the brain trauma cases were classified as severe (loss of consciousness or amnesia for more than 24 h, subdural hematoma, or brain contusion). The long-term occurrence of seizures beyond the incidence rate of idiopathic epilepsy is low after moderate traumatic brain injury, but this study demonstrates a rate of 10 excess cases per 1000 brain injuries per year in cases of severe traumatic brain injury.<sup>110</sup> In a separate Olmsted County study, the relative risk of seizures was found to be 1.5 after mild injuries, 2.9 after moderate injuries, and 17.2 after severe brain injuries. Significant risk factors in this study were identified to be brain contusion with subdural hematoma, skull fracture, loss of consciousness or amnesia of 1 day or more, and an age over 65 years.<sup>111</sup>

Many seizures may be subclinical and occur covertly in the neurosurgical intensive care unit (ICU) before the patient is discharged from the hospital. One study monitored 94 patients with moderate to severe brain injuries while in the ICU. Continuous electroencephalography (EEG) monitoring began at admission and extended up to 14 days postinjury. Convulsive and nonconvulsive seizures occurred in 22% of the 94 patients, with 6 patients displaying status epilepticus. In more than half of the patients, the seizures were nonconvulsive, clinically undetected, and diagnosed on the basis of EEG studies alone. No differences in key prognostic factors such as the Glasgow Coma Scale score, early hypoxemia, or early hypotension were found between the patients with seizures and those without. The authors concluded that posttraumatic seizures occur in more than one in five patients during the first week after moderate to severe brain injury and may play a role in secondary conditions associated with primary brain injury.<sup>112</sup> This study is contrasted with another study wherein patients were not monitored by EEG, but all patients had a moderate brain injury based on a Glasgow Coma Scale of 9 to 12 after trauma. Of 106 patients, only 4.1% experienced a detectable seizure within 1 week after head injury. While this database is not entirely comparable to the Vespa et al.<sup>112</sup> database, it may further indicate that many early nonconvulsive seizures are in fact missed.<sup>113</sup>

Children must again be emphasized due to their reactions to posttraumatic seizures. Seizures can be serious complications of head injury in children because they can worsen secondary brain damage. The incidence of early posttraumatic seizures among children with a Glasgow Coma Scale below 8 was 10 times greater than among children with a Glasgow Coma Scale of 13 to 15. Sixty percent of children in this study were less than 3 years old.<sup>114</sup>

**POSTTRAUMATIC HEADACHE**

Headache is the most common neurologic symptom following minor closed-head injury. The onset of head pain often leads to other psychiatric disorders such as depression or anxiety. Moreover, chronic head pain can induce minor neuropsychological abnormalities such as impaired attention and vigilance. Just as the exact pathophysiology is unknown for migraine headaches, the exact pathophysiology of posttraumatic headache is still unknown in many cases.<sup>115</sup> The term *posttraumatic headache* is often used as a term of art rather than a specific diagnosis. Differentiation of trauma as a cause from other myriad etiologies for headache is difficult.<sup>116</sup>

The alterations in brain biochemistry are similar between posttraumatic headache associated with mild head injury and that with migraine. These alterations include increased extracellular potassium and intracellular sodium, calcium, and chloride; excessive release of excitatory amino acids; alterations in serotonin; abnormalities in catecholamines and endogenous opioids; decline in magnesium levels and increase in intracellular calcium; impaired glucose utilization; abnormalities in nitric oxide formation and function; and alterations in neuropeptides.<sup>117</sup> One study suggests that patients with posttraumatic headache have reduced regional cerebral blood flow, and regional and hemispheric blood flow asymmetries. These cerebral hemodynamic alterations are considered support for an organic basis to chronic posttraumatic headache.<sup>118</sup>

Interestingly, in adults, there is an inverse relation between the extent of head injury and the occurrence of chronic daily headache. Patients with minimal head injury may have an 80% rate of chronic daily headache, whereas those patients who suffered moderate to severe head injury report about a 27% rate of chronic daily headache, and 68% of these patients have no headaches at all. This suggests that the risk of developing posttraumatic chronic daily headache is greater for less severe head injury than for moderate to severe head injury. However, the possible reasons for this relationship are unclear.<sup>119</sup> One study of 100 children who sustained head injury revealed 83% of children had headache after brain concussion, but only 3% had migraine-type headache syndromes.<sup>120</sup>

## **NORMAL-PRESSURE HYDROCEPHALUS**

Subarachnoid hemorrhage is a common consequence of many types of head injuries. The blood products released during the hemorrhage may lead to occlusion of the subarachnoid spaces, which can result in the development of normal-pressure hydrocephalus and the onset of a dementia syndrome. Patients may present with a progressive loss of intellectual ability associated with a reduction in gait excursion and speed with urinary incontinence.<sup>121</sup> However, posttraumatic ventriculomegaly determined on CT or MRI scan may be misleading. It is difficult for the clinician to know whether he or she is dealing with increased ventricular pressure or *ex vacuo* changes in the ventricle size due to brain atrophy following trauma. Cerebral spinal fluid dynamics are quite useful to distinguish between atrophy and hydrocephalus as two possible causes of posttraumatic ventriculomegaly.<sup>122</sup>

For those patients who truly have posttraumatic hydrocephalus, ventriculoperitoneal shunts are currently the treatment of choice. The response rate is variable, but up to 70% of patients may improve. Moreover, recent technology now allows the use of programmable shunt valves to be used in the management of most patients with hydrocephalus secondary to subarachnoid hemorrhage or traumatic brain injury.<sup>123</sup>

The incidence rate of symptomatic posttraumatic hydrocephalus ranges from 0.7 to 29%. If CT scan ventriculomegaly criteria are used, the incidence has been reported to be from 30 to 86%. There are significant differences in diagnostic criteria, and thus incidence rates vary substantially depending on the study criteria used. As noted previously, it is very important to differentiate posttraumatic ventriculomegaly from ventricular enlargement secondary to atrophy, as atrophic patients are less likely to respond to shunting.<sup>125</sup> While there are numerous reports of functional gains after shunt placement for posttraumatic hydrocephalus in adults, there are only rare observations about children. However, one study demonstrated substantial cognitive improvement in a 7-year-old child following shunt placement.<sup>126</sup>

## **POSTTRAUMATIC HYPERSOMNOLENCE**

Idiopathic hypersomnia must be differentiated from several disorders of sleepiness, such as narcolepsy, sleep apnea syndromes, periodic limb movement disorder, depression, and posttraumatic hypersomnia.<sup>127</sup> The complaint of sleepiness and a positive finding on a multiple-sleep latency test may be a sequela of severe head trauma.<sup>128</sup> Some patients with posttraumatic hypersomnia may develop progressive increasing hypersomnia in the months after injury. This is in contrast to



the more usual frequent complaints of hypersomnia immediately after head injury, which progressively decline postinjury.<sup>129</sup>

The etiology of the hypersomnolence is unclear. However, rapid eye movement (REM) sleep duration is very sensitive to brain damage and is reduced in all patients who demonstrate EEG changes following brain trauma. Another consideration that must be made of the sleepy patient who has sustained brain trauma is the possibility of obstructive sleep apnea. A recent study demonstrated the incidence of sleep apnea to be 36% in patients following traumatic brain injury.<sup>130</sup> Some patients may demonstrate hypersomnia following brain injury as a consequence of chronic insomnia. Posttraumatic patients report more difficulty in initiating and maintaining sleep at night and thus have greater sleepiness during the daytime. Depression or pain from coincident physical injuries may aggravate the insomnia due to intrusion of painful impulses into sleep.<sup>131,132</sup>

## PSYCHIATRIC SYNDROMES

### INTRODUCTION

We have seen in [Chapter 1](#) that traumatic brain injury of the closed-head type preferentially causes lesions in the frontal and temporal brain structures. Within these structures lie the primary neural systems for regulation of affect and mood. Thus, it is not surprising that the more classical psychiatric syndromes might be seen following brain injury.<sup>218</sup> Neuropsychiatric syndromes following traumatic brain injury described earlier were distinguished by complex brain-behavior relationships that affect cognition or that might result in neurobehavioral syndromes. On the other hand, the more pure forms of psychiatric disturbance also occur following brain injury and do not necessarily carry the accompanying cognitive impairment of the neuropsychiatric disorders. McAllister and Green and others have reminded us that many psychiatric disorders, including mood disorders, psychotic disorders, anxiety disorders, and obsessive-compulsive disorders, occur with significantly increased frequency among those who have sustained traumatic brain injury.<sup>133,134</sup> Motor disorders and tics often are reported as an associated feature of obsessive-compulsive disorder. They have also been reported following craniocerebral trauma. They occur without evidence of structural lesions of the basal ganglia or the brain stem, but extensive bifrontal lesions have been found in at least one person demonstrating posttraumatic tics.<sup>180</sup>

### MOOD DISORDERS

Green has noted that often there is impairment in the regulation of affect following traumatic brain injury.<sup>135</sup> Following traumatic brain injury, mood disorders in general occur at a greater frequency than they do in the general population. Estimates range from 25 to 50% for major depression, 15 to 30% for dysthymia, and 9% for mania. Both depression and mania present with symptoms very similar to those seen in nontraumatically brain-injured patients. In fact, mood disorder symptoms can be well discriminated from the other neuropsychiatric and psychiatric symptoms that occur following brain injury.<sup>136</sup>

### Depression

Fedoroff et al. have reported a depression rate of approximately 25% in patients following traumatic brain injury.<sup>137</sup> Jorge et al. report similar rates.<sup>138</sup> Holsinger et al. determined that the risk of depression remains elevated for decades following head injury.<sup>219</sup> Interestingly, there seemed to be no relationship between the severity of brain injury and the development of depression or mania<sup>139</sup> until the recent data from Duke University.<sup>219</sup> In the studies by Fishman et al., traumatic brain-injured patients were found to be similar to non-brain-injured depressed patients in self-reported symptoms reported on the *Beck Depression Inventory*. However, the brain-injured group had

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**TABLE 2.10**  
**Depression Following Brain Injury**

- Level of severity predicts depression.
  - Level of social support varies inversely with depression.
  - Suicidal risk is increased by low intellect and concrete thinking.
  - Psychological dysfunction causes depression more than physical dysfunction.
  - Children are more likely to develop psychiatric illness than adults.
- 

significantly greater negative attitudes and suicidal ideas related to body image and fatigue. Moreover, they worried at a greater rate about physical problems, and they were more somatically preoccupied and sleep disordered than non-brain-disordered depressed patients.<sup>140</sup> The diagnostic criteria for mood disorder associated with a traumatic brain injury, within the psychiatric profession, is found in DSM-IV. It is described as “mood disorder due to a general medical condition.” The criteria for depressive symptoms are exactly the same as those for a major depressive episode, but they occur as a direct physiological consequence of a medical condition, in this case a traumatic brain injury.<sup>141</sup>

Numerous studies have attempted to determine predictors for those who might develop depression following a traumatic brain injury. It appears that a combination of neuroanatomic, neurochemical, and psychosocial factors is responsible for the onset and maintenance of depression following brain injury.<sup>142</sup> Evaluation of depression appears to be complex. For instance, the Beck Depression Inventory has recently been studied and found to be unsuitable for measuring depression in patients with traumatic brain injury when used as a single instrument. It was found to have a low sensitivity for discriminating depressed from nondepressed individuals.<sup>143</sup> At 12 months, McCleary et al. found that there was no significant difference in terms of frequency of depressive symptomatology among patients with poor, moderate, or good outcome following traumatic brain injury,<sup>144</sup> but this is opposite from the long-term findings of Holsinger et al.<sup>219</sup> There is one predictor that seems to correlate with the onset of depression. The level of social support available to the injured patient correlates inversely with the occurrence of depression. In other words, the greater the social support, the less the likelihood that clinical depression will develop.<sup>145</sup> A recent Spanish study suggests that suicidal risk is increased in those patients who show concrete thoughts, have problem-solving difficulties, and have few intellectual resources to cope with their surroundings. They seem particularly unable to distance themselves from the emotional aspect of the situation in which they find themselves.<sup>146</sup> Psychosocial disabilities appear to be more strongly associated to the development of a mood disorder than to the presence of physical disabilities.<sup>147</sup> There is some evidence that prior to traumatic brain injury, a significant percentage of afflicted individuals present with substance use disorders. Following traumatic brain injury, the most frequent Axis I psychiatric diagnoses were major depression and specific anxiety disorders. Comorbidity was very common, with 44% of individuals presenting with two or more Axis I diagnoses following traumatic brain injury.<sup>148</sup> Caregiver depression can occur as a consequence of serving the traumatically brain-injured person (see [Chapter 8](#)). The likelihood of caregiver stress causing depression appears to be associated with the level of adverse effects on family members.<sup>149</sup> [Table 2.10](#) lists common features of brain injury depression. Lastly, the development of new psychiatric disorders, including depression, in pediatric patients following traumatic brain injury seems to occur at a higher rate than in adult patients.<sup>150</sup>

## Mania

Mania is not as common following traumatic brain injury as is depression. However, it is more commonly found than in the general population, and it has been reported to occur in about 9% of

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**TABLE 2.11**  
**Mania Following Brain Injury**

- Mania is not as common as depression.
  - Mania of a brain injury is termed *secondary mania*.
  - Mania is usually associated with poor cognition.
  - Lesion location is not predictive of developing mania.
  - Manic features resemble those of classic mania.
- 

brain-injured patients.<sup>151</sup> Mania, when it occurs following traumatic brain injury, is generally termed *secondary mania*. As with other mood disorders following traumatic brain injury, there is no recognition of these disorders in the DSM-IV classification system other than as “mood disorder due to a general medical condition.”<sup>152</sup>

Krauthammer and Klerman defined secondary mania as a psychotic disorder due to a medical, pharmacologic, or other “organic” dysfunction. They pointed out that it must last at least 1 week and that it was characterized by elated or irritable mood and behavior. They required two or more other features: hyperactivity, pressured speech, distractibility, lack of judgment, grandiosity, flight of ideas, and decreased sleep. However, their classic article did not mention head injury or traumatic brain injury as one of the potential causes.<sup>153</sup> In 1987, the first major medical literature demonstrating that mania could occur following traumatic brain injury was published.<sup>154,155</sup> With the exception of Jorge et al.,<sup>151</sup> there are no other published substantial incidence figures for mania following traumatic brain injury. The lifetime prevalence of bipolar I disorder in the general population is 0.4 to 1.6%, and bipolar II disorder in the general population is reported with a lifetime prevalence of 0.5%.<sup>156</sup> It is important to note that traumatic brain-injured patients with mania often display concurrent problems of cognition, behavior, and physical complaints.<sup>2,157</sup>

Mayberg has proposed a limbic-cortical dysregulation as a causative factor in the etiology of mood disturbances following traumatic brain injury.<sup>158</sup> Neural signaling dysfunction may also play a role.<sup>220</sup> Mayberg’s model implicates the frontal lobes, temporal lobes, and the basal ganglia in the modulation of mood associated with traumatic brain injury. Jorge et al. followed 66 acute traumatic brain-injured patients for 1 year. They found that the presence of anterior focal brain lesions correlated significantly with the development of major depression. They also observed that anxious depression was significantly correlated with right hemispheric lesions. Depression without anxiety was significantly associated with more anteriorly placed left brain lesions.<sup>159,160</sup> The data of Jorge and others have been subanalyzed to demonstrate that patients who developed depression within the first 3 months following injury showed a significant correlation among lesions in the deep white matter, basal ganglia, brain stem, and cerebellum. No correlation between lesion location and the delayed onset of depression was found.<sup>6</sup> There are no medical reports to substantiate that lesion location following traumatic brain injury can reliably predict the development of mania. [Table 2.11](#) outlines features of mania following brain injury.

## ANXIETY DISORDER

Anxiety disorders are described in patients following traumatic brain injury, and the range in frequency varies from 11 to 70% in older studies.<sup>161,162</sup> The DSM-IV classification system lists five major anxiety disorders: generalized anxiety disorder, social anxiety disorder, posttraumatic stress disorder (PTSD), obsessive-compulsive disorder, and panic disorder. Other forms of anxiety are known as well; these would include phobic disorders and acute stress disorders. Right hemisphere brain lesions may be more often associated with anxiety disorders than left-sided lesions.<sup>160</sup>

Acute stress disorder has been reported in the mild traumatic brain injury population. Harvey and Bryant<sup>163</sup> found acute stress disorder in 14% of patients, and they believe 5% of patients had

subsyndromal acute stress disorder. Acute stress disorder has been shown to predict posttraumatic stress disorder. Eighty percent of persons who met the criteria for acute stress disorder were diagnosed with posttraumatic stress disorder 2 years later.<sup>164</sup> Bryant and Harvey have also noted that impaired consciousness at the time of trauma may reduce the frequency of traumatic memories in the initial month after trauma. However, mild traumatic brain injury does not produce a different profile of posttraumatic stress disorder than that which occurs due to psychiatric stressors other than brain injury.<sup>165</sup>

Whether posttraumatic stress disorder occurs following traumatic brain injury has been somewhat controversial in the last decade. Evidence to support the view that posttraumatic stress disorder can occur after traumatic brain injury continues to grow. However, the reported incidence of cases ranges widely from less than 1% to more than 50%.<sup>166</sup> Some authors have stated that posttraumatic stress disorder and mild traumatic brain injury are “two mutually exclusive disorders,” and that it is highly unlikely that mild traumatic brain-injured patients develop posttraumatic stress disorder symptoms.<sup>167</sup> On the other hand, posttraumatic amnesia following moderate or severe head injury may protect against recurring memories and the development of posttraumatic stress disorder. However, it has been reported that some patients with neurogenic amnesia may develop a form of posttraumatic stress disorder without flashbacks.<sup>168</sup> Bryant has noted that posttraumatic stress disorder is rarely diagnosed in patients with significant or severe head injury. However, he has reviewed cases indicating that head-injured patients with amnesia can suffer pseudomemories that are phenomenologically similar to flashbacks observed in posttraumatic stress disorder.<sup>169</sup> Moreover, Feinstein et al. have reported that even patients with posttraumatic amnesia for more than 1 week recounted intrusive and avoidant PTSD-type symptoms and psychological stress. This study did document that the shorter the posttraumatic amnesia (PTA), the greater the likelihood of PTSD symptomatology.<sup>221</sup>

The so-called postconcussion disorder may be exacerbated by anxiety associated with posttraumatic stress.<sup>170</sup> Regardless of whether a precise diagnosis of posttraumatic stress disorder can be made following traumatic brain injury, in those patients who demonstrate symptoms consistent with posttraumatic stress disorder, effective rehabilitation generally requires that these symptoms be managed.<sup>171</sup> Bryant and his colleagues have discovered that an avoidant coping style and a history of prior unemployment are significant predictors of posttraumatic stress severity following a brain injury.<sup>172</sup> Other patients following traumatic brain injury may develop difficulties with chronic pain. Posttraumatic stress disorder may play a role in maintaining high dysfunction levels from chronic pain.<sup>173</sup>

Posttraumatic stress symptomatology after childhood traumatic brain injury varies somewhat when compared with adult forms. The more severe the brain injury, the more likely the child is to develop stress symptoms. Parents of children with severe traumatic brain injury reported higher levels of posttraumatic stress disorder symptoms than did parents of children with moderate or mild traumatic brain injury at 6- and 12-month follow-up periods.<sup>174</sup> Max et al. followed children for 2 years or more and found a range of symptom expression from 68% in the first 3 months to 12% at 2 years following injury. Again, the greater the injury severity, the more likely the child was to develop posttraumatic stress disorder.<sup>175</sup> See [Table 2.12](#) for a summary of posttraumatic anxiety.

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**TABLE 2.12**  
**Anxiety Following Brain Injury**

- Acute stress disorder predicts development of PTSD.
  - PTSD without flashbacks can occur.
  - Preinjury avoidant coping style predicts development of PTSD.
  - Right hemisphere lesions may have higher anxiety rates than left.
  - Severity of injury predicts PTSD in children.
-

Obsessive-compulsive disorder is a rare outcome of traumatic brain injury. Comorbid psychiatric diagnoses are common and include posttraumatic stress disorder, anxiety with panic attacks, depression, and intermittent explosive disorder. The patterns of cognitive deficits and findings on magnetic resonance imaging suggest dysfunction of frontal and subcortical brain circuits.<sup>176</sup> However, reports in the literature are too small to draw conclusions about the incidence of obsessional disorders following traumatic brain injury.<sup>177</sup> Whereas there is evidence that classical obsessive-compulsive disorders are associated with subcortical lesions, Max et al.<sup>178</sup> have reported that frontal and temporal lesions alone may be sufficient to precipitate obsessive-compulsive disorder in the absence of clear striatal injury and that compulsivity and impulsivity may represent different psychophysiological states following traumatic brain injury. Croetzer et al. have found no support for an overlap in executive dysfunction in traumatic brain injury and obsessive-compulsive disorder.<sup>179</sup> As noted previously, tics may be seen with obsessive-compulsive features following brain injury.<sup>180</sup>

## PSYCHOTIC DISORDERS

Davison and Bagley first reported a series of psychotic patients following traumatic brain injury. They reported this to be a schizophrenia-like psychosis, and most of the patients did not have a family history of schizophrenia. Their report suggested an incidence of 0.7 to 9.8% of psychosis following brain injury.<sup>181</sup> These reports are contrasted with a recent report by Sachdev et al. indicating that head injury-related psychosis is usually paranoid-hallucinatory and subacute or chronic in its presentation. A genetic predisposition to schizophrenia and severity of injury with significant brain damage and cognitive impairment may be vulnerability factors.<sup>182</sup>

McAllister<sup>183</sup> has pointed out that certain key brain regions are damaged following traumatic brain injury. These include the dorsolateral prefrontal cortex, temporal lobe structures, basal ganglia, thalamus, and cingulate gyrus. However, in his review of the literature, McAllister states that psychotic syndromes covary with posttraumatic amnesia, mania, depression, and posttraumatic epilepsy following brain injury. Both right and left hemisphere lesions have been implicated in the genesis of psychosis, so there is no present confirmed support for laterality involved in the etiology.<sup>184,185</sup>

Lishman studied 670 soldiers with penetrating head injuries and followed them for 4 years subsequent to their injuries. He found the incidence of psychotic syndromes to be 0.7%.<sup>185</sup> Hillbom studied 415 Finnish war veterans with head injuries. He found an 8% incidence of psychosis in these men; yet only one-third of them had a psychosis similar to that seen in schizophrenia. Those with the schizophrenia-like disorders had more severe injuries than the other men and tended to preferentially have a left hemispheric injury. Temporal lobe lesions were found within 40% of his veteran group.<sup>186</sup>

A few studies have been published utilizing the *Minnesota Multiphasic Personality Inventory* (MMPI) to determine evidence of psychosis. Traumatic brain injury patients often show elevations on scale 8 or other subscales designed to assess psychosis.<sup>187–189</sup> The more severe the injury, the greater the likelihood of psychosis. Disorders of both the content and form of thinking may complicate the patient's recovery from a traumatic brain injury. While the neuropathological changes following brain injury do not represent the brain tissue changes that have been reported in schizophrenia, we cannot help but note that there is a high rate of prior traumatic brain injury in patients suffering schizophrenia.<sup>183</sup> [Table 2.13](#) outlines the core features of brain injury psychosis.

Psychotic syndromes may be seen in traumatic brain injury-related depression, mania, or posttraumatic epilepsy.<sup>155,190,191</sup> Temporal lobe injury is very common in traumatic brain injury, and we have known since Penfield and Perot's<sup>192</sup> work that direct stimulation of the temporal lobe can cause auditory hallucinations. Thus, it should not surprise us when auditory hallucinations occur within the context of a traumatic brain injury. Visual hallucinations are often a hallmark of organic brain lesions. While they are not a frequent occurrence in schizophrenia, they are not unknown in numerous organic mental conditions. Generally, any lesion that disrupts primary visual input can generate a visual hallucination. These are often referred to as Charles–Bonnet syndromes.<sup>193</sup> Organic

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**TABLE 2.13**  
**Psychosis Following Brain Injury**

- Psychosis is usually paranoid-hallucinatory.
  - Laterality of lesions does not predict psychosis.
  - Psychotic syndromes covary with amnesia, mania, depression, or epilepsy.
  - MMPI profiles often show elevations on scale 8.
  - Severity of organic delusions varies inversely with injury severity.
- 

delusions tend to be simple and less complex in association with significant dementia. However, where cognitive impairment is less, delusions may be more complex.<sup>194</sup> Delusions of being controlled tend to be related to left temporal lobe pathology, whereas misidentification syndromes such as Capgras syndrome, Fregoli's syndrome, and reduplicative paramnesia are more likely to occur with lesions in the right hemisphere.<sup>195–197</sup> It has been suggested that frontal lobe dysfunction, which is of course very common in traumatic brain injury, may play a key role in the maintenance or cause of delusional behavior.<sup>198</sup>

### **PERSONALITY CHANGES FOLLOWING TRAUMATIC BRAIN INJURY**

Following traumatic brain injury, families and friends will report that personality change is the most significant problem, whether they are asked at 1, 5, or 15 years postinjury.<sup>199</sup> In most instances, alterations of personality are in fact exaggerations of preinjury personality traits. Attempts to measure personality changes following traumatic brain injury have not been very successful. Quantitative methods of evaluating self awareness by having the brain-injured patient make self-assessments have serious shortcomings.<sup>200</sup> When relatives' reports are used to measure personality change, the correlations are strongest for stress and role changes associated with caring for the injured person.<sup>201</sup> Attempts have been made to study the stability of normal personality traits after traumatic brain injury, and these have proved difficult to determine as well.<sup>202</sup>

As noted in the “Neuropsychiatric Syndromes” section, frontal lobe syndromes are commonly seen following traumatic brain injury. Frontal lobe control of personality is often an issue. Some individuals are described as developing “acquired sociopathy” following traumatic brain injury, whereas others develop syndromes similar to borderline personality disorder.<sup>203</sup> Focal brain injuries have been known to cause severe outlandish behavior, as was demonstrated when Phineas Gage accidentally blew a tamping rod through his frontal brain.<sup>204</sup> The innate sense of self may be damaged following traumatic brain injury.<sup>199</sup> Judgment is often significantly impaired and may relate both to linguistic and nonlinguistic aspects of language and the inability of the patient to monitor his or her linguistic and expressive behavior.<sup>205,206</sup>

Max et al. have studied personality changes in children following traumatic brain injury. In a sample of 37 severely traumatically brain-injured children, the labile subtype of personality change was the most common and was seen in 49% of these children. It was followed in frequency by an aggressive and disinhibited subtype of personality change in 38% of children. The remaining children were either apathetic or paranoid at a 14% and 5% rate, respectively. Perseveration was seen in one-third of the children.<sup>207</sup> A further analysis of these data revealed that approximately 40% of consecutively hospitalized severe traumatic brain-injured children had ongoing persistent personality change at 2 years postinjury. Another 20% of these youngsters had a history of a more transient personality change that remitted. Personality changes were found at a rate of 5% in mild–moderate traumatic brain injury, but it was always transient. These findings suggest that personality change is a frequent diagnosis following severe traumatic brain injury in children and adolescents, but it is much less common following mild–moderate traumatic brain injury.<sup>208</sup> [Table 2.14](#) describes personality changes following brain trauma.

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**TABLE 2.14**  
**Personality Changes from Brain Injury**

- Personality changes are cited by families as the most significant changes.
  - Measurement of personality changes is difficult at best.
  - Development of sociopathic or borderline traits may occur.
  - Children are more likely to show labile subtype of personality change.
  - Poor judgment may relate to linguistic and nonlinguistic impairment.
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## **AGGRESSION AND ANGER**

Families and caregivers of those who have sustained a traumatic brain injury point out that the major stress they experience is a result of irritability, agitation, and aggressive behavior from the brain injury victim.<sup>209</sup> Complex syndromes containing aggression are seen frequently in both the acute and chronic stages following traumatic brain injury. The prevalence has been reported to be between 5 and 70%.<sup>210</sup> Rao and Lyketsos have suggested that these disorders should be called behavior dyscontrol disorder, major or minor variant.<sup>2</sup> Agitation is most frequently seen in the first 2 weeks of hospitalization following brain injury and generally resolves within that time. Restlessness is seen in the subacute phase of recovery and generally appears after 2 months and may persist for 4 to 6 weeks. Agitated behavior is reported in one-third to two-thirds of patients within the acute recovery period.<sup>211</sup>

Aggressive behavior following traumatic brain injury is thought to be caused by dysfunction within the hypothalamus, limbic system, and prefrontal cortex. The Viet Nam Head Injury Study has provided some support for localization patterns in aggression. A comparison was made between two subgroups from this study and included 279 veterans with penetrating brain injury and 85 age-matched control veterans who spent equivalent time in Viet Nam but did not sustain a head injury during combat. Those veterans with ventromedial frontal lobe injuries were given the highest rating for violence by relatives and friends, while veterans with orbitofrontal lesions were reported to be aggressive but had the least amount of insight into their aggression. There was no relationship found between the size of the brain injury, seizures, and aggression.<sup>212</sup>

Investigations have been made to determine the impact of traumatic brain injury upon domestic violence. In one study, batterers differed from nonbatterers across several cognitive domains, including executive, learning, memory, and verbal functioning. Batterers were reliably discriminated from nonbatterers based on three neuropsychological tasks: *Digit Symbol*, *Recognition Memory Test-Words*, and *Wisconsin Card Sorting Test*. Neuropsychological performance was the strongest correlate with domestic violence of all clinical variables measured. However, the inclusion of two other variables, severity of emotional distress and history of head injury, together with the previously noted neuropsychological indices, provided the strongest correlation with those who battered. These findings suggest that current cognitive state and a prior brain injury may contribute, along with coexisting emotional distress, to a propensity for domestic violence.<sup>213</sup>

Prior studies have suggested that aggressiveness, substance abuse, and criminality contribute to poor outcomes following brain injury. Kreutzer et al. studied 327 patients varying in severity of traumatic brain injury. They reviewed alcohol use patterns, arrest histories, behavioral characteristics, and psychiatric treatment histories. Relative to the uninjured population, their analysis revealed a relatively high incidence of heavy alcohol consumption both before and after injury, particularly among patients with a history of arrest. In addition, history of arrest was associated with a greater likelihood of having been psychiatrically treated. Aggressive behaviors were quite high in this group.<sup>214</sup> A recent study indicated that substance abuse history proved to be a strong predictor of long-term outcomes, while a brain injury as the result of violence was a less influential predictor. Almost 80% of persons suffering brain injury from violence-related causes had a history of substance

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**TABLE 2.15**  
**Brain-Injury-Induced Aggression and Anger**

- Aggression is more likely with frontal cortex injuries.
  - Brain-injured domestic batterers show neuropsychological impairment.
  - Aggressive behaviors are high in those with preinjury substance abuse.
  - Brain injury is a risk factor for being murdered.
  - Aggressive and violent behaviors impair reintegration.
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abuse.<sup>215</sup> A Swedish study of 1739 homicides between 1978 and 1994 revealed that traumatic brain injury, in both men and women, is a risk factor for being murdered. It is not clear if the brain injury marks risk-taking behavior in general or if it may cause provocative behavior that increases the risk of being murdered.<sup>216</sup> See [Table 2.15](#) for a review of brain-injury-induced aggression.

Anderson and Silver have concluded that aggressive and violent behaviors are wide ranging and may result from traumatic brain injury, among other causes. Moreover, they point out that disruptive behavior is often the largest barrier to reintegration into the community for those patients who have suffered a traumatic brain injury. It is among the most distressing of symptoms that the caregiving families must confront. While many neurobiological and neuropathological factors may lead to aggression following brain injury, lesions that involve the temporal lobes may be more frequently associated with aggression. Dyscontrol is often associated with disruption of frontal lobe function as well. The modulation of aggressive impulses may result from disruption of neurotransmitter pathways and may lower the threshold for expression of violent impulses.<sup>217</sup>

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# 3 Gathering the Neuropsychiatric History Following Brain Trauma

## INTRODUCTION

The body of information to be gathered from the interview can be termed part of the neuropsychiatric database. It has a long and hallowed presence within the practice of medicine regardless of the orientation of the practitioner. The gathering of an appropriate history has been the *sine qua non* of the practice of medicine since at least the time of Hippocrates.<sup>129</sup> Benjamin Rush first provided the U.S. with important methods for medical inquiry of the mind in 1812.<sup>130</sup> Gowers provided a manual for exploring diseases of the nervous system in the late 1880s.<sup>131</sup> Thus, the art of a neuropsychiatric history is first based upon fundamental principles of history taking in a general medical examination; it has developed further as an amalgamation of a fundamental psychiatric and neurological historical examination of the patient. In neuropsychiatry, attention to cerebral organization must not be matched by neglect of psychosocial variables, for these may have a substantial impact on symptom expression, impairment, and disability.<sup>132</sup> This attention to cerebral organization and psychosocial issues is required prominently within the evaluation of the traumatic brain injury. It is important for the skilled practitioner undertaking the evaluation of a patient who has sustained a traumatic brain injury to not allow the important aspect of history taking to lose its relevance vis-à-vis the remarkable advances that have been made in structural and functional brain imaging and cognitive neuropsychology.

## TAKING THE ADULT BRAIN INJURY HISTORY

### POSTTRAUMA SYMPTOMS AND TREATMENT

Classically, in medical practice the physician asks the patient for a chief complaint and then takes the history of the present illness. The neuropsychiatric history taking from an adult following traumatic brain injury is no different. However, the physician must first determine the level of competency of the person to give her own history. Many persons with brain injury are amnesic for the brain trauma (as noted in [Chapter 2](#)) or, due to lingering cognitive deficits, the person has limited new learning ability and does not self-monitor changes in her behavior. [Table 3.1](#) lists a simple schema for inquiring about posttraumatic symptoms following brain injury. The elements within [Table 3.1](#) are the mental functions that are questioned during the neuropsychiatric examination and the treatments used by the patient at that time. The more complex issues of posttraumatic physical impairments such as hemiparesis, blindness, or orthopedic dysfunction are covered in the “Review of Systems” section.

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**TABLE 3.1**  
**The Adult Neuropsychiatric Symptom History**  
**Following Brain Trauma**

- Chief complaint
  - Are there problems with:
    - Attention
    - Speech and language
    - Memory or orientation
    - Visuospatial or constructional ability
    - Executive function
    - Affect and mood
    - Thought processing or perception
    - Risk to self or others
  - What is the current treatment?
    - Antidepressants
    - Antiepileptic drugs
    - Lithium salts
    - Neuroleptic drugs
    - Anxiolytics
    - Cholinergics
    - Psychostimulants
    - Dopamine agonists
    - Cognitive rehabilitation
    - Individual psychotherapy
    - Family psychotherapy
- 

## Attention

Individuals should be asked if they have noted any fluctuating awareness or difficulty paying attention to what they hear or what they see. Traumatic brain injury may preferentially interfere with visual attention more so than auditory attention, or the reverse may be true. Depending upon the age and sophistication of the adult, probing questions about auditory attention can be explored. If the patient is a college student, does the patient have difficulty paying attention to oral lectures? For a working person who uses a computer or reads, the individual should be asked whether he finds it difficult to maintain visual attention while reading or if he loses his place when using a computer. However, asking a person about computer skills in an attempt to determine attention can be misleading. Much of human computer operations are served by procedural memory rather than attentional or factual memory. As has been previously noted in [Chapter 2](#), procedural memory is usually spared in traumatic brain injury; it is declarative or factual memory that is generally impaired.

The simplest way to explore attentional deficits in the neuropsychiatric history is first to determine something about the person's lifestyle and then focus questions specifically on auditory, visual, and tactile systems to determine if the individual has noted changes. It also is important to determine whether attention is varying due to hypersomnolence. Hypersomnolence is a frequent outcome of traumatic brain injury, and if the person is chronically sleepy, he will thereby have an apparent reduction in attention.<sup>1</sup>

One way to approach the history of attention is to be aware that attention is both a point source phenomenon and a longitudinal phenomenon. There is instantaneous attention, there is concentration (vigilance), and there is tracking attention either visually or auditorially. In practical terms, they are difficult to separate. Visual tracking inattention or point source inattention appears as either visual distractibility or interference with following movement. It is much easier to determine if the

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**TABLE 3.2**  
**Screening Questions for Attentional Deficits**

- Can you pay attention while others are speaking?
  - Can you concentrate when reading a magazine or book?
  - Can you repeatedly point and click when using the computer?
  - Are others speaking too fast for you?
  - Do others say you repeat yourself?
  - Can you follow the story line in a television program or movie?
- 

person has difficulty with auditory vigilance by asking if he has difficulty following the story line on television or particularly while listening to the radio.

Another important question to ask the brain-injured adult is whether information processing is slowed. For instance, a simple question such as “Are you thinking slower than you used to?” can be followed with queries as to whether the person finds that people talk faster than they did prior to injury or whether the environment seems to be moving much faster than it did before the brain injury. The sensation that the environment is moving faster than the person is not uncommon if information processing has been slowed. This sensation exists because the individual cannot keep up with the ordinary pace of the attentional demands placed by the environment. Moreover, the person can be asked if she takes longer to react or if performance has slipped in tasks that require speed.<sup>2,3</sup> The examiner may not be able to differentiate the exact causation of apparent attentional problems by history alone. Tromp and Mulder’s studies<sup>3</sup> suggest that memory activation is a critical problem for many brain-injured patients, and this may appear to both the patient and the examiner as slowness. More critical evaluation neuropsychologically may be required to differentiate attentional difficulties from memory activation impairment. [Table 3.2](#) provides some questions to help determine the presence of inattention.

Disturbances of the attentional matrix may present as symptoms of impersistence, perseveration, distractibility, and an inability to inhibit immediate but inappropriate responses.<sup>4</sup> Sometimes a more accurate history of these deficits is obtained from collateral sources, such as family or employers. Many times, the patient also may have a component of neglect and be unable to properly self-monitor to answer the examiner’s questions. A brain-injured person may not be aware of perseveration and repeating herself incessantly to family members. She may also not be aware of her level of distractibility without observant response from an objective person. Inappropriate verbal responses may go undetected by the brain-injured individual. As we shall see later, if the examiner is concerned that attentional deficits such as response inhibition are present, these may be measured using the *Stroop* procedure, the *Trail-Making Test*, and similar test instruments.<sup>5,6</sup>

## Speech and Language

The examiner should ask the brain-injured person whether he has noticed difficulty articulating words or pronouncing words. Articulation is best determined by listening to speech, and fluency of language is likewise determined best by the examiner’s focused listening. However, many times following brain injury, subjects may be aware that they cannot form or pronounce words the way they used to. Simple questions may be useful here such as asking the person if he has difficulty saying the “Pledge of Allegiance” or repeating prayers in his place of worship. Detailed assessment of speech and language can be obtained by the collateral interview sources noted next.

Several questions can be asked regarding word finding. How difficult is it for the person to find words when she wishes to speak with someone? Does she use the wrong word or misplace an initial sound in a word? Does she confuse the meaning of words? Does the individual find that she speaks slower or with more effort than she did prior to the injury? As noted in [Chapter 4](#), the examiner will be on notice as to the fluency of the individual merely by speaking with her. Speech

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**TABLE 3.3**  
**Screening Questions for Language Deficits**

- Can you find words while speaking?
  - Can you name common objects?
  - Has your ability to communicate changed?
  - Have others said you speak differently?
  - Can you repeat prayers or songs?
- 

is “fluent” if the phrase length and melody are appropriate and “nonfluent” if phrase length is less than four words and the speech is halting or dysarthric. Anterior brain lesions in the dominant hemisphere are more likely to result in nonfluent language, whereas posterior lesions tend to result in fluent but paraphasic speech (phonemic misstatements or misuse of word meaning).

Detection of language errors in locations other than the dominant cerebral hemisphere by history alone may be difficult. For instance, injury in the medial portions of the frontal lobes can affect the initiation and maintenance of speech. These also play a significant role in attentional and emotional influences upon speech. Damage in these areas will not cause a pure language disorder, but rather varying degrees of difficulty in the initiation of speech, or it may even produce mutism.<sup>7</sup> Since the person’s drive to communicate is no longer present, he may in fact not be able to tell the examiner of the reduction in speech rate or of the difficulty he has in producing speech. [Table 3.3](#) outlines language-deficit screening questions.

It is important to discuss with the patient whether she has had a change in her ability to communicate with others, particularly in her ability to obtain meaning from the communication of others. As we shall see in [Chapters 4](#) and [6](#), the dominant hemisphere controls the expression and reception of symbolic language, but the emotional coloring (affective prosody) is supplied by the nondominant hemisphere. In most individuals, the nondominant side is within the right hemisphere, and the prosody of language and the kinesics of language constitute the paralinguistic portions of language reception and expression. It may be useful to ask the patient if anyone has noticed a change in the pitch, intonation, tempo, stresses, cadence, and loudness of her language since brain injury (how it sounds to others).<sup>8</sup> Kinesics refers to the limb, truncal, and facial movements that accompany language output. The gesturing and facial expression associated with language modulates the verbal message being communicated.<sup>9</sup> Since impaired communication is one of the major variables that will determine whether a brain-injured patient can return to functional life or to the workplace, the examiner should ask whether or not the patient has noted difficulties expressing ideas and whether it has been brought to her attention that there has been a change in facial expression. Oftentimes, the “flatness” described in brain-injured patients is in fact an element of aprosodia or dysprosody (an impairment of the production, comprehension, and repetition of affective prosody without disrupting the propositional elements of language).<sup>10</sup> The detection of aprosodia or dysprosody requires a significant amount of skill; more discussion about this matter is presented in [Chapters 4](#) and [6](#).

### **Memory and Orientation**

Most patients who have sustained even a mild traumatic brain injury will complain of memory disturbance.<sup>11</sup> When taking the history from a patient who may have sustained a memory disorder, it is important to remember that the patient may not be fully competent to give her own historical data about the presence or lack thereof of memory deficits. Collateral information will be very important in this regard. However, the patient should be asked directly if she has noticed any changes in her ability to remember, with simple questions such as the following:

- Have you had to keep lists?
- Do you forget what others tell you?

Do you have difficulty completing your study assignments (if a student)?  
Can you remember what you read or watch on television?  
Do you have difficulty keeping up with current events from the news?  
Have others commented to you that your memory is poor?

The examiner should be aware that limbic-dependent memory is primarily for factual events, and it is either episodic, based upon what goes on around the individual, or declarative, semantic, explicit, and associated with the meaning of facts. On the other hand, limbic-independent memory is primarily nondeclarative, implicit, or procedural. This form of memory incorporates the skills and habits that we develop such as driving, playing golf, or cooking. However, of those who cook, it is often important to ask if the individual can remember recipes, as this would be a declarative or factual portion of memory rather than procedural. The aspects of turning on a stove, watching a pot boil, or monitoring a roast while it cooks are aspects of procedural memory.<sup>12,13</sup>

Questions of orientation are fairly simple and straightforward. Much of this is covered in more detail in [Chapter 4](#) in terms of performing the mental status examination. However, with a seriously injured person, it is probably wise to determine orientation fairly quickly into the interview so that the examiner understands what modifications may be necessary in order to take a history and whether it will be necessary to quickly move to taking required information from collateral sources.

It often is useful to subtype factual or declarative memory into personal events and general facts. Episodic memory refers to specific events in one's biography, and these events are embedded in time and place. Episodic memory is actively *remembered*, while semantic information is only *known*.<sup>14</sup> When taking the history from a patient who may have an impairment of episodic memory, simple questions to determine whether the person is longitudinally storing memories as they occur should be asked. For instance, "How did you get to my office today?" may enable the examiner to determine if the individual is processing events as they occur. Another simple request might be "Tell me about the last birthday party you attended." The examiner will be able to develop other simple biographical questions to assist with the determination of episodic memory. To question the individual about semantic memory requires only the simplest of questions and may be included in the mental state examination. A quick review of possible semantic memory impairments can be obtained by asking the following: Who is the president of the U.S.? What is the capital of this state? Who is the mayor of your town? With regard to long-term memory, Tulving and Markowitch<sup>12</sup> hold that episodic memory is an extension of semantic memory. They view these memories as being content-dependent subdivisions. Semantic memory is used to "know the present," while episodic memory is used for "remembering the past."<sup>15</sup>

When asking the person about procedural memory, generally the patient will not admit to problems unless the brain injury has been quite severe and within specific areas of brain function. Even as early as 1912, Kurt Schneider<sup>16</sup> noted that amnesic persons could learn to solve jigsaw puzzles even though they could not remember new episodes. The famous memory case, H.M., learned new motor skills without significant difficulty such as those involved in a rotor pursuit.<sup>17</sup> The difference seems to be in whether the traumatically brain-injured patient has received injury in the limbic areas anterior in the brain or in the basal ganglia areas deep in the brain. Patients with amnesias caused by limbic lesions can usually acquire perceptual, motor, and strategy skills,<sup>18</sup> whereas those persons who suffer lesions of the basal ganglia are generally severely impaired in such abilities.<sup>19</sup> Memory screening questions are found in [Table 3.4](#).

## Visuospatial and Constructional History

This section of inquiry focuses upon disorders of complex visual processing.<sup>20</sup> These disorders are very complicated. They can be screened and evaluated based on history and neurological examination, but a comprehensive appraisal of defects and quantification of these defects requires neuropsychologic, neuroimaging, or neuro-ophthalmologic evaluations. There are some very simple

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**TABLE 3.4**  
**Screening Questions for Memory Deficits**

- Can you keep track of dates and important events?
  - Do you need to keep lists or a journal?
  - Can you remember what you read or see on television?
  - How did you get here today?
  - Tell me how you will return to your home.
  - Have you lost memory for any skills?
- 

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**TABLE 3.5**  
**Screening Questions for Visuospatial/Constructional Deficits**

- Can you find your way alone to an office within a building?
  - Can you name the color of a banana, blood, or a crow?
  - Can you keep your handwriting on a line?
  - Can you draw objects?
  - Can you describe the routes you will take to return home?
- 

historical questions that may elicit information suggesting that the brain injury victim has either a visuospatial or a constructional difficulty.

Topographic orientation is simple to evaluate historically. Defects of this nature generally reflect impairments in visuospatial memory. Simple questions to the patient such as her ability to locate a public building in a city, find her room at home, or describe by means of a map how to get to a specific place are simple screening techniques that can be incorporated into the historical evaluation. Geographic disorientation is seen frequently following moderate to severe head injury. It occurs in patients with both bilateral and unilateral posterior cerebral lesions.<sup>21,22</sup> While taking the history, the patient should be asked if she has noticed differences in her ability to perceive colors, name colors, or associate colors with specific items, such as the color of blood or the color of a banana.

Constructional ability and visuospatial ability are sometimes judged using the directional orientation of lines. This will be covered in more detail in [Chapter 6](#). The patient may be asked if he has difficulty keeping his handwriting on the line or if he can write well without using lined paper. The clock-writing test noted in [Chapter 4](#) is useful for clinically determining difficulties with visuospatial orientation and judgment. In the *Judgment of Line Orientation Test*,<sup>23</sup> the patient is presented with pairs of lines that have been placed at a given angle. The person is requested to point to similarly oriented lines in a different array. If the individual cannot perform this test well, the results are strongly correlated with lesions in the right posterior brain.<sup>24</sup> Visuospatial screening questions are noted in [Table 3.5](#).

The term *constructional apraxia* was introduced by Kleist.<sup>25</sup> Today, this is more properly referred to as a disturbance of *visuoconstructive ability* or *constructional ability*, rather than as an apraxia. The patient should be asked if she has noticed any differences in her ability to draw two-dimensional objects. The examiner may have a clue to ask about visuoconstructive ability as defects caused by left hemisphere lesions tend to be associated with dysphasias as well. The dysphasia is generally fluent in nature and due to a posterior injury rather than an anterior brain injury.

### **Executive Function History**

Lezak describes executive functions as conceptualized by four components: (1) volition, (2) planning, (3) purposive action, and (4) effective performance.<sup>26</sup> On the other hand, Stuss and Benson

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**TABLE 3.6**  
**Screening Questions for Executive Deficits**

- Could you plan a party if you wished?
  - Has your motivation or interest changed?
  - Can you control aggressive or angry impulses?
  - Are you as creative as you used to be?
  - Are you less able to control your emotions?
  - Do you have difficulty controlling your sexual impulses?
- 

describe the behavioral characteristics of executive function as at least the following: anticipation, goal selection, preplanning, monitoring, and use of feedback.<sup>27</sup> Regardless of which orientation one follows in terms of what exactly constitutes executive function, it is obviously simple to ask questions about these areas of patient function. However, since executive dysfunction generally is always associated with frontal lobe disorders, it must be remembered that the patient may be either unaware of her deficits or unable to sufficiently self-monitor to provide accurate history. Again, collateral sources of information may be necessary.

Volition or drive can be assessed historically by asking the patient if he has noticed any changes in his motivation or ability to stay interested. Planning may be assessed by inquiry into the patient's postinjury ability to plan a dinner, plan school preparation for his child, plan a curriculum if a student, or plan something as simple as a game. What Lezak calls "effective performance" is described by Stuss and Benson<sup>27</sup> as self-monitoring and use of feedback. The brain-injured patient may be able to tell the examiner about difficulties monitoring impulsiveness, aggressive impulses, or making course corrections when he determines that a planned event is not going according to the plan. Further inquiry can be made as to how the individual handles novel situations that require new solutions. Many individuals with executive dysfunction are unable to make moment-to-moment adjustments necessary for dealing with novel social situations. See [Table 3.6](#) for screening questions for executive dysfunction.

[Chapter 2](#) delineated the types of clinical frontal lobe syndromes often seen following traumatic brain injury. In terms of gathering historical information, there are two basic types of frontal syndromes. In one type, the loss of creativity, initiative, and curiosity predominates, and the patient is apathetic and emotionally blunted. Neurologists call this the syndrome of *frontal abulia*. The second type causes the patient to be impulsive and without judgment, insight, or foresight. This is a syndrome of *frontal disinhibition*.<sup>28</sup> Relatives may more accurately provide the historical context to differentiate these two major frontal lobe syndromes. Oftentimes, however, patients can report that they have no interest and are apathetic, whereas others are able to describe anger outbursts, impulsive sexual activity, and inability to make correct judgments.

### **Obtaining the History of Affective and Mood Changes**

[Chapter 4](#) delineates in detail the significant differences between affect and mood. For purposes of history taking, the focus is upon changes in mood since mood is an internally represented feeling state, whereas affect carries observable behavioral components. It is not unusual for patients following traumatic brain injury to develop "emotional incontinence" wherein they will "cry at the drop of a hat" or have rapid fluctuations in the internal perception of happiness. Some of these mood changes may be related to injury to the amygdala, which lies within the anterior temporal lobe. The amygdala plays a critical role in the channeling of drive and emotion, which was graphically demonstrated by Downer's experiments in monkeys.<sup>29</sup> The human amygdala plays a crucial role in modulating the neural impact of sensory stimuli. An emotional valence is placed upon a sensory stimulus by the amygdala. Damage to the amygdala can produce states of hypoe-

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**TABLE 3.7**  
**Screening Questions for Affective or**  
**Mood Changes**

- Has your mood changed since your injury?
  - Do you ever feel sad or possibly too happy?
  - Have you been nervous, easily startled, or tense?
  - Do you relive the injury in your mind?
  - Do you have nightmares about the injury?
  - Do certain events cause you to relive the injury?
- 

motionality in humans.<sup>30</sup> Therefore, it is not unusual for the brain-injured adult to report changes in both the control of emotions and the internal perception of mood.

Thus, it is appropriate to ask the patient if he has noticed difficulty controlling his emotions or if he has felt depressed. Particularly if the patient has suffered preferential injury to the left hemisphere, these questions should be pursued, as it is now well recognized that left hemisphere injury is more likely to result in depression than right hemisphere injury.<sup>28</sup> The examiner will probably note in a person reporting depression following traumatic brain injury that the usual diurnal variation of mood commonly associated with major depression is generally not present; if a reduction in mood is reported by the patient, it is usually fairly consistent throughout the day. On the other hand, it is not unusual to get a report of depression associated with lethargy and increased need for sleep following traumatic brain injury. This more commonly follows the traditional drop in mood seen in bipolar patients, but it is without the cyclical variation of mood associated with bipolar illness. Recent longitudinal studies of brain injuries caused by military injury indicate that chronic mood changes following traumatic brain injury may persist for decades.<sup>31</sup> Also, while questioning the patient regarding alterations of mood, it is important for the examiner to remember that mood changes rarely occur coincidentally with a traumatic brain injury. They are more likely to occur many weeks and even months posttrauma. As a result of this clinical fact, some physicians may fail to see the connection between the drop in mood and the original traumatic brain injury and attempt to correlate the mood change with adversity in the person's life due to the brain injury. While altered life circumstances following brain injury can be a necessary cause for inducing depression following traumatic brain injury, they are rarely if ever a fully sufficient cause, and the brain trauma itself plays a primary role in the induction of depression.

As is standard in any good psychiatric history, it is best after listening to open-ended discourse from the patient to then ask some direct screening questions regarding mood. One simple format is to ask the person if she has noticed any change in her mood or how she feels. Asking her if she has been uncomfortable, tense, overly vigilant, or sad is appropriate. It is particularly important to look for dysphoric mood, and the patient should be asked if she has noticed unpleasant or negative mood states or a sense of feeling low or blue. On the other hand, since mood can be discordant between observed affects, it is also important to ask her if she has noted elevations in mood, increased intensity of feelings, or feelings of aggression, anger, irritability, or anxiety. [Table 3.7](#) lists common inquiries of affective and mood changes.

It has been well recognized that depression can complicate the clinical presentation of a brain disorder.<sup>32</sup> When questioning the person depressed following brain injury, it must be remembered that a number of studies have not found significant memory impairments in non-brain-injured depressed patients.<sup>33</sup> However, depression very likely coexists with memory disturbance in the traumatically brain-injured. Therefore, it will not be unusual, when asking about mood symptoms of a brain-injured patient, for him also to complain of memory and other cognitive disorders.



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**TABLE 3.8**  
**Screening Questions for Changes in Thinking**

- Do you ever hear voices or see things others cannot see?
  - Do you ever feel you would be better off dead?
  - Have you made plans to take your life?
  - Has your ability to think changed in any way?
  - Can you connect ideas in your head?
- 

### **Taking the History of Thought Processing**

Questioning a person about how she thinks is difficult at best. If brain dysfunction or a disease is of sufficient proportions to interfere with thinking, oftentimes the patient's self-monitoring skills are so poor that she is not aware she has a thinking disturbance. For those physicians familiar with thought disorders evaluated in general psychiatry, obviously patients would not be delusional if they were able to sort out the reality of their own thoughts. Therefore, taking the history from a brain-injured patient who may have disturbances of thought must be done carefully. It is often useful to ask the patient if she has noticed that she has been addled, has found it difficult to think, has noticed that her ideas do not connect, or cannot find thoughts when scanning for them in her mind.

Thought disorders are a diverse group of mental abnormalities. They usually feature abnormal content of thinking and disturbed processing of thoughts. They generally occur within a setting of inadequate self-monitoring or poor mental control following traumatic brain injury. The thought disorders that occur following traumatic brain injury probably arise from structurally or functionally based neurologic dysfunction and may comprise several different neural systems.<sup>34</sup> We infer when examining patients that, during normal conversation, the speech output is consistent with the underlying thoughts.<sup>35</sup> It is probably important, therefore, to discuss the patient's thinking with family members. Observations by others are more likely to be objective when determining by history whether a thinking disturbance is present. [Table 3.8](#) outlines questions used to gather history of thinking.

### **Questioning the Patient about Risk to Self or Others**

Suicide attempts are almost unheard-of in an acutely brain-injured person. Moreover, there is no substantial medical evidence that suicide risk *de novo* is increased following acute traumatic brain injury. However, these statements may not hold in persons who had bipolar affective disorder, major depression, or some other disorder of mood prior to the head injury. Therefore, if the past neuropsychiatric history contains elements of preinjury mood disorder or prior attempts at self-harm or harm to others, extra inquiry about risk of danger to self or others must be undertaken. Moreover, suicide risk increases in the chronic phase of traumatic brain injury.<sup>127,128</sup>

Suicidal ideation varies in its intensity and in its context. Some patients passively think of suicide (I wish I would not wake up) or have active thoughts of suicide (I will save up my pills to take so I will not wake up). Others may consider or think about killing themselves and have no specific plan to do so. The examiner must carefully distinguish between active or passive thoughts of suicide and if the development of a specific plan to carry out these acts is in place.

Inexperienced or poorly trained physicians erroneously avoid discussing suicidal ideation because they are concerned with "putting the thought" into the mind of the patient. There is no medical evidence that asking about suicide increases risk. In fact, to the contrary, asking about suicide may reduce risk. Therefore, questions such as the following should be framed to determine either active or passive suicidal thoughts:

- Has your status in life changed so much that you wish you were dead?
- Are you unable to get pleasure from life since your injury?

Do you feel like your life is no longer worth living?  
Do you ever wish that you would die or that you would not wake up in the morning?  
Have you ever made a plan as to how you would take your life?

Obviously, the reader can frame many other approaches to ask a person about self-destructive ideas.

Another aspect of self-destructive behavior that should be considered is the borderline personality disordered patient or the impulsive antisocial patient who later sustains a traumatic brain injury. Inquiries should be made as to whether he has increased thoughts of cutting himself, harming others, burning himself, or causing self-mutilation. Particularly those patients who have sustained orbitofrontal brain injuries (see [Chapter 2](#)) may be at risk for significant disinhibition, accelerating the level of their premorbid brain injury behaviors.

## **History of Behavioral Treatment Following Traumatic Brain Injury**

[Chapter 2](#) considered the more common psychiatric and neuropsychiatric syndromes following brain injury. Since the neuropsychiatric evaluation of traumatic brain injury, in most instances, occurs postrehabilitation, the patient probably will be medicated at the time of the neuropsychiatric screening examination. Thus, careful inquiry about treatment strategies employed with the patient is of importance. As a general rule, posttrauma brain-injured patients receive antidepressants, cognitive enhancers, antiepileptic drugs for mood regulation, or atypical neuroleptics. Some individuals will have posttrauma-induced aggression as a result of their brain injuries, and atypical antipsychotic agents and beta-blockers have been used successfully with those patients.<sup>36</sup> It also may be relevant to ask the patient, or the patient's family, if experimental neuroprotective drugs were used while the person was within the neurosurgical intensive care unit (ICU), if such treatment played a role in recovery. Multiple protocols exist for such drugs, and many patients are now receiving these, either within experimental protocols or as a matter of course during neurosurgical treatment.<sup>37-40</sup> Risperidone has been used to treat psychosis following traumatic brain injury, and it, of the novel antipsychotic agents, has probably been studied the most extensively to date.<sup>41,42</sup> On theoretical grounds, all of the atypical antipsychotic agents may be more effective than traditional antipsychotic drugs in aggressive and violent populations following brain injury.<sup>36</sup> Moreover, they are preferred rather than typical neuroleptics, as brain injury is a risk factor for developing tardive dyskinesia.

The patient should be questioned as to any possible medication side effects that may play a role during the neuropsychiatric examination. Furthermore, a side-effect review should be performed, as medications may have caused difficulties for the patient early in rehabilitation.<sup>43,44</sup> It is particularly noteworthy to determine if the patient is taking antiepileptic drugs at the time of the neuropsychiatric examination.<sup>45</sup> Significantly high doses of these medicines can cause cognitive slowing and adversely affect vigilance. On the other hand, if the patient is taking cognitive enhancers, there is evidence that cholinesterase inhibitors may improve cognitive impairments following traumatic brain injury.<sup>46</sup> The most frequent medications taken by persons who sustain a traumatic brain injury are antidepressants.<sup>47,48</sup> Other recent reviews have examined the pharmacologic treatment of psychosis, mood disorders, and anger and aggression.<sup>49-51</sup>

## **ACTIVITIES OF DAILY LIVING**

For those clinicians treating patients following a traumatic brain injury, daily activities are often one of the most useful portions of the history-gathering process. Obviously, the brain-injured patient who has been rendered quadriplegic will report a severely reduced level of activity, whereas the person who sustained a mild traumatic brain injury with little cognitive impairment may report few, if any, alterations of daily activities. In general, it is useful to begin with where the patient is currently living. Many times, living situations have changed following a brain injury. Moreover, if the patient's injuries have been a sufficient stress upon the family, divorce or separation may have occurred. Due to the physical changes that may occur in association with a traumatic brain injury,

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**TABLE 3.9**  
**Common Screening Questions for Activities of Daily Living**

- Are you presently working?
  - What is your time of arising and retiring?
  - Can you use a checkbook?
  - What household duties do you perform?
  - Can you play video games or use a computer?
  - Can you take overnight trips?
  - What are your hobbies?
  - What work do you perform in the yard, in the garden, or on the farm?
  - Do you attend sporting events, hunt, or fish?
  - Can you dial and use a telephone?
  - Can you dress and bathe yourself?
  - Can you have sex?
  - Do you have any urinary or bowel impairments?
- 

it is useful to inquire about biological markers of vegetative function. Ask the patient what time he retires at night and what time he arises. An inquiry should be made whether there has been a change in bathroom functions or sexual function.

Questions about activities that most normal people engage in are the most informative. For instance, does the patient have hobbies he pursues, and if he no longer pursues hobbies, why not? Can the person watch television, and if so, how much? Has there been any alteration in the person's ability to read or write? What literature does the individual read, and has there been any alteration in the complexity of literature that the person can understand? How many hours of television does the person watch daily, and has there been an increase or decrease in the level of viewing? Does the person fix his own breakfast? Can he drive an automobile or other vehicle, and if not, has there been a change in his ability to do so? Can the person prepare meals, wash dishes, clean his home, and see to ordinary household and daily activities? If the person is ambulatory, can he leave his home to purchase groceries and other household items? Is he able to organize his day and activities sufficiently to leave home and see to his daily needs?

One of the major purposes in taking a history of activities of daily living is to determine two fundamental issues about the patient's life: (1) Has there been a change in the individual's ability to care for herself? and (2) If there has been a change, how significant has it been? For instance, can the individual now maintain a checkbook? Is she able to pay her own bills? Can she compose a simple letter? Does she use the telephone, and if so, how many times weekly? Is she able to eat outside her home socially, and if so, how many times monthly? Does she have friends or visitors into her home, and if so, how often monthly? Can the patient garden, tend to houseplants, or care for pets? Other questions regarding activities of daily living will be specific to the individual's lifestyle. It is one thing to ask questions of a 61-year-old widowed woman who was living alone at the time of her traumatic brain injury, and another to ask questions of a 47-year-old accountant who was operating his own accounting firm. Thus, the creativity of the examiner will be called into play to determine lifestyle-specific changes in activities of daily living. The accumulation of this data, especially information about one's work product, will be covered in greater detail where it is relative to forensic applications. [Table 3.9](#) provides a schema for historical screening of activities of daily living.

## **PAST MEDICAL HISTORY**

With a brain-injured adult, it is important to take a good childhood history of basic development in order to determine if there are any preexisting brain or mental difficulties that may interact with

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**TABLE 3.10**  
**Relevant Past Medical Historical Questions**

- Did you have any problems with development?
  - What was your birth weight?
  - Have you sustained prior head trauma or loss of consciousness?
  - Have you fractured any bones?
  - What medical problems did you have prior to your injury?
  - What surgeries did you have prior to this trauma?
  - What medications were you prescribed before your injury?
- 

the brain injury or exacerbate cognitive symptoms of brain injury. Most people know their birth weights, and that should be asked. Persons born prematurely, or those persons who spent a considerable portion of their early lives in neonatal units, may have some preexisting neurobehavioral difficulties prior to traumatic brain injury. Problems of development are also important to note. These may be markers of childhood developmental delays that have persisted into adulthood. Were there any childhood illnesses that impact upon brain injury? For instance, is there a history of childhood brain trauma or brain infections? Is there evidence of preinjury mental retardation, learning disability, attention deficit disorder, Tourette's syndrome, or other common childhood neuropsychiatric conditions that may have persisted into adulthood?

As the history becomes more focused upon adult health problems, it is important to determine whether there is a prior history of trauma. In other words, the brain injury being evaluated at the present time may not be the index brain injury. It is particularly important to inquire about prior motor vehicle accidents and their association with loss of consciousness, skull fractures, or head trauma. Has the person been in a motor vehicle accident sufficient to break bones and require a stay in the hospital? The patient should always be asked about a prior history of bone fractures. Many times, these are associated with slips and falls, significant work-related trauma, or other aspects of trauma wherein the person may have incidentally also sustained a blow to the head. [Table 3.10](#) provides a focus for obtaining relevant past medical history.

In discussing preinjury medical problems with the patient, of course, all medical problems have some importance. However, the focus will clearly be upon those medical problems that may have a direct bearing on how the person's brain injury affects her or a direct bearing upon diseases that may have an adverse impact upon function following a brain injury. The neuropsychiatric history of preinjury medical problems should be fairly extensive, but certainly not at the level of an internal medicine physician. In fact, it is important to focus upon diseases of the nervous system, as they are the most likely preinjury medical problems to impact directly upon functioning following brain injury. One should inquire whether the patient has had any adult forms of meningitis, encephalitis, or other infections of the central nervous system. Moreover, did the individual have childhood or adult epilepsy of some form? Has there been a preinjury stroke? It is not unusual to find a middle-age person who has had a stroke and subsequently sustains a traumatic brain injury in a motor vehicle accident. In most instances, the preinjury stroke would play some role in the postinjury symptomatology of the patient. Clearly, the examiner wants to know if there is any past history of intracranial hematomas, arteriovenous malformations, or multiple sclerosis. Diabetes is particularly problematic for a person who sustains a traumatic brain injury if the diabetes has been in place sufficiently long to cause angiopathy of the brain. Endocrinopathies are likewise important factors to consider in the medical history of a brain-injured patient, particularly hypothyroidism, which can impact adversely upon cognition. Heart disease is often a marker for possible cerebrovascular disease. The menopausal woman who needs, but yet is not receiving, estrogen replacement should be noted, particularly those women who sustain posttrauma depression and who may be estrogen deficient. Recent evidence suggests that these women do poorly on antidepressants unless they also receive estrogen supplementation.<sup>52</sup>

A preinjury surgical history should be taken and recorded. It is especially important to focus upon any intracranial surgery that may have occurred prior to brain injury. Cardiovascular surgery also plays an important role. It is useful to determine whether the person has had coronary artery bypass grafting “on-pump or off-pump.” There is significant evidence available that many coronary artery bypass surgeries result in substantial cognitive dysfunction following surgery.<sup>53</sup> Thus, a person may have had heart disease and subsequent surgery 2 years prior to brain injury. Cognitive disturbance could well be present following the heart surgery, which is then exacerbated by a closed-head injury. Other surgeries may be important markers for potential disease that could have an impact upon posttrauma brain function. In particular, peripheral vascular surgery or carotid endarterectomy should be noted. The need for carotid endarterectomy is often associated with cognitive disturbance from cerebrovascular disease, and complications from carotid endarterectomy can lead to cognitive dysfunctions.<sup>54</sup>

Careful history of preinjury medication usage should be obtained if possible. What medications the patient has used prior to brain trauma may be a marker for diseases that were present prior to brain trauma that currently affect the outcome of the injury. For instance, a long history of hypertension and the need for multiple antihypertensives to control the hypertension could be revealing regarding potential hypertensive brain changes. Diabetic medications and their length of usage are important subjects to note. Endocrine disorders, particularly thyroid function, may provide the examiner with insight regarding possible hypothyroidism. A prior history of cancer and use of chemotherapeutic agents is important. There is substantial evidence that chemotherapy may cause lasting cognitive disturbance after its usage.<sup>55</sup> Clearly, if the patient has been prescribed cognitive enhancers, such as cholinesterase inhibitors, the patient probably had a cognitive disorder prior to brain injury.<sup>56</sup>

## **PAST NEUROPSYCHIATRIC HISTORY**

The reason for the taking of this history is, of course, self-evident. With the adult, it is important to determine if there were psychiatric syndromes that developed in childhood, even if they did not require treatment. As noted previously, a history of attention deficit hyperactivity disorder or Tourette’s syndrome may play a role in adult behavior and adversely affect symptomatology following traumatic brain injury. Other disorders, such as autism spectrum disorder, may never have been diagnosed.<sup>57</sup> The taking of the psychiatric history in an adult is fairly standard and has been well covered in many modern textbooks of psychiatry.<sup>58,59</sup> Common sense dictates that traumatic brain injury will rarely improve most existing psychiatric disorders. In a person who had a preinjury psychiatric syndrome or illness, the traumatic brain injury may well produce a comorbid or dual-diagnosis situation. A person with bipolar I disorder with a rapid cycling variant, who then develops an orbitofrontal syndrome following a traumatic brain injury, may become an extremely difficult patient to manage. Those physicians treating the homeless or impoverished should recall that many homeless schizophrenic persons sustain traumatic brain injuries due to assaults.<sup>60</sup>

A careful inquiry of psychiatric treatment is important. Has the patient been treated on a chronic basis for a psychiatric disorder? What medications did the patient take prior to brain injury? How old was the patient when he first manifested his psychiatric illness? The examiner should carefully inquire regarding preinjury mood disorders, anxiety disorders, obsessional syndromes, psychotic conditions, and personality disorders, as all of these may be exacerbated or complicated by a traumatic brain injury. It is important to determine if there have been any psychiatric hospitalizations, as these are important markers for serious mental disorder.

With regard to preinjury neuropsychiatric conditions, it is important to inquire as to the preinjury presence of epilepsy and related syndromes, when they occurred, and how they were treated. Preinjury strokes have been mentioned previously. Preinjury dementias are a common complicating factor in traumatic brain injury, particularly in slips and falls among the elderly. In addition, it is important to inquire as to preinjury aggressive syndromes, antisocial personality and borderline

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**TABLE 3.11**  
**Taking the Neuropsychiatric History**

- Were you treated for any childhood psychiatric conditions?
  - While in school, could you learn, pay attention, and sit still?
  - Did you have behavioral problems in school?
  - Did your behavior ever get you into difficulty with juvenile legal authorities?
  - Have you been treated for substance abuse or alcoholism?
  - As an adult, were you treated for any psychiatric or psychological condition prior to injury?
  - Did any physician or family doctor ever prescribe nerve medicines, tranquilizers, or antidepressants?
  - Have you ever been in psychotherapy or counseling?
- 

personality disorders, and other syndromes that may have a brain–behavior basis. [Table 3.11](#) provides a starting point for developing a neuropsychiatric history.

### **FAMILY HISTORY**

The purpose of the family history is to differentiate preexisting brain injury factors that may play a role in the patient’s biological and psychological response to the brain injury. Taking a family history is essentially taking a history of genetic patterns of disease within the patient’s family and attempting to identify disease patterns that may have a familial basis. For instance, in neuropsychiatry, it is incontrovertible that alcoholism is familial and apparently can be specifically transmitted from parent to child whether or not the child is exposed to the alcoholic parent.<sup>61</sup> Antisocial personality is probably overrepresented with a genetic tendency in families. Antisocial personality disorder is clearly more common among the first-degree biological relatives of those with the disorder than among the general population.<sup>62</sup>

In taking the family history, it is important to focus upon illnesses in first-degree relatives. The neuropsychiatric examiner should not only screen for basic neurological and psychiatric conditions, but also give attention to hypertension, thyroid illnesses, diabetes, cancer, heart disease, lung disease, kidney disease, and liver or gastrointestinal disease. Specific inquiries should be made regarding the frequency in the patient’s first-degree relatives of epilepsy, neurological disease, Alzheimer’s disease, and stroke. In the psychiatric portion of the family history, the language should be appropriate to the person being examined. The examiner might initially ask if anyone in the family has had a “nervous breakdown.” If the answer is affirmative, then more specific questions can be directed to determine if the disorder was a bipolar affective illness, major depression, schizophrenia, or other more specific psychiatric condition. One should also ask the patient about a family history of markers that may represent psychiatric illness in families. This would include asking about the presence of suicides, homicides, violence toward others, child abuse, and spouse abuse within the family.

### **SOCIAL HISTORY**

Taking a quality social history, within the context of a traumatic brain injury examination, is quite helpful in terms of treatment planning for the patient. The social context of a traumatized individual is always important, and it may be predictive of how the patient will fare in rehabilitation. The history should first put the brain-injured individual into social context. This is best determined by developing a profile of the patient’s home of origin. It is important to ask where she was born, how many siblings she had, and if employed, what occupations her parents pursued. One should ask if the parents are currently living, how they are doing, and whether the injured patient was involved in caregiving of the parents, particularly if they are elderly.

A simple question to ask is “Who raised you?” We often assume as physicians that people are raised by their parents. However, by age 18, approximately 20% of youngsters have lost one of

their parents through death or divorce. Moreover, it is surprising how many youngsters are raised by grandparents, aunts, or other care providers, rather than the biological parents. Simple inquiry can be made as to whether the person's home life was happy. Was there abuse in the home, or was it a threatening place in which to live? Was the home of origin abusive, and did it cause the patient to feel depressed when young? It is now customary to ask men or women if they have ever been sexually or physically abused. This includes asking men and women whether they have ever been raped. It is amazing what answers are returned from this inquiry. Persons struggling with issues of abuse who then become brain damaged may have an extraordinarily difficult time with recovery due to unresolved issues of past abuse.

As noted in [Chapter 2](#), aggression may be an outcome of brain injury. Thus, it is important to ask in the social history if the patient has a preinjury history of violence to others. It is important to determine if he has ever harmed another person or shot, stabbed, or beaten another person. It is useful to ask if the person has ever killed another person, even if by accident. Specific inquiries should be made as to whether guns are in the home. Has the individual ever been in legal or personal difficulty due to his sexual behavior?

The educational history is an important marker for the patient's preinjury academic attainment. More specific inquiry into educational history will be noted in the forensic section, where level of education has importance in determining causation. However, in the clinical brain-injured patient, treatment planning may well change direction, depending upon the level of preinjury education. If the patient did not finish high school, it is important to determine the reason the person quit his education. Also, it is important to ask if the individual required special education classes, or while in grade school or high school, did teachers think he was difficult to control or was it difficult to obtain his attention? Further inquiry should be made as to post-high-school education, whether or not the person attended vocational school, and if the person is a high school dropout, did he attain a GED?

The person should be asked if he has ever been married or divorced and how many times. If more than one marriage is involved, it is worthwhile to learn why the person divorced and which party asked for the divorce. Direct questions should be asked generally regarding the quality of the present marriage, if the patient is in a stable marital situation. It is important to distinguish whether the quality of the marriage has been impacted by the brain injury in the spouse. Moreover, it is important to determine if the brain injury has had an impact upon the present relationship with regard to aggression, sexual dysfunction, and intimacy. As discussed in [Chapter 4](#), alterations of prosody may impact the maintenance of romantic relationships.

Within the context of the social history, the examiner should inquire as to any legal history. Specific questions as to whether the individual has ever been convicted of a felony or misdemeanor are important. Many brain injuries occur within the context of assaults or other criminal activity. Moreover, those persons with a predisposition to criminal activity are more likely to suffer a brain injury. A useful question is whether anyone has ever gotten a restraining order or emergency protective order against him, and likewise, has he in turn ever obtained a restraining order or emergency protective order against another person? A useful follow-up question is to determine if the patient has ever been charged with spouse abuse, child abuse, child neglect, or terroristic threatening. [Table 3.12](#) provides a structure for exploring family and social histories.

The employment–vocational history is important in the brain-injured individual. The examinee may be involved in consultation with vocational rehabilitation specialists, or the person may well need assistance with obtaining disability benefits. A simple chronology of preinjury employments should be obtained, and a rough job description of the patient's most recent employment may be a useful addition. Ask about military history in all patients who have been brain-injured. Historically, the majority of those who served in the military were males. That, of course, is no longer true. Important social information is gleaned regarding military history. Not only should one be asked if she has ever been involved in military service, but it is also useful to know if the person has ever attempted to enlist into military service or a service academy and been denied induction. If the

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**TABLE 3.12**  
**Exploring the Family and Social History**

- Inquire as to specific mental disease frequencies in first-degree relatives.
  - Develop a profile of the patient's home of origin.
  - Determine if prior sexual or physical abuse is an issue.
  - Have you been physically violent toward others?
  - Have you ever been arrested for terroristic threatening, spouse abuse, child abuse, or other violence?
  - Have you ever been a party in an emergency protective order or restraining order?
  - Have you been convicted of DUI, drug abuse, or drug distribution/possession?
  - What firearms are in your home?
  - Have you ever killed another person, even if by accident?
  - Determine the educational history and school performance.
  - Determine the marital and relationship history.
  - Determine the military history, if any.
  - Explore the employment history.
- 

individual has served in the military, it is important to determine the branch of service, years served, and rank at the time of discharge. Specifically, the individual should be asked if she has an honorable discharge. Those persons who were found unfit for military duty due to psychiatric disorder or inability to adjust to military life will have a military discharge other than an honorable one. While it may not be a dishonorable discharge, it may well be given under medical conditions, or it could be a “general discharge” under honorable conditions. Further inquiry should be made if there were any disciplinary actions taken against the individual while in military service and, of course, if in military service, whether the person served in a combat zone and whether he was wounded. Preinjury issues of posttraumatic stress disorder from military action are obvious possibilities.

## REVIEW OF SYSTEMS

The general review should focus upon vegetative signs and general health. Has the person had a change in weight, either up or down, or a change in sleeping pattern since the trauma? Has the person noticed fatigue or a change in appetite? In taking the head, eye, ear, nose, and throat history, careful attention should be paid to this area in the review of systems. Maxillofacial and scalp injury is a frequent comorbid condition in traumatic brain injury for obvious reasons.<sup>63</sup> Mandibular fractures may result in TMJ syndromes, and fractures into the maxillary and frontal sinuses may result in significant nasal airflow dysfunction and even increase the likelihood of obstructive sleep apnea. Orbital fractures can result in diplopia. Surgical techniques have advanced greatly the management of these fractures, but multiple residual symptoms may persist.<sup>64,65</sup>

In the system review of the chest, it is important to determine if posttraumatic complications persist that may have an effect upon the person's psychological or cognitive state. It is important to remember that severe trauma sufficient to injure the brain oftentimes produces thoracic vascular or lung injury in patients.<sup>66</sup> The patient may have sustained bleeding, embolization, or thrombosis of blood vessels that supply neurological structures. A careful review of the medical records, as noted in the “Review of Medical Records” section, will determine whether the patient sustained an aortic arch injury, injury to the descending thoracic aorta, or had embolization due to foreign bodies or air. The neuropsychiatric examiner is more likely to encounter complaints of causalgia due to thoracic outlet vascular injury as a result of trauma to the chest. Even more frequently encountered, though, are seatbelt injuries to the carotid arteries.<sup>67,68</sup>

In the cardiovascular review, it must be remembered that myocardial injury may occur in up to 50% of head-injured patients, even in the absence of coronary artery disease. Some myocardial damage is due to direct blunt-force trauma to the anterior chest wall, resulting in a myocardial



contusion. However, even more difficult to understand is the apparent distant cerebral effect upon the myocardium itself.<sup>69</sup> Penetrating wounds of the chest are common in trauma sufficient to produce brain injury as well, particularly in urban centers. These, in fact, may result in direct damage to the myocardium or to the great vessels surrounding the heart.<sup>70</sup> Thus, it is important to ask the usual cardiac questions. Has the patient experienced chest pain with exercise, shortness of breath on walking, collection of fluid in the lungs associated with swelling in the legs, or shortness of breath that awakens him at night?

The combination of abdominal and head injuries has been found to be particularly lethal.<sup>71</sup> Particularly in motor vehicle accidents, blunt abdominal trauma associated with a traumatic brain injury is very common. The patient may also have sustained a diaphragmatic rupture or a duodenal or colonic injury. Gastric injury is fairly rare from blunt trauma, but it does occur. However, small bowel injury is more common in blunt trauma, with a 5 to 15% incidence.<sup>72</sup> Due to bowel injury, the patient may have a malabsorption syndrome, chronic diarrhea, chronic constipation, nausea, or other abdominal symptomatology. Moreover, not infrequently following injuries of this type, the patient will complain of excessive gas or abdominal pain. With constipation, of course, inquiry should be made about laxative use.

In the genitourinary system review, the examiner should recall that injury to the urinary system or the genitals themselves occurs not infrequently in association with traumatic brain injury.<sup>73</sup> Contusions of the kidney are not uncommon at all, nor are contusions of the bladder or outright urinary bladder rupture. In the male, penetrating penile or testicular injury may have occurred. Chronic urinary tract difficulty may persist following brain injury. If the patient has been rendered paraplegic or quadriplegic, chronic need for catheterization may result in frequent urinary tract infections and their attendant morbidity.

Orthopedic injuries are extremely common in persons who have sustained traumatic brain injury. Obviously, many of the traumas to the body are as severe as the trauma to the head. However, there is an interesting aspect to this issue that some physicians do not consider. There is some evidence that suggests that the rate of fracture healing is accelerated in patients with a severe head injury, although the mechanism for this is not well elucidated by research or clinical experience.<sup>74</sup> The issue of enhanced bone healing in patients with fractures associated with neurological impairment was first reported by Riedel in 1883.<sup>75</sup> Rapid callus formation occurring in fractures associated with significant neurological insult or closed-head injury was reported in French by Benassy and associates in 1963.<sup>76</sup> Even more unusual, heterotopic bone formation may occur in soft tissues outside the skeleton in association with head injury.<sup>77</sup> However, not all orthopedic surgeons agree that excess callus formation or heterotopic ossification occurs. In fact, there is a present controversy in the orthopedic profession as to whether this is the case.<sup>78</sup> Be that as it may, orthopedists seem to be unified in their opinion that closed-head-injury patients who have concomitant orthopedic injuries require meticulous care to maintain alignment during fixation of fractures.<sup>79</sup> Thus, it is important to take a careful history regarding orthopedic complications following traumatic brain injury. The brain-injured patient may be sufficiently impaired that he cannot see to his physical rehabilitation. Moreover, significant pain and dysfunction may result from alterations of ossification during bone healing following traumatic brain injury.

## **TAKING THE CHILD BRAIN INJURY HISTORY**

### **POSTTRAUMA SYMPTOMS AND TREATMENT**

As was discussed in [Chapter 2](#), there are some distinct neuropsychiatric differences in brain trauma outcomes seen in children vs. those in adults. However, extensive research indicates that brain injury in children can produce deficits similar to those in adults in various domains. Thus, the history of neurobehavioral consequences can be taken from the child in a manner very similar to that from the adult. With the very young child or the middle school-age child, clearly many of the

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**TABLE 3.13**  
**Taking the Child's History of Attention/Language**

- Is the child easily distracted or poor at tasks?
  - Has the child's ability to converse or use language changed?
  - Does the teacher report a deterioration of verbal skills in speaking, reading, or writing?
  - Does the child read less at home or display disinterest in television?
  - Can the child tell a story or a joke?
  - Can the child focus upon video games?
- 

questions will have to be posed to the parents or custodian of the child. Many prominent research centers have published studies outlining the neurobehavioral consequences of traumatic brain injury in children, and it is suggested that if required, these sources be consulted.<sup>80-84</sup>

### **Attention**

While parents and children alike complain of attentional problems following traumatic head injury, the research studies supporting an objective measure of attentional deficit in children following head injury are rare. One way to get at potential attentional deficits in children is to ask whether the child is easily distracted. It is also useful to ask if the child is slower in reaction time than prior to the injury. As noted in [Chapter 2](#), expect that the deficits will be greater in children injured quite early in life vs. their older counterparts.<sup>85</sup> Most of the research on children with attentional deficits following head injury has focused upon continuous-performance tasks. This appears to result from the continuing low development of psychological tests in young children that measure the entire panoply of attentional deficits.

Attention may be found to be particularly impaired in children following closed-head injuries who are examined in the early postinjury period. These children may develop disorientation and confusion. Thus, if the evaluator is seeing the child within the first 3 months following a traumatic brain injury, specific questions regarding orientation and confusion are appropriate, if the child is old enough to be oriented. A number of standardized methods have been developed for measuring posttraumatic amnesia and orientation in children following head injury. These include the *Children's Orientation and Amnesia Test*.<sup>86</sup> [Table 3.13](#) describes approaches to exploring attention and language deficits in younger children.

### **Speech and Language**

As we have been reminded elsewhere in this text, children with closed-head injuries may display more pronounced difficulties with the pragmatic aspects of language than their adult counterparts.<sup>87</sup> While taking the history of speech and language changes in children, it is best to ask if notice has been made of difficulty formulating sentences from individual words? Has there been any change in the child's ability to carry on discourse? Of course, one has to take into account the age of the child when asking these questions. However, [Chapter 4](#) points out that most children after age 7 can use six- or seven-word sentences and recite their numbers into the 30s. If the child had a severe closed-head injury, he may use fewer words in sentences within his stories. The stories may contain less information and may not be as well organized. In the child from kindergarten age upward, this type of information can be obtained more easily from teachers possibly than parents, unless the more observant parent is intimately involved in assisting the child with homework. Deficits in discourse can cause substantial academic difficulties in children with closed-head injuries. Thus, the parents should be asked if teachers have written notes to the home regarding changes in the child's language skills following injury. Children, like adults, rarely develop a full aphasia or

substantial dysphasia following closed-head injury, so these are generally not likely to be seen except in a very small percentage of children.

## Memory and Orientation

Memory is a global rather than specific concept for most adults. Therefore, when discussing memory deficits in children with the parent, it is important to bring some focus to the history taking. Parents generally do not describe their children in terms of having verbal memories vs. visual memories, and this differentiation should be made clear for parents. In taking the history, it will be beyond most parents to differentiate more specifically verbal memory disorders in their child. It is known that memory deficits occur in a variety of amnesic components, including problems of storage, retention, and retrieval.<sup>88</sup> Yet, it is not likely that a parent will be able to differentiate this for the examiner, and if that differentiation is required, it is best secured from teachers or from neuropsychological data as described in [Chapter 6](#).

Since explicit memory involves the recollection of past events or facts and implicit memory involves performance in the absence of conscious recollection, it is important to distinguish with the parent whether the child's memory deficit is for facts and events or skills. Memory for skills generally remains intact in children following brain trauma. The child may well have motor impairment from a traumatic brain injury that interferes in his ability to perform skills, but he should remember how to use a computer or ride a bicycle, even after brain injury. On the other hand, factual memory in the child may show glaring deficits following brain injury.<sup>89</sup>

## Visuospatial and Constructional History

Generally, children who have been brain-injured demonstrate a decline in performance IQ relative to verbal IQ as measured by standard intellectual assessment batteries (e.g., *Wechsler Intelligence Scales for Children-III*). Many nonverbal skills, including both visuoperceptual and constructional abilities, may be impaired following brain trauma, thus driving down the performance IQ. This is covered in more detail in [Chapter 6](#), but constructional dysfunction has been reported using a three-dimensional block task.<sup>90</sup> Thus, it is useful to ask the parent if the youngster has demonstrated impairment in playing checkers, drawing two-dimensional objects, or handwriting. Most of the studies in children have included measures of visual-perceptual or visual-spatial skills requiring motor ability. Two studies noted some children with closed-head injuries show deficits on tasks involving facial discrimination<sup>90</sup> and picture matching.<sup>91</sup> It might be useful to ask the parent if the child demonstrated any inability to recognize known relatives or friends following the brain injury. Further information can be obtained from school teachers of young children. It may be useful to inquire as to whether there has been a deterioration in the child's constructional ability in cutting paper if the child is a preschooler, or in drawing and artistic skills if the child is kindergarten or early school age. [Table 3.14](#) provides guidance for taking a history of child memory or visuospatial dysfunction.

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**TABLE 3.14**  
**Uncovering Memory or Visuospatial Deficits**  
**in Children**

- Has the child displayed memory deficits for facts?
  - Has the child deteriorated in skills?
  - Can the child write on a line (if old enough to do so)?
  - Has the child's drawing skill deteriorated?
  - Can the child name common objects in her room?
  - Have the child's cutting skills deteriorated?
-

## Executive Function History

Current research evidence indicates that maturation of the frontal lobes extends in the human at least into adolescence, if not into young adulthood.<sup>92,93</sup> The child with executive dysfunction may not be able to filter out interfering or competing stimuli. The *Stroop Test* discussed in [Chapter 6](#) may prove abnormal in children with this tendency. Children have poor judgment to begin with, but generally childhood judgment deteriorates following frontal lobe injury. The child may become irritable, assaultive, or even sexually disinhibited. Verbal fluency may be impaired, and the child may also perseverate on drawing tasks or writing numbers. Thus, it is useful to ask the parent about these possible dysfunctions in a child who has sustained frontal lobe injury. Again, inquiry of school authorities or school psychologists may be useful as well.

Baddeley and Wilson have characterized a childhood dysexecutive syndrome associated with “metamemory.”<sup>94</sup> This is characterized by poor attentional control, diminished speed of information processing, and a breakdown of boundaries between different memory domains for various categories of information. This results in confabulation, intrusions, faulty retrieval, or memory deficits for semantic information. The patient is unable to set goals and carry them out, and the child may demonstrate poor organization and poor planning skills.

## Obtaining the History of Affective and Mood Changes

It is well recognized that it is difficult to diagnose a mood disorder in a prepubertal child, particularly if the child is below 7 years of age. Verbal communication is paramount in diagnosing a mood disorder in either adults or children, and most children under age 7 lack sufficient communication skills to describe their moods adequately. However, preschoolers with depression may look sad and have a reduced verbal communication following a brain injury. The parent or guardian should be asked about this in detail. Moreover, the child may move or talk more slowly. The normal communication of happiness through facial expression may alter following a brain injury. Common symptoms of depression in preschoolers also include loss of weight, a left shift on the growth curve, increased irritability and tearfulness, and somatic symptoms, particularly gastrointestinal discomfort.<sup>95</sup> With the older child, the examiner may be able to take the history directly from the youngster. Children between 7 and 12 years of age are able to admit to low mood, sadness, or feeling worthless. The parents or school authorities may be able to tell the examiner about alterations in concentration, reduced academic performance, and increased irritability and crying. The examiner should ask about suicidal ideation or if the child is expressing a desire not to live. Somatic symptoms are very common in this age group, and the most common symptoms following a brain injury are headaches and abdominal pain. The parents should be asked if the number of pediatrician visits has increased due to nonspecific complaints for which no sound medical basis can be found. These increased doctor visits may signal depression or anxiety. The diagnosis of a depression or anxiety disorder following brain injury in children will follow the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) guidelines for a mood disorder due to a general medical condition.

Results of research with normal infants suggest that hemispheric specialization for the perception and expression of positive and negative emotions is already present within the first year of life. As with adults, a polarity theory has developed in which the left hemisphere has a positive emotional valence and the right hemisphere possesses a negative valence.<sup>96</sup> Few studies of behavioral functioning following childhood closed-head injuries have been available until the late 1990s. Max and his colleagues have made a special study of behavioral function following closed-head injury in youngsters. They found that the onset of a “novel” psychiatric disorder, defined as one never before present in the child, occurred in almost half of children following traumatic brain injury. These diagnoses included organic personality syndrome, major depression, attention deficit disorder, and oppositional defiant disorder. A large percentage of these children were found to be depressed.<sup>97,98</sup> [Table 3.15](#) describes a line of questioning to uncover executive dyscontrol or mood deregulation.

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**TABLE 3.15**  
**Asking about Childhood Executive Dysfunction or**  
**Affective/Mood Changes**

- Is the child more irritable, assaultive, or sexually disinhibited?
  - Does the child now fight with peers?
  - Can the child resist focusing on extraneous stimuli?
  - Is the child sad, or does he speak of death?
  - Are gastrointestinal complaints more frequent?
  - Have general pediatric visits increased?
- 

When discussing mood or affective changes with the parent of the brain-injured child, the examiner should recall that behavioral functioning following childhood closed-head injuries does not closely correlate with cognitive outcomes. The cognitive and behavioral outcomes in children may be somewhat independent and not concordant following a closed-head injury. Thus, the examiner should not make any assumptions about mood changes in children and attempt to relate them to severity of brain injury. Careful inquiry regarding affective changes in children must be made on a case-by-case basis.

### **ACTIVITIES OF DAILY LIVING**

In many respects, this is a more important historical section for the child than for the adult brain-injured patient. Brain injury in children often affects observable behavior in ways easy to detect by reviewing possible changes in the child's daytime activities. It is important to inquire as to what time the child gets up in the morning, what time the child goes to bed at night, and how much sleep the child receives. For school-age children, it is important to inquire as to who cares for the child between 3:00 and 6:00 P.M. on school days. Has a parent given up employment to care for the child? An inquiry as to hobbies the child pursues and what the child reads is important. A review of television shows favored by the child as well as video games or computer games favored may give important historical information.

It can be revealing to determine if the child has altered overnight trips or stopped visiting with school friends. Indirect information can be gleaned by determining how many movies the child rents per month to watch in the home, how many times the child sleeps away from home in a year, and whether the child attends ball games or pursues outdoor activities. Inquiry should be made whether there has been an alteration in the child's socialization. For instance, how many times a month does the child eat outside the home socially? How many times a month do friends or family visit the child at home? If the child is old enough to use the telephone, how many times a week does the child call another. If the age is appropriate, it should be determined whether the child can dress himself or herself. Can the child bathe alone? Has there been any alteration in the child's bathroom functions, such as an increase in bed-wetting or incontinence.

### **NEUROPSYCHIATRIC DEVELOPMENT HISTORY**

Inquiry should be made of the child's birth weight and whether the child was born prematurely. If the child was born prematurely, the examiner should carefully record the birth weight and make some attempt to learn of neonatal problems or attendant neonatal issues. Was there evidence, for instance, of a perinatal birth injury? Did the child as an infant spend an inordinate time in the hospital after birth? The examiner should attempt to determine the developmental milestones for the child, such as age when capable of sitting alone, age when the child first crawled, and age when the child could pull himself up, and an attempt should be made to determine when the child could stand alone, could walk alone, and was potty trained.

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**TABLE 3.16**  
**Taking the Preinjury Pediatric History**

- Has the child had significant prior medical or surgical illnesses?
  - What medications did the child use prior to injury?
  - What was the prenatal, perinatal, and birth history?
  - Has the child developed in an expected manner?
  - What were the ages at expected developmental milestones?
- 

Some general estimation of preinjury childhood temperament should be assessed. The mother in particular should be interviewed if possible concerning social interaction problems, eccentric and odd personality styles, learning disorders, dyslexia, or need for special education. The mother should be asked if the child demonstrated preinjury hyperactivity, motor clumsiness, tics, epilepsy, eating disorders, or aberrations of thinking prior to the injury. It is important to ask the mother regarding her prenatal history and whether she had complications during the prenatal period or during labor and delivery. The mother should be diplomatically asked regarding her use of alcohol or drugs during pregnancy or whether she had an eating disorder.

It is mandatory to make an inquiry regarding the child's academic progress prior to the brain injury. This helps establish a baseline of preinjury cognitive ability and also provides a benchmark for determining any posttraumatic changes in academic performance. It is important to determine if the child had difficulty sitting still in school. Were there any noteworthy learning difficulties in school? Was the child able to keep her mind on tasks in the classroom, and did teachers bring any neurobehavioral issues to the parents' attention? If the child is of appropriate age, did the child have difficulty learning to read? Did teachers complain that the child was too active in the classroom? Was there any evidence that the child was a behavioral problem prior to the brain injury? Have there been legal issues with juvenile authorities?

## **PAST PEDIATRIC HISTORY**

It is important to review whether the child has experienced any serious medical or surgical illnesses independent of the traumatic brain injury and prior to the traumatic brain injury. [Table 3.16](#) provides a simple structure for review. A careful inquiry should be made regarding the prior presence of obesity, seizures, diabetes, thyroid disease, anemia, congenital heart disease, pulmonary conditions such as asthma, orthopedic deformities, gastrointestinal difficulty, or urinary tract problems. Inquiry should be made regarding prior hospitalizations or injuries from motor vehicle accidents. It is important to determine if the child has ever been rendered unconscious or had a previous brain injury. Has the child ever been in a coma for any reason, including meningitis or encephalitis? Has the child broken any bones, and if so, which bones and how were they fractured? Are there any preexisting child abuse injuries? A review of surgical procedures will generally be revealing of significant preinjury medical problems.

A careful review of the child's medication history in the year prior to the traumatic brain injury is important. The examiner should not forget to inquire regarding the use of over-the-counter medications or herbs or natural products, as parents often do not recognize these as drugs. Does the child have any history of drug allergies or drug reactions? This would include contrast dyes or other imaging substances. In today's cultural climate, careful inquiry should be made regarding the child's use of tobacco products, alcohol-containing substances, or illicit substances. Is there any prior history of glue sniffing, gasoline sniffing, paint huffing, or other organic solvents? Has the child ever received treatment for drug, alcohol, or substance abuse? Does the child consume caffeinated beverages of any kind, and if so, how many per day? For postpubertal girls, inquiry should be made as to any possible pregnancies, menstrual irregularity, or other gynecological issues.

## **PAST PEDIATRIC NEUROPSYCHIATRIC HISTORY**

In general, if a child has a preinjury neuropsychiatric disorder, traumatic brain injury worsens or exacerbates the condition in most instances. Thus, it is important to carefully inquire about preinjury neuropsychiatric conditions that may subsequently result in comorbid neurobehavioral pathology. Other authors have reviewed these issues extensively and in more detail than will be covered in this text.<sup>96,99</sup> Indirect inquiry may determine whether there was an undiagnosed neuropsychiatric condition present prior to the brain trauma. For instance, the parents should be asked if the child has ever been hospitalized for psychiatric, drug abuse, alcohol, or mental problems. Has the child ever been discharged from a hospital against medical advice? This is often a revealing question, as the parent may have been advised to admit the child and refused to do so. Has the child ever been prescribed any form of nerve medicines, antidepressants, tranquilizers, or other psychiatric medicines? Has the parent ever been advised by any doctor, health practitioner, or school counselor to get mental health or psychological treatment for the child? Has the parent or guardian ever refused mental treatment when recommended by a doctor? Has the child ever received any type of office treatment by a family doctor, psychologist, nonmedical therapist, or psychiatrist for any nervous condition, psychological, psychiatric, or family problem?

More specific inquiry for markers of childhood mental disorders should be undertaken. For instance, has the child ever intentionally overdosed on drugs or medicines? Has the child ever attempted to take her life? Has the child ever intentionally cut, burned, or disfigured himself? Has the child ever hurt, abused, or killed animals? Specific inquiry regarding preexisting brain trauma syndromes should be made. As noted previously, it is important to inquire as to whether learning disabilities were present prior to the trauma. Is there any preinjury history of epilepsy or seizures? Is there a preinjury history of attention deficit disorder, Tourette's syndrome, or motor tics? In today's infectious disease climate, it is important to determine if there are any neurobehavioral manifestations related to pediatric AIDS or HIV infection. Inquiry as to odd behaviors or lack of social reciprocity that may be associated with autism spectrum disorders should be made.

## **FAMILY HISTORY**

In a neuropsychiatric examination, of course, the family history focuses upon neurobehavioral disorders rather than general pediatric conditions. It is important to inquire of the parent whether first-degree relatives (parents and siblings) have evidenced conduct problems, violence toward others, suicides, attention deficit disorders, mood disorders, anxiety disorders, psychotic illnesses, or substance abuse and alcoholism.<sup>100</sup> More specific neurobehavioral inquiry should be made as well, and this would include a review of familial mental retardation syndromes, learning disabilities, dementias, movement disorders, early onset strokes, migraine headaches, or specific genetic illnesses such as Huntington's disease. The purpose of the neuropsychiatric family history is to determine if possible genetic predispositions to disease exist, which may play a role in the genesis of illness in the child.

## **SOCIAL HISTORY**

Recent studies have demonstrated that the role of environmental influences as predictors of outcome following childhood traumatic brain injury is quite important. Environmental influences are a significant predictor of both cognitive and behavioral outcome following traumatic brain injuries in children as well as adults. Socioeconomic status, family demographics, family status, and social environment are specific and consistent predictors of neurobehavioral outcome following traumatic brain injury in children.<sup>101</sup>

In taking a social history, it is important to determine the employment level of the parents and how many children are in the child's family of origin. Inquiry should be made into family finances and whether there is enough money for the child. If the parents are divorced, inquiry should be

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**TABLE 3.17**  
**The Neuropsychiatric, Family, and Social History of the Child**

- What was the child's preinjury temperament?
  - Did the child display clumsiness, tics, odd behaviors, poor attention, or hyperactivity prior to injury?
  - Prior to injury, had the school determined dyslexia, learning impairment, or a need for educational assistance?
  - Has the school ever developed an individualized educational plan?
  - Has the child displayed a lack of social reciprocity, poor peer relations, difficulty making friends, or aggression prior to injury?
  - Has the child ever attempted to harm himself?
  - What is the parental family history of psychiatric disorders?
  - Explore the current home milieu of the child.
  - Is there a history of sexual or physical abuse to the child?
- 

made into these issues and what the custody arrangements are and whether that is an additive stressor for the child. Table 3.17 describes exploration of the neuropsychiatric, family, and social history. A diplomatic inquiry should be made of the mother as to whether the father abuses her and whether the child has ever been sexually or physically abused. It is important to determine if the child has been or is bullied at school. It should be asked of all children whether guns are in the home and whether the child has access to guns.

## REVIEW OF SYSTEMS

Review of systems follows the same format as noted earlier for the adult historical inquiry. Obviously, specific factors regarding pediatric issues must be taken into account. However, the comorbid injuries to other body parts associated with traumatic brain injury are essentially the same in the child as they are in the adult. The examiner may be guided in taking the review of systems by information gleaned from review of the medical records.

## REVIEW OF THE MEDICAL RECORDS

### EMERGENCY ROOM RECORDS

No commonly accepted guideline is followed for the management of head injury in the emergency room. Efforts are currently being made to synthesize this knowledge, and as new knowledge is acquired, attempts are being made to develop a protocol.<sup>102</sup> Most American emergency departments now use the Glasgow Coma Scale (GCS) to quantify the neurological findings on a scalar basis. This has at least improved uniformity of descriptors for patients who have sustained head injuries.<sup>103,104</sup>

All clinicians should obtain the emergency room record of a patient following brain trauma if possible. Significant elements of useful information can be gleaned that may assist in the neuropsychiatric evaluation of traumatized patients. When reviewing the emergency room record, if the Glasgow Coma Scale has been followed, the issue of coma is generally well understood. Coma is defined as the inability to obey commands, utter words, and open the eyes.<sup>105</sup> The fully oriented patient will score 15 points on the GCS. A flaccid patient who does not open his eyes, vocalize, or move to stimulus will score 3 points on the GCS. No single score within the range of 3 to 15 points forms a basis for a diagnosis of coma. However, it is generally agreed among neurosurgeons that 90% of all patients with a score of 8 or less, and none of those with a score of 9 or more, are found to be in coma using the preceding definition. Therefore, a Glasgow Coma Scale score of 8 or less has become the generally accepted emergency department definition of coma.

In general, the clinician will find that a patient who has sustained a mild head injury (defined as GCS = 13 to 15) will undergo a general examination to exclude systemic injuries, and he receives



a limited neurological examination. X-rays, usually of the cervical spine, are obtained. A blood alcohol level and urine drug abuse screen are often obtained. A computed tomography (CT) scan of the head is the standard of care in all patients except those who are completely asymptomatic and neurologically normal. In a patient with a moderate head injury (defined as GCS = 9 to 12), the patient generally is confused or somnolent but still able to follow simple commands. The initial workup is the same as noted previously, but a CT scan is mandatory in all cases. The patient should be admitted for observation, and frequent follow-up neurological checks are made. If there is any deterioration in the cognitive state, a follow-up CT scan is obtained.

A severe head-injured patient (defined as GCS = 3 to 8) is unable to follow even simple commands, as consciousness is too impaired to allow response. The emergency room physician, in most cases, will obtain neurosurgical consultation in these patients, and the neurosurgeon generally will check for pupillary light reaction and oculocephalic reflex (doll's eyes), and possibly perform caloric testing to measure the oculovestibular reflex. Mannitol may be administered, but there is controversy regarding whether it should be administered only when focal neurologic findings are present or on a routine basis. Hyperventilation is also recommended, and the observer may note that this has been performed. Most experts recommend that hyperventilation be used judiciously in an effort to keep the  $P_{CO_2}$  at approximately 30 mmHg. Excessive hyperventilation can cause cerebrovascular constriction of such severity that cerebral ischemia may develop.<sup>106</sup>

The evaluating physician should carefully review the emergency room record to see if other common associated factors have occurred with the brain injury. This can be determined by a review of what physicians did in the emergency department. Is there indication that the patient was intoxicated at the time of injury? Does the patient have nontraumatic coma? For instance, is this a person who was assumed to have suffered a head injury but later was found to have another etiology for her coma? Was there an associated spinal cord injury? Were transfusions needed because of bleeding elsewhere? While mannitol, hyperventilation, and fluid resuscitation are used generally in traumatized patients who have sustained a brain injury, steroids are now discouraged. Clear proof of benefit has not been shown, and some patients have sustained deleterious effects following their use.<sup>107</sup>

## **THE HOSPITAL RECORD**

Basically, review of the hospital record is indicated to determine if complications arose that may have a bearing on the posthospital management of the patient. Moreover, in the neuropsychiatric examination, there may have been ancillary injuries that have played an adverse role on the neuropsychiatric outcome of the patient following brain injury, or have contributed to difficulties in rehabilitation. The patient may have sustained a significant neurogenic cardiovascular complication from the brain injury. Myocardial injury may occur in up to 50% of head-injured patients, even in the absence of coronary artery disease.<sup>69</sup> Myocardial dysfunction following brain trauma has been well described in adults, but it is also seen in children.<sup>108</sup> The lesions produced in the heart are similar to those seen after an acute myocardial infarction, with pheochromocytoma, or following catecholamine infusion. At autopsy, subendocardial hemorrhages are commonly found. No clear relationship has been found pathologically, but it is thought that there may be an association between hypothalamic lesions and myocardial damage.<sup>109</sup> In those patients who have sustained such myocardial injury, the neuropsychiatric examiner will generally find that catecholamine inhibitors and adrenergic inhibitors have been used for treatment while the patient was in the neurosurgical ICU.

Following brain injury, many patients complain that they breathe poorly or cannot breathe as well as they did prior to their brain injuries. Respiratory system dysfunction is commonly found as a complication of traumatic brain injury. The most dramatic disorder the examiner may note in the medical records is neurogenic pulmonary edema. This is a variant of the adult respiratory distress syndrome (ARDS) seen with general body trauma and other diseases. A common cause of

death in patients who have sustained an intracranial hemorrhage or severe isolated head injury is neurogenic pulmonary edema.<sup>110,111</sup> Other pulmonary complications of head injury include infection. Neurosurgeons have learned that many earlier causes of pneumonia found in the brain-injured patient came about as a result of neutralization of gastric pH by antacids or H<sub>2</sub> receptor blockers. This allowed overgrowth of gram-negative bacteria in the stomach, which colonized the trachea. Sucralfate has become more commonly used as a stress gastric prophylaxis rather than H<sub>2</sub> blockers in many instances.<sup>112</sup> It is also common for head-injured patients to remain at risk for pulmonary problems during rehabilitation, and some traumatically brain-injured patients will remain with variable compromise of pulmonary function.

Head-injured patients are noteworthy for having greater risk of deep vein thrombosis and secondary pulmonary embolus. These patients are at moderate to high risk for such complications, and the neuropsychiatric review of the medical records should determine if these have in fact occurred. Some patients may have even required a Greenfield filter by vena cava placement.<sup>113</sup> Coagulopathy is another common adverse outcome following traumatic head injury. Brain tissue is a potent stimulator of disseminated intravascular coagulopathy (DIC) and, in fact, is used as an agent to initiate clotting in certain blood tests. Brain tissue injury, together with injury to endothelial cells of local vessels, can initiate DIC, which may be exacerbated by the accompanying catecholamine release due to severe injury.<sup>114</sup> As a result of DIC, delayed or postoperative intracranial hematomas may occur. Even more problematic is the patient who may have received a ventriculostomy and then developed a hematoma along the path of the catheter.<sup>115,116</sup> As the neuropsychiatric examiner is reviewing the medical records, the presence of DIC will generally be indicated by the need for replacement of depleted clotting factors, generally with fresh-frozen plasma. Cryoprecipitate may also be used.

Brain trauma results in a severe physiological stress to the body and elevates adrenocorticotropic hormone (ACTH) release. This secondarily increases cortisol secretion. Brain injury to the frontal brain parts may damage the hypothalamus and pituitary. If the injury is severe, the result is usually death. Rarely, the patient develops a syndrome of inappropriate antidiuretic hormone secretion (SIADH) or panhypopituitarism. Infrequently, loss of thermoregulation may occur due to hypothalamic damage. These are usually low-likelihood events occurring in less than 1% of brain-injured persons; but when they do occur, they can cause significant difficulty to the patient.<sup>109</sup> An endocrinologist may be required to manage some patients following brain injury, and, depending on the particular releasing factor deficiency, adjunctive hormonal replacements may be needed.<sup>117</sup>

Gastrointestinal complications frequently occur following head injury. In reviewing the medical records, the neuropsychiatric examiner may notice that enteral feeding was instituted. It is thought that this nutritional support decreases infectious complications in patients, particularly the development of pneumonia. Moreover, the gut may be an important central engine for the development of multiple-organ failure syndromes, and the early institution of feedings is often done in neurosurgical centers today.<sup>118</sup> Stress gastritis frequently occurs in head-injured patients with a clinical incidence of up to 75%.<sup>119</sup>

## **COGNITIVE REHABILITATION RECORDS**

If the traumatic brain injury produced significant cognitive deficits in the individual, or if the person has substantial evidence of physical impairments, the person is usually transferred to a brain injury rehabilitation unit following discharge from the acute care hospital. There are vast differences in the quality of cognitive rehabilitation programs, and the examiner should keep this in mind when reviewing these records. At the most basic level, cognitive rehabilitation programs may focus on individual skill development through repetitions or rely upon devices such as memory notebooks. However, these may not be effective in overall cognitive rehabilitation.<sup>120</sup> On the other hand, many skilled facilities across the U.S. provide superb care. In general, the evaluator will notice three major foci of rehabilitation techniques: (1) attentional rehabilitation, (2) feature

identification rehabilitation, and (3) categorization rehabilitation. The treatment environment for attention deficits uses stimulus-enhanced techniques. These generally are both auditory and visual stimuli. Some cognitive rehabilitation specialists utilize the Premack principle. This principle assumes that any behaviors that are spontaneously produced may be viewed as reinforcing to the organism, and techniques utilizing this principle are often provided to patients requiring attentional deficit rehabilitation.<sup>121</sup>

Many brain-injured persons perseverate. Perseveration is thought by many to be an inability to shift a focus of attention, and therefore, the person continually repeats the behavior or task. The perseveration behavior may coexist with inability to maintain vigilance. Vigilance often refers to an individual's ability to maintain a focus of attention and self-monitor incoming stimuli in order to screen for a specific set of features. Vigilance is one of the most complicated attentional skills, and therapy may concentrate on maintaining a focus of attention in a stimulus-rich environment where multiple distracters are present. Another attentional deficit often seen and treated during cognitive rehabilitation is the inability to cognitively shift. It is more complicated than either vigilance or suppression of perseveration. Cognitive shifting requires the person to mentally shift between activities with the least amount of disruption to the information being received and stored. Generally, a therapist will have the patient begin with shifting physical tasks from one task to another, then progress to shifting from a physical task to a mental task, and then lastly focus upon shifting strictly from one mental task to another.

Feature identification is done automatically by all of us. However, brain-injured persons have specific difficulties performing this skill. From the time of early infant development, all individuals must learn to attend to and identify the iconic features of objects. This includes such features as color, shape, texture, weight, etc. Individuals with language disorders may become unable to describe or name an object and instead will mention its function. For instance, instead of naming a cup, the individual may describe its use as a drinking utensil. The remediation of deficits of feature identification generally requires the individual to focus on a checklist of seven or eight iconic features such as color, shape, etc. Then the person progresses through steps in the hierarchy to gradually increase her skill at feature identification.

After a person relearns to identify features of objects, the rehabilitation then helps the individual learn to categorize. Categorization is learned very much like feature identification in that the person is guided to separate the color from the form of an object. For instance, an apple, fire truck, and cardinal all share the same red color. The individual is gradually challenged in an increasingly difficult hierarchy to define symbolic or functional categories and separate these from features, such as color, that place separate categories into the same group.

## **OCCUPATIONAL AND PHYSICAL THERAPY RECORDS**

The rehabilitation records should contain considerable information regarding the person's ability to manipulate objects. Moreover, documentation of balance is usually available. However, depending on the level of skill of the examiner in the rehabilitation facility, it may not provide the neuropsychiatric examiner with adequate information. This, of course, can be obtained during psychiatric observation or neurological testing. The record should be examined for complaints of headache, blurred vision, or nausea, particularly after physical activity or a change in the attitude of the head in space. This may indicate vestibular dysfunction.<sup>122</sup>

Generally, information will be contained in these records regarding the range of motion of extremities and trunk. Also, statements about the neurologic status and whether hemiparesis is present can be found generally. Physical therapy records will be most important in determining the strength in extremities and overall physical endurance of the person. If the person is hemiparetic, or has quadriparesis, the physical and occupational therapy records will yield information generally regarding the quality of movement. Information explaining how the injured person transfers from wheelchair to car, from car to wheelchair, from bed to wheelchair, from bed to commode, and

other important motor information can be determined. This information is very useful to the neuropsychiatric examiner as the examination may take place a significant time following discharge from rehabilitation. Thus, a comparison of continued progress can be made qualitatively, if not quantitatively.

## **SPEECH AND LANGUAGE PATHOLOGY RECORDS**

Early on in traumatic brain injury, particularly while in the acute care hospital or early in the rehabilitation hospital, the speech and language pathologist will make at least an initial screening assessment. This information will be contained in the record where the evaluation proceeds. In those rare instances where a true language disorder exists following traumatic brain injury (see [Chapter 2](#)), extensive language rehabilitation may be undertaken.

The speech and language records are very helpful in determining whether the person had oromotor dyspraxia or dysarthria. If the patient has a brain stem injury, speech and language pathologists often assist radiologists in performing cineographic swallowing studies. These are particularly important in a patient who may be at risk for aspiration. With regard to voice production, the records may be revealing regarding velopharyngeal integrity and whether the communication skills of the patient are impaired.<sup>123,124</sup>

## **TAKING THE COLLATERAL HISTORY**

When dealing with moderately to severely brain-injured patients, collateral history may be very important. During the neuropsychiatric examination, the practitioner is making an effort to both establish baseline behaviors and function and presently determine functional abilities following the injury. Two basic issues rise to the forefront in the collateral histories: (1) ancillary information regarding the injured patient's physical and mental deficits, and (2) issues of caregiver stress within the domicile of the patient.

With regard to the former, it is sometimes helpful to use an instrument such as the *Neurobehavioral Rating Scale*<sup>125</sup> to assist in the collection of relevant data regarding the patient's current functioning (see [Table 3.18](#)). If collateral information is needed, the best person to supply that is the individual most intimately familiar with the day-to-day activities of the injured person. The so-called activities of daily living may be very objectively described by either the spouse or, in the case of a child, the parent. The examiner will want to know how the patient functions in the areas of hygiene, toileting, dressing, grooming, feeding, meal planning, meal preparation, shopping, laundry, medication taking, telephone usage, computer usage, motor vehicle operation, hobbies, time management, and health and safety issues. For instance, individuals who have sustained injury to the frontal lobes may not be able to set goals, plan, have foresight to the future, or maintain persistence and initiation.<sup>126</sup> Collateral history is often much more accurate in the determination of residual frontal lobe impairment than is information from a patient who may not be aware of his deficits. The collateral interview may be extremely telling in determining whether the person is verbally or physically aggressive. Many times, patients either poorly self-monitor these behaviors or outright deny that they exist.

Very important information can be obtained from collateral sources regarding the individual's community skills. How does the individual drive a vehicle? Is the person able to use community transportation? How does the individual pursue leisure activities or hobbies? Is there impairment in the person's ability to communicate and socialize with others? If the person requires special needs such as transportation assists or wheelchairs, is the individual capable of managing these special needs?

**TABLE 3.18**  
**Neurobehavioral Rating Scale**

	Not Present	Very Mild	Mild–Moderate	Moderate–Severe	Severe	Extremely Severe
1. <b>Inattention/reduced alertness</b> — fails to sustain attention, easily distracted, fails to notice aspects of environment, difficulty directing attention, decreased alertness	—	—	—	—	—	—
2. <b>Somatic concern</b> — volunteers complaints or elaborates about somatic symptoms (e.g., headache, dizziness, blurred vision) and about physical health in general	—	—	—	—	—	—
3. <b>Disorientation</b> — confusion or lack of proper association for person, place, or time	—	—	—	—	—	—
4. <b>Anxiety</b> — worry, fear, overconcern for present or future	—	—	—	—	—	—
5. <b>Expressive deficit</b> — word-finding disturbance, anomia, pauses in speech, effortful and agrammatic speech, circumlocution	—	—	—	—	—	—
6. <b>Emotional withdrawal</b> — lack of spontaneous interaction, isolation, deficiency in relating to others	—	—	—	—	—	—
7. <b>Conceptual disorganization</b> — thought processes confused, disconnected, disorganized, disrupted; tangential social communication; perseverative	—	—	—	—	—	—
8. <b>Disinhibition</b> — socially inappropriate comments or actions, including aggressive and sexual content, or inappropriate to the situation; outbursts of temper	—	—	—	—	—	—
9. <b>Guilt feelings</b> — self-blame, shame, remorse for past behavior	—	—	—	—	—	—
10. <b>Memory deficit</b> — difficulty learning new information; rapidly forgets recent events, although immediate recall (forward digit span) may be intact	—	—	—	—	—	—
11. <b>Agitation</b> — motor manifestations of overactivation (e.g., kicking, arm flailing, picking, roaming, restlessness, talkativeness)	—	—	—	—	—	—
12. <b>Inaccurate insight and self-appraisal</b> — poor insight, exaggerated self-opinion, overrates level of ability and underrates personality change in comparison with evaluation by clinicians and family	—	—	—	—	—	—
13. <b>Depressive mood</b> — sorrow, sadness, despondency, pessimism	—	—	—	—	—	—
14. <b>Hostility/uncooperativeness</b> — animosity, irritability, belligerence, disdain for others, defiance of authority	—	—	—	—	—	—
15. <b>Decreased initiative/motivation</b> — lacks normal initiative in work or leisure, fails to persist in tasks, is reluctant to accept new challenges	—	—	—	—	—	—
16. <b>Suspiciousness</b> — mistrust, belief that others harbor malicious or discriminatory intent	—	—	—	—	—	—
17. <b>Fatigability</b> — rapidly fatigues on challenging cognitive tasks or complex activities, lethargic	—	—	—	—	—	—
18. <b>Hallucinatory behavior</b> — perceptions without normal external stimulus correspondence	—	—	—	—	—	—
19. <b>Motor retardation</b> — slowed movements or speech (excluding primary weakness)	—	—	—	—	—	—
20. <b>Unusual thought content</b> — unusual, odd, strange, bizarre thought content	—	—	—	—	—	—
21. <b>Blunted affect</b> — reduced emotional tone, reduction in normal intensity of feelings, flatness	—	—	—	—	—	—

**TABLE 3.18 (Continued)**  
**Neurobehavioral Rating Scale**

	Not Present	Very Mild	Mild–Moderate	Moderate–Severe	Severe	Extremely Severe
22. <b>Excitement</b> — heightened emotional tone, increased reactivity	—	—	—	—	—	—
23. <b>Poor planning</b> — unrealistic goals, poorly formulated plans for the future, disregards prerequisites (e.g., training), fails to take disability into account	—	—	—	—	—	—
24. <b>Lability of mood</b> — sudden change in mood that is disproportionate to the situation	—	—	—	—	—	—
25. <b>Tension</b> — postural and facial expression of heightened tension, without the necessity of excessive activity involving the limbs or trunk	—	—	—	—	—	—
26. <b>Comprehension deficit</b> — difficulty in understanding oral instructions on single- or multistage commands	—	—	—	—	—	—
27. <b>Speech articulation defect</b> — misarticulation, slurring, or substitution of sounds that affect intelligibility (rating is independent of linguistic content)	—	—	—	—	—	—

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# 4 The Neuropsychiatric Mental Status and Neurological Examinations Following Traumatic Brain Injury

## INTRODUCTION

Cognition, behavior, and neurological status are often affected in traumatically brain-injured adults or children. Depending on the nature of the trauma, location of the trauma within the brain, age of the patient, and any posttraumatic complications, manifestations may include disorders of intellect, memory and learning, language, executive function, mood and affect, motivational behavior, and neurological functioning. This chapter focuses first upon a detailed mental and neurological examination of the adult, and follows that with a similar detailed explanation of these examinations in the child. It should be noted that the mental examination of the traumatically brain-injured patient expands upon the classic Meyerian mental examination of the psychiatric patient. In the brain-injured patient, the examination focuses upon brain–behavior relationships, and this model follows a neuropsychiatric structure rather than a classical psychiatric approach. For a more extensive review of mental examination procedures and techniques, refer to the texts by Strub and Black, Trzepacz and Baker, and Lezak.<sup>1–3</sup>

The neuropsychiatric mental status examination will consider specific syndromes that have as their basis a neuropsychiatric dysfunction. These syndromes are outlined in [Table 4.1](#). This schema offers a useful format for characterizing neurobehavioral syndromes that may be seen after a closed-brain injury or a penetrating brain injury. Elements of these syndromes have been described previously (see [Chapter 2](#)). The neurological examination of the traumatically brain-injured patient has a different focus than the mental examination. The focus of both components of the neuropsychiatric examination is variable depending upon the stage of the patient within his recovery. Most

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**TABLE 4.1**  
**Specific Neuropsychiatric Disorders**

- Frontal lobe disorders: apathetic, disinhibited, and dysexecutive syndromes
  - Temporal lobe disorders: amnesic disorders, personality dysfunction, and temporal lobe-based seizure syndromes
  - Basal ganglia or brain stem dysfunctions: movement disorders, arousal disorders, and subcortical cognitive dysfunction
  - Language and prosody disorders
  - Visual processing disorders
  - Disorders of motor or sensory behaviors
  - Denial and neglect syndromes
-

neuropsychiatric examinations will not be performed upon the acutely brain-injured patient. Generally, the neuropsychiatric examination is utilized to determine residual cognitive and behavioral dysfunction and develop a treatment plan sometime after the trauma. Whereas the neurological examination serves to localize the site and extent of brain injury in the acute patient, examination in the postacute patient attempts to identify physical, neurological, cognitive, and psychiatric deficits that may limit the patient's function.<sup>10</sup>

## THE ADULT MENTAL EXAMINATION

### APPEARANCE AND LEVEL OF CONSCIOUSNESS

This portion of the adult mental examination enables the examiner to develop a mental picture for the reader of the report regarding the patient's appearance and demeanor. For example, the record might state the following:

R.K. is a 52-year-old Caucasian male who appears older than his stated age. He is disheveled in his dress, and he ambulates poorly due to an obvious right hemiplegia. He makes poor eye contact with the examiner and mumbles when answering questions. He has a large port-wine hemangioma over the left periorbital region. He manifests slow thought and motor speed.

In most instances, the neuropsychiatric mental and neurological examination of the traumatically brain-injured patient will occur in the postacute phase. That is, the patient will have been released from an acute care medical facility and the first neuropsychiatric examination will generally occur either during rehabilitation or at a time later following the patient's discharge to his home. Thus, only in the rarest instances would the neuropsychiatric examination be attempted in a patient in a stuporous, semicomatose, or comatose state, as cognition could not be measured. Examinations of the acutely injured person will generally be dictated by the neurosurgical or neurological needs of the patient.

Most clinicians distinguish five levels of consciousness: (1) alertness, (2) lethargy, (3) obtundation, (4) stupor, and (5) coma.<sup>4</sup> In general, the postacute neuropsychiatric evaluation will find the patient to be at one of the first three levels: alert, lethargic, or obtunded. The alert patient is fully awake and responds appropriately to external and internal stimuli. The examiner should not confuse alertness with lack of cognitive impairment. A person may be fully alert yet have measurable cognitive deficits. The lethargic patient is not fully alert and tends to drift in awareness or consciousness when not actively stimulated. In general, motor speed is reduced as well, and the examiner will find that the patient attends poorly to the examination. The obtunded patient generally presents a level of consciousness lying somewhere between lethargy and stupor. In this instance, the patient may be difficult to arouse and is generally confused. Cooperation and ability to pay attention are marginal or overtly impaired. Detailed neuropsychological examination generally cannot be performed. Cooperation during neurological evaluation is generally limited. [Table 4.2](#) includes the elements of appearance and level of consciousness commonly noted in a mental status examination.

### ATTENTION

Attention and concentration are often impaired in patients sustaining traumatic brain injury, particularly injury to the anterior cerebral hemispheres.<sup>5,6</sup> The ability to attend to a stimulus is critical to neuropsychological functioning. Unless the patient can pay attention and receive the stimulus into the appropriate brain area, sensory data external to the patient cannot be utilized. Thus, attention is the patient's ability to bring focus to a specific stimulus without being distracted by extraneous internal or environmental stimuli.<sup>7</sup> Attention maintained longitudinally over time is known as vigilance. Other terms for vigilance are *sustained attention* or *concentration*. Thus, it can be seen

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**TABLE 4.2**  
**Common Mental Examination Elements of**  
**Appearance/Level of Consciousness**

- Apparent age
  - Level of consciousness
  - Dress and grooming
  - Eye contact
  - Physical abnormalities
  - Speed of mental/motor function
- 

that attention is much more focused and requires a specific orienting response, whereas vigilance is nonspecific and refers to a more basic tonic arousal process in which the awake patient can respond to any stimulus appearing in the environment.<sup>1</sup> As we shall see in [Chapter 6](#), attention can be divided into any of the five senses. There is a specific attentional capacity for each sense. In most instances of brain injury assessment, the cognitive measurement of attention focuses on the sensory modalities of visual, auditory, and sometimes tactile function. In most evaluations of a traumatically brain-injured patient, olfactory sensation is examined during neurological assessment only, and gustatory sensations are usually not measured.

In the evaluation of attention, one of the most qualitatively valid sources of information is the clinician's own experience and training. The examiner should note the patient's behavior and watch for distractibility, difficulty in attending to the examiner's questions, and problems within the patient as she attempts to maintain vigilance while being examined. The patient's basic level of auditory attention can be assessed by using the digit repetition test, which has been commonly used by psychiatrists and neurologists for almost a century. Single digits are recited to the patient in a series of increasing length. After each series is repeated to the patient, the patient is asked to repeat it aloud. Writing is not permitted, as this would introduce a language and motor or proprioceptive component and take the test beyond the measurement of auditory attention. The examiner should initiate the examination by saying, for example, "I want you to repeat the following numbers after I say them: 3-1-9-2." Then the patient should reply back exactly, "3-1-9-2." It is important to recite the digits in a monotone, except for the last digit, which should be said at a slightly lowered pitch so that the patient understands that it is the final digit of the series. If the examiner does not speak in a monotone, the prosody (musical or nonverbal nature of speech) may provide a cue to the patient and inappropriately improve the patient's ability to repeat the digits. Generally, about a 1-sec interval should exist between each digit as it is recited to the patient.

Generally speaking, the examiner can start with a two- or three-digit series and then increase the span by one digit with each series to the point that the patient cannot repeat the digits correctly. The maximum number of digits repeated is the *digit span*. Normal forward digit span length is six  $\pm$  one digit. This ability to recite at least six digits should remain stable into old age. In fact, most normal and healthy adults can perform seven digits forward and five digits backward. It must be remembered that reciting digits backward is not a pure measure of auditory attention, as it also introduces a parallel processed working memory task. The patient must divide her attention: remember the forward order of the digits, and then mentally reverse them before repeating them back to the examiner. However, traumatically brain-injured patients often have frontal lobe difficulty and reciting a digit span backward can be quite challenging to them. The challenge arises because reciting a digit span backward, while requiring divided attention, also measures concentration or vigilance. For instance, during performance of the *Mini-Mental State Exam*,<sup>35</sup> spelling *world* backward is used as an alternative to digit span repetition or the *Serial 7s Test*. Again, this apparently simple test is measuring divided attention and concentration as well as pure auditory attention. [Table 4.3](#) lists common approaches to evaluating attention during the mental status examination.

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**TABLE 4.3**  
**The Face-to-Face Assessment of Attention**

- Each sensory modality has an orienting or attentional component.
  - The classic mental status examination generally evaluates only auditory and sometimes visual attention.
  - Auditory attention can be tested by digit repetition.
  - Visual attention can be tested by a letter cancellation or similar task.
  - In aphasic persons, digit repetition or letter cancellation cannot be tested validly.
- 

Another popular test of vigilance is the Serial 7 Subtraction. This simple bedside examination technique has been part of psychiatry and neurology for many years. It is a measurement of vigilance, dual tracking, and concentration, rather than focused attention, and the patient is asked to start at 100 and subtract 7, and then to keep subtracting 7 from each answer. The expected response from the patient is “100, 93, 86, 79, 72, 65 ... .” Any interval of seven completions is considered within normal limits.<sup>2</sup> Obviously, the greater the number of errors, the poorer the concentrating ability of the patient.

## **SPEECH AND LANGUAGE**

Speech generally refers to the motor-driven articulatory component of language. Language is the symbolic representation and cognitive processing necessary for communicative speaking, reading, and writing. Language skills are more likely to be impaired in traumatic brain injuries involving the dominant cerebral hemisphere. As noted in [Chapter 2](#), aphasic disorders comprise about only 2% of cognitive deficits seen following traumatic brain injury. It was further noted that language disorders in children may differ significantly from adult language disorders (see [Chapter 2](#)).

Dysarthria, an impairment in articulation, is a common outcome of traumatic brain injury and generally is caused by weakness or incoordination of the tongue or pharyngeal muscles. The pattern of deficit depends on the location of brain injury.<sup>8</sup> In patients who have sustained head trauma causing either brain stem or complex facial injuries, injury to nerve XII may cause unilateral tongue weakness and difficulty articulating lingual consonants (*T, D, L, R, N*). Patients with substantial nerve VII weakness may have difficulty with labial and dentilabial consonants (*P, B, M, W, F, V*). Bilateral involvement of corticobulbar pathways in the brain stem results in “pseudobulbar” speech characterized by a slow, labored speech production and a strained quality as the patient attempts to produce speech. Cerebellar damage causes dysrhythmic speech seen with irregularities in pitch and loudness. Basal ganglia injuries may result in jerky, dysrhythmic speech and are often associated with movement disorders such as choreoathetosis or loss of prosody and Parkinsonian features.<sup>9,10</sup>

The examiner must be careful not to confuse dysarthria with dysprosody. Dysprosody is an interruption of speech melody (e.g., tone, accent, tempo, and affect). It is these musical aspects of speech that are altered with dysprosody, and it sometimes can be mistaken for a foreign accent.<sup>11</sup> Dysprosody often results from nondominant cerebral hemispheric damage (see [Chapter 6](#)). The left cerebral hemisphere anteriorly drives and posteriorly receives symbolic language in parallel with the nondominant right hemisphere. Thus, the expressive nonverbal components of speech are produced simultaneously in the nondominant hemisphere, whereas the anterior dominant hemisphere produces the symbolic phonemes of language. Alternatively, the posterior nondominant hemisphere decodes the facial expression, verbal affect, tonal quality, and nonverbal body movements of others in the same fashion that the dominant hemisphere decodes the symbolic phonemic language elements of the speaker.<sup>12</sup>

Language function is evaluated in the face-to-face mental status examination by listening to verbal fluency, assessing comprehension, determining whether the person can repeat, assessing the ability to name objects and find words, and asking the individual to read, write, and spell. This

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**TABLE 4.4**  
**Impairment of Speech Articulation**

- Dysarthria is distortion of speech sounds.
  - Nerve XII impairment affects lingual consonants *t, d, l, r,* and *n*.
  - Facial weakness affects labial consonants *p, b, m, w, f,* and *v*.
  - Cerebellar lesions cause irregularities of pitch and loudness.
  - Basal ganglia injury may result in dysrhythmic speech sounds with choreoathetotic movements.
- 

evaluation should be done within the context of determining the handedness of the person because of the close relationship between handedness and cerebral dominance for language. Moreover, while listening to spontaneous speech, the examiner should determine whether the speech is dysarthric, dysprosodic, or fluent and listen for evidence of specific aphasic elements such as syntax errors, word-finding pauses, paraphasias, or the insertion of new or unrelated words. Table 4.4 outlines a simple listening approach to the mental status examination assessment of articulation. The expressive language of many patients with aphasia cannot be classified strictly by its fluency. The primary goal of the examiner in the evaluation of a brain-damaged patient is to recognize that the language production *is in fact aphasic*. A more formal language evaluation can best be performed by a speech pathologist if it is needed. However, the experienced neuropsychiatric examiner can accurately recognize aphasic patterns and often localize brain injury by that pattern. For instance, the vast majority of nonfluent aphasic patients will have an anterior dominant hemisphere lesion, where those with fluent aphasia usually have dominant hemisphere posterior lesions.<sup>13</sup> This anterior/nonfluent and posterior/fluent schema is accurate in about 85% of patients who have language dysfunction, but in 15% of cases, the reverse is true.<sup>1</sup>

Fluent aphasic speech is easily recognizable as language. The sounds flow easily and seem normal. There may be a slight press of speech in some patients. The striking finding as one listens carefully is the lack of nouns and verbs. In fact, the content consists mostly of small words such as articles, conjunctions, interjections, or even curse words. The nouns and verbs are often paraphasic. Paraphasias exist in two forms. In the first, the meaning may be substituted (semantic paraphasia) for the correct word (e.g., “I wore my car”). The other form is phonemic and a syllable may be substituted (e.g., “I wore my pat”). If a nonsense word is substituted, this is called neologistic paraphasia (e.g., “I wore my pash”). In traumatically brain-injured patients, the most common language disorder that will be heard is that of naming impairment. The patient will not be able to produce the names of common items in the environment during the face-to-face mental examination, or on a more formal examination, such as the *Boston Naming Test*, the patient will demonstrate impairment.

Most experienced clinicians can determine overall fluency in a patient by listening to the patient’s spontaneous speech. The same can be said for detection of paraphasic errors. However, subtle defects in fluency can be elicited only through specific fluency tests such as those outlined in Chapter 6. Two easily administered tests used in the face-to-face examination are the *Animal-Naming Test*<sup>14</sup> and the *FAS Test*.<sup>15</sup> Strub and Black<sup>1</sup> employ the Animal-Naming Test in their examinations. They find it particularly useful in patients who display significant deterioration of cognitive function. The patient is instructed to recall and name as many animals as possible within 60 sec. The score is the number of correct responses, as well as any paraphasic productions. A normal individual should produce from 18 to 22 animals’ names in 1 min, with the expected variation being  $\pm 5$  to 7.<sup>14</sup> This test is age sensitive, and normal individuals above age 70 produce approximately 17 names  $\pm 2.8$  in the eighth decade and 15.5 names  $\pm 4.8$  in the ninth decade. A score below 13 in an otherwise normal person should raise the question of impaired verbal fluency.

The FAS Test is a controlled oral word association paradigm. A similar test is noted in Chapter 6 and explained more fully. In the FAS Test, the patient is instructed to name as many words as



possible that begin with the letters *F*, *A*, and *S*, respectively. The person must produce these words during three 60-sec trials. Normal persons will name from 36 to 60 words, and an inability to name 12 or more words per letter attempted is indicative of reduced verbal fluency. However, this test is IQ sensitive, and full-scale IQ scores of less than 85 increase the likelihood of false positives.<sup>15</sup>

Assessing the comprehension of spoken language is done primarily by giving confrontational directions to the patient or by listening to spontaneous conversation. However, comprehension must be tested in a structured fashion in order to accurately assess this ability. Testing the patient's ability to comprehend by having him name simple objects in his visual field and asking him to point to them is generally sufficient. If one wants to increase the complexity of the examination, the patient can be required to point to an increasing number of objects in a sequence by chaining the command (e.g., "Point to the telephone, your watch, and your right eye"). A person of average intelligence without aphasia should be able to point to four chained objects or more before failure.<sup>1</sup> After confrontational pointing, the examiner can ask a series of simple and complex questions that require only "yes" or "no" answers, for example: Are we in a restaurant? Am I wearing a baseball cap today? Do you wash your clothes with gasoline? One must be careful and alternate questions that require "yes" and "no" answers randomly, as brain-injured patients may perseverate and it is not uncommon for patients to answer "yes" consecutively without knowing the correct answer to any of the questions asked. Strub and Black<sup>1</sup> recommend that clinicians not test language comprehension by asking patients to carry out motor commands such as "Show me how to light a cigarette" or "Stick out your tongue." Many aphasic patients have an apraxia and may fail the command because of impairment of higher-level motor integration and not because of poor verbal comprehension.<sup>1</sup> Roughly 90% of the population is right-hand dominant. Of the 90%, 99% or more are dominant for language within the left hemisphere.<sup>17</sup> Left-handed individuals demonstrate a substantially different pattern of cerebral language dominance. Of left-handed persons, approximately 70% are left hemisphere dominant for language. Another 13% are dominant for language in the right hemisphere, and the remainder show mixed patterns of dominance.<sup>18</sup> Individuals who have a strong family history of left-handedness are more likely to demonstrate a mixed dominance pattern. Left-handed individuals who have no family history of left-hand dominance have the strongest left hemisphere dominance for language location.<sup>1</sup> Table 4.5 describes a simple approach to language assessment.

The ability to name objects is acquired very early in our development and is one of the most basic of language functions. This ability stays remarkably stable over decades, and normal 80-year-olds generally perform as well as normal 25-year-olds.<sup>16</sup> Naming is invariably disturbed in all types of aphasia. Naming may be impaired in some traumatically brain-injured persons who otherwise do not demonstrate classic aphasia. Word-finding difficulty is closely related to anomia, the reduced ability to retrieve the nouns and verbs used in spontaneous speech. It is also frequently abnormal in persons who have suffered traumatic brain injury. Patients with word-finding difficulty may show impairment on the Picture Completion subtest of the *Wechsler Adult Intelligence Scale-III*. Anomia can be objectively tested face-to-face by asking the patient to name objects or pictures to which the examiner points in the room.

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**TABLE 4.5**  
**The Face-to-Face Assessment of Language**

- Does the speech sound dysarthric or dysprosodic?
  - Are there specific language errors in syntax, word finding, and semantic or phonemic expression?
  - Is language output fluent or nonfluent?
  - Does the patient comprehend pointing commands or questions that can be answered "yes" or "no"?
  - Can the person repeat words or sentences?
  - Can the patient read, write, and spell?
-

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**TABLE 4.6**  
**Repetition Syndromes**

<u>Impaired Repetition</u>	<u>Intact Repetition</u>
<u>Perisylvian Syndromes</u>	<u>Nonperisylvian Syndromes</u>
A. Broca's aphasia	A. Anomic aphasia
B. Wernicke's aphasia	B. Transcortical aphasia
C. Conduction aphasia	C. Subcortical aphasia
D. Global aphasia	

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The assessment of reading, writing, and spelling is fairly straightforward in a face-to-face examination, as no formal measurements generally are made. Both reading comprehension and reading-aloud ability should be tested. Most patients with a true aphasia are usually defective in both. However, either can be disturbed in isolation, but generally this is not demonstrated in traumatically brain-injured patients. Those who may show skill in one area and a defect in the other are more likely to be found within populations of stroke, tumor, or other nontraumatically brain-injured patients. Writing is tested in a fashion similar to that in a reading test. If the patient does show evidence of aphasia, he or she will undoubtedly show an impairment in the ability to write. Moreover, the types of language errors one hears in spoken language will essentially be represented as the same error in the written language. Asking a patient to write her name is not recommended, as this ability may remain even in those persons who have severe aphasia. Just as reading ability is directly related to educational experience, so too is spelling ability. In the mental status examination, spelling can be evaluated by asking the patient to spell dictated words. This type of examination has no particular specificity, and if it is important to establish an actual level of spelling competence, a standardized achievement test such as the *Wide Range Achievement Test-III* should be administered.

Asking the patient to repeat phrases or words is a very useful tool for determining the probable anatomic location of language syndromes relative to the sylvian fissure. Adequate assessment of language requires listening and testing for (1) fluency, (2) comprehension, (3) repetition, (4) naming, (5) reading, (6) writing, and (7) spelling. Within the context of the neuropsychiatric examination discussed in this text, fluency, comprehension, and repetition generally are tested face-to-face, whereas naming, reading, writing, and spelling are evaluated within the context of the neuropsychological evaluation (see [Chapter 6](#)). [Table 4.6](#) lists the separation of perisylvian syndromes from nonperisylvian syndromes. The patient can be asked to recite very simple repetition sequences such as “no ifs, ands, or buts” or “Methodist Episcopal” or “around the rugged rock the ragged rascal ran.” These simple repeating themes also are useful to screen for dysarthria. If repetition is impaired, the anatomical localization is in the perisylvian area of the dominant cerebral hemisphere. Anterior aphasias will have the characteristics of a Broca's language disturbance, whereas posterior aphasias will have the characteristic of a Wernicke's disorder. Persons with conduction and global aphasias also will demonstrate impaired repetition. On the other hand, if the patient can repeat the short phrases required, the aphasia is anatomically outside the perisylvian area and is usually an anomic transcortical or subcortical disorder. Subcortical aphasias can infrequently be an element of traumatic brain injury syndromes, particularly with brain stem involvement. Transcortical aphasias are not likely to be seen in traumatic brain injuries, but are more likely found in hypoxic or toxic brain syndromes affecting the vascular watershed areas of the cerebral hemispheres.

## MEMORY AND ORIENTATION

Moderate to severe brain injury can lead to chronic confusion and disorientation. Disorientation most often is a consequence of diffuse cerebral injury, particularly anterior injuries affecting the

limbic structures. However, during the examination of a patient who has been traumatically brain-injured, the practitioner must pay careful attention to other factors such as psychotropic medications, which may produce confusion or disorientation, as well as metabolic or endocrine disorders resulting from traumatic hypothalamic or pituitary injury.

The basic rule of testing orientation is to inquire as to the person's ability to temporally localize by person, place, and time. Orientation of person can be assessed simply by asking the person his name. Place is easily determined by asking the patient the day of the week, the month, and the current year. If the patient exhibits lack of orientation to these questions, the examiner should determine if the patient knows the season of the year. Location can be examined by asking the location of the examiner's office, the city that the office is in, the building that the office is in, and, if necessary, the state the office occupies. The ability to temporally sequence and maintain orientation can be determined by asking the patient's birth date and age; however, this also tests certain aspects of past memory, and these questions are not directed purely toward orientation.

Memory, like the attentional processing noted above, has a specific component for each sensory modality. Both long-term and short-term memory may be affected in the head-injured patient as a result of injury to the medial temporal lobes and the thalamus.<sup>19</sup> Retrograde memory (memories before the injury) as well as anterograde memory (new learning after the injury) may be involved too. Verbal memories are affected to the greatest extent in those patients who sustain a left cerebral injury, whereas patients demonstrating spatial and perceptual memory impairment generally have injuries preferential to the right hemisphere. The duration of anterograde memory impairment (posttraumatic amnesia) is an important early prognostic factor with regard to recovery. Injured persons with prolonged posttraumatic amnesia tend to demonstrate greater cognitive impairment and have poorer functional outcomes than those patients with no posttraumatic amnesia or otherwise short periods of amnesia.<sup>20</sup> Table 4.7 outlines the basic schema of memory functions.

There is no singular or universally accepted theory of memory. In fact, the diversity of approaches to memory research is the rule rather than the exception. Multiple reviews of memory studies have been written, and all current theories divide memory into different psychological or neurophysiological processes.<sup>21–25</sup> Five such processes have been described by Signoret.<sup>26</sup> He suggests that a holding process occurs in which information is retained momentarily until other memory processing can take place. This is referred to in Table 4.7 as working memory. An acquiring process then follows that encodes data selected for placement into general memory. The acquiring process can be subdivided into "chunking," which is the efficient gathering of information and subsequent "linking" (the correlation of discrete elements of information). The storing process is often called consolidation. During this function, information is placed into a permanent or semi-permanent storage system that includes new memory traces, rehearsal, and maintenance of the

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**TABLE 4.7**  
**A Schema for Memory Functions**

- Data are registered without requiring focused attention by the primary sensory cortex. If attention to the stimulus does not occur, data are lost in 1 to 2 sec.
  - Data are organized by the secondary sensory cortex and attention is brought to bear.
  - If effort is made, seven to nine items can be held. This is working memory or short-term memory, and data are held for approximately 15 to 20 sec if no effort is made to remember.
  - With rehearsal or memory work, memory becomes consolidated in 30 sec to 30 min.
  - Long-term memory is stored in secondary and tertiary (heteromodal) areas. Affect paired with a memory increases the strength of long-term storage (e.g., death of a loved one). Long-term memory is of two types: procedural/implicit (e.g., driving a vehicle, a skill) and declarative/explicit (factual).
  - Declarative memory (explicit) is composed of semantic memory (general information) and episodic memory (autobiographical experiences).
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memory information. The recall of memory is a retrieval process wherein previously learned information is recaptured and made useable. Furthermore, a scanning process occurs that allows items relevant to the person's current environmental situation to be selected from a vast array of stored memory traces.

When information is first registered within the brain, it does not require focused attention by the primary sensory cortex to which the information is being sent (e.g., primary auditory cortex). If the brain does not attend to this input within a few seconds, the information is not remembered or stored. Part of initial registration requires the immediate organization of memory data into patterns by a secondary sensory cortex that lies anatomically near the primary sensory cortex. Attention is paid cognitively to the input at this stage in memory. For instance, in the input of auditory information, individuals self-monitor their language as they speak to another person. They can immediately relate their conversation to others, and by self-monitoring, they maintain their place in the flow and sequence of their oral communication. In brain-injured patients, this pattern is often disrupted; the patient cannot self-monitor, and the speech output is fragmented or rambling. This rambling speech often presents as circumstantial thinking.

Working memory is not a true memory but an attentional process that holds information for 20 or 30 sec until it is processed further. This stage of memory function is tested by measurement of digit span or by the immediate recall of words (verbal) or diagrams (visual). Memory can be consolidated if an effort is made to remember the information by rehearsing it. This is a form of new learning, and it requires from seconds to extended minutes to be completed. Any sensory modality can "remember" in this fashion, but in the mental status examination, generally only verbal and visual components are tested. As shall be seen in later chapters, tactile learning can also be tested by procedures such as fingertip writing and finger naming while the person is blindfolded. During the mental status examination, verbal new learning ability can be tested easily by asking the person to learn a series of eight or nine words by repeating the list until it is memorized. Recall then can be tested 20 or 30 min later to confirm the level of learning that has taken place. The words chosen for the person to remember should not be easy to link phonetically or semantically, as this will provide memory cues and falsely increase the efficiency of the learning process.<sup>1</sup> Another good test of verbal learning is to read a short paragraph to the person being tested and, after 20 min, ask for a recall of the story. The specific number of memory elements in the story must be known by the examiner beforehand. Most persons without a brain dysfunction can recall 15 to 17 items of a 25-item story, and after 20 min, they should be able to remember two-thirds of their original score.<sup>25</sup> Standardized methods for assessing verbal memory are explained further in [Chapter 6](#). Strub and Black<sup>1</sup> recommend hiding five objects in front of the person to test visual memory and then, after a period of time, asking her to find the objects.

To test long-term memory, examiners either have to know something specific about the individual's life or they should ask the patient about commonly known historical facts. Adequacy of long-term memory is influenced by educational level and intellect, but most neurologically intact persons should be able to name the current U.S. president or the governors of their states. Moreover, they should be aware of major historical events or persons who have had historical impact (e.g., the September 11, 2001 World Trade Center attack or "Who was Hitler?").

As noted, immediate recall can be tested by measuring digit span, whereas concentration can be tested by using a letter cancellation test.<sup>22</sup> The inability to attain or maintain a mental sequence of information may be reflective of frontal lobe dysfunction. The anatomical location of the dysfunction leading to alterations of attention or mental sequencing is not well known.<sup>24-27</sup>

The ability to learn new information is subserved by the parahippocampal cortex (posterior mesial-temporal regions) and the cerebellar neocortex (if motor responses are involved in procedural memory). Storage is subserved by the hippocampi, amygdalae, cerebellum, and cerebral cortex. Recall is subserved by anterior mesial-temporal areas.<sup>25</sup> If the patient has difficulty with recall and is a poor historian, two analyses can be applied to this clinical situation. If the patient cannot recall details or sequences of events, the anterior mesial-temporal cortex is the most likely area to be

involved. If the patient can remember details but confuses the sequence or combines sequences in his life or environment incorrectly, this is usually due to poor executive function from frontal lobe injury.<sup>24,25</sup> As a general rule, recall of information is subserved primarily by the anterior frontal and temporal brain areas, whereas storage is confined more to the posterior temporal-parietal brain areas. The exception to this is motor information (such as in procedural memory), which is thought to be stored in the cerebellum and basal ganglia. Language and symbolic information, such as mathematics, is preferentially stored in the dominant cerebral hemisphere, whereas visual-spatial information is preferentially stored in the nondominant cerebral hemisphere.

## **VISUOSPATIAL AND CONSTRUCTIONAL ABILITY**

Since the visual system occupies such a large portion of the anterior-posterior axis of the brain, visuospatial skills can be disrupted at many levels. These include visual field cuts due to damage to the retina, optic nerve, optic chiasm, optic tract, lateral geniculate body, optic radiations, or the occipital visual cortex.<sup>28</sup> Previously, neuropsychiatric literature described the visual cortex as an analyzer. However, recent research suggests that the function of the sensory parts of the visual cortex is to act as a categorizer of visual stimuli in our environment. This categorization is according to color, texture, sound, and so on.<sup>29</sup>

Clinical experience strongly indicates that the right posterior hemisphere is more important for visuospatial discrimination than other cerebral areas.<sup>30</sup> This area can be considered dominant for visuospatial competence in the same way as the left hemisphere is generally dominant for language.<sup>31</sup> Patients with focal injuries to the nondominant parietal lobe often demonstrate impairment with spatial orientation and perceptual tasks. Visuospatial skills usually are assessed by paper-and-pencil drawing exercises. Patients may be asked to draw circles, triangles, three-dimensional cubes, intersecting pentagons, flowers, or a person. Often the patient is asked to draw the face of a clock with the hands placed at a particular time. In addition to drawing tasks, patients may be asked to manipulate by hand either tokens or three-dimensional blocks to make a series of designs.

The impaired patient, when drawing two-dimensional or three-dimensional figures, often omits major elements of the figure being copied. Angles often are rounded, the form of the figure may be lost, or the patient is unable to copy alternating designs. The clock test is a useful screening device for visuospatial neglect. If the patient has, for instance, a right hemisphere lesion, the individual may neglect the left hemispace and place all the clock numbers to the right side of the clock. Drawing tasks may also demonstrate perseverative responses in the patient. The patient may continue to draw repeating lines without closing in a figure or, for instance, when drawing numbers on a clock, may repetitively draw 1 and forget the numerical sequence 1, 2, 3 ...

Visuospatial ability can be entirely a cognitive ability. When constructional ability is added to the screening for visuospatial skills, it must be remembered that not only is intact vision a prerequisite for constructional ability, but so are intact motor coordination, strength, praxis, and tactile sensation. Patients who fail constructional tasks may require testing for other disorders that could interfere with their ability to complete the task. For instance, in addition to visual deficits, the patient also could have writing dyspraxia or visual agnosia.

Constructional ability and visuospatial function are absolutely essential to performing many everyday activities. In fact, neuropsychiatric and neuropsychological testing has been discussed in the medical and psychological literature under the topic of ecological validity; that is, is there a relation between test performance and real-world abilities? Constructional ability and visuospatial functions are necessary to drive vehicles, function in a kitchen, use a vacuum cleaner, read maps, drive around a city, use a computer, and generally function within the environment and remain topographically and geographically oriented. Impairment of constructional ability and visuospatial ability is seen generally in individuals who sustain traumatic brain injury sufficient to produce tissue-based brain injury. Often, this level of injury is demonstrable on structural or functional brain imaging.

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**TABLE 4.8**  
**Assessment of Visuospatial/Constructional Ability**

- These skills usually are assessed by paper-and-pencil copying of two- or three-dimensional figures.
  - Skills can be disrupted by visual field cuts from retinal, optic nerve, chiasm, optic tract, lateral geniculate body, optic radiation, or primary visual cortex damage.
  - For those patients who cannot use their hands, cognitive identification of geometric shapes in different planar orientations can be attempted.
  - Focal injuries to the parietal right hemisphere are more likely to impair visuospatial function than analogous left hemisphere injuries.
- 

Some traumatically injured persons who sustain a brain injury may also have an inability to use their upper extremities. In examining visuospatial ability in these individuals, one cannot rely on motor activity for the assessment. An alternative approach is to ask the patient to identify particular geometric figures among a series of figures oriented in different planes. These are presented to the patient visually, and the person can then respond verbally to the examiner's questions about figure orientation. These tasks require cognitive manipulation of figures without the need for motor output. [Table 4.8](#) outlines important elements of visuospatial and constructional ability.

## EXECUTIVE FUNCTION

In the traditional psychiatric mental status examination, *executive function* is not a term usually expressed by psychiatrists. Executive function generally refers to abilities subserved by the prefrontal cortex or the portion of the frontal lobe lying anterior to the unimodal motor association cortex. Some neuropsychiatrists describe these functions as frontal lobe abilities.<sup>25</sup> Behavioral neurologists often describe these functions as higher mental control abilities<sup>24</sup>; whereas psychiatrists may use terms such as abstraction ability, conceptualization, insight, and judgment.<sup>2</sup> Clinical neurologists often subserve executive function under the rubric of higher cognitive function.<sup>1</sup> Neuropsychologists are the most likely clinicians to use the term *executive function*.<sup>3</sup> Lezak conceptualizes executive functions as having four components: (1) volition, (2) planning, (3) purposive action, and (4) effective performance.<sup>3</sup> Stuss and Benson propose four higher control functions attributed to the prefrontal cortex. These include (1) sequencing, (2) drive, (3) executive control, and (4) future memory.<sup>27</sup> Ingvar adds self-awareness as a fifth function.<sup>32</sup>

The screening of executive function by face-to-face examination has been systematized recently by a number of authors. Power and colleagues have developed a screening test for detecting dementia in patients with AIDS. This instrument includes measures of attention (repeating four words), measures of memory, free and semantically cued recall of words, measures of psychomotor speed (writing the alphabet), visuospatial function (copying a cube), and response inhibition (anti-saccadic eye movements).<sup>33</sup> The *Executive Interview (EXIT)*<sup>34</sup> has been tested and proves to be a better predictor of independent functioning in several geriatric test samples than the *Mini-Mental State Examination* of Folstein et al.<sup>35</sup> The *Behavioral Dyscontrol Scale*<sup>36</sup> is designed specifically to predict everyday functional capacity. This test instrument uses go/no-go motor sequencing and alphanumeric sequencing tasks to analyze the ability to organize goal-directed behavior. The alphanumeric sequencing portion provides a brief measure of psychomotor speed and working memory. For those patients with motor or movement disorders, the *Frontal Assessment Battery*<sup>37</sup> may be useful. This instrument requires 5 min to perform and surveys motor sequencing, spontaneous word-list generation, and response inhibition on a go/no-go task. [Table 4.9](#) describes common signs or symptoms of executive dysfunction.<sup>38</sup>

Benson has listed executive functions as executive control abilities.<sup>24</sup> He and other behavioral neurologists attribute this umbrella term, *executive control*, to a number of mental functions considered to be subserved by prefrontal brain activity. These include anticipation, goal selection,

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**TABLE 4.9**  
**Signs and Symptoms of Executive Dysfunction**

- Outrageous, disinhibited behavior
  - Impulsiveness or perseveration of oral or written information
  - Reduced ability to express words
  - Poor visual or auditory attention
  - Reduction in motivation or drive
  - Inability to switch sets or inhibit responses
- 

response formulation, monitoring of the planned response, initiation of response, and monitoring of the response and its consequences. As we shall see later in [Chapter 6](#), these are generally evaluated during neuropsychological assessment, particularly with executive function tests such as the *Category Test*, the *Wisconsin Card Sorting Test*, the *Trail-Making Tests*, and the *Stroop Test*. These complex control abilities are very difficult to quantitatively assess within a simple face-to-face mental examination paradigm.

## AFFECT AND MOOD

*Affect* refers to the outward display of emotion, that is, emotions that can be visually or auditorially perceived by the examiner. *Mood* is a term for the unobserved internal, perceived, or felt aspects of emotion. Affect and thought content are generally congruous and well correlated. If the examiner determines a significant disparity between the outward display of emotion (affect) and the content of thought, as expressed by the patient, one should suspect a psychiatric disorder, an anatomical or physiological disconnection of limbic or subcortical areas associated with emotional regulation, or a metabolic-toxic derangement of emotional control.<sup>28</sup>

Mood and affect often are difficult to distinguish from each other within the context of a neuropsychiatric examination. This is particularly true if there is an overlay of substantial organic dysfunction displayed as neglect or denial or other higher-order cognitive processing disturbance. Some psychiatrists rely on variability to differentiate mood from affect. Those psychiatrists describe mood as a consistent, sustained-feeling state, whereas affect is the moment-to-moment expression of the feelings related to the mood or distinct from the mood.<sup>2</sup> As we have seen in this text (see [Chapter 2](#)), disturbances of mood and affect are extremely common following traumatic brain injury of any type. In fact, mood disturbances are probably the most common psychiatric manifestation of traumatic brain injury and may persist for decades.<sup>97</sup>

The assessment of mood is performed within the context of the entire brain injury examination. However, within the mental status examination, mood is assessed in general by observation of the person being examined and by careful attention to the behavioral observations and context of the interview determined within the entire examination. Patients should be encouraged to describe their moods in their own words. However, many laymen do not understand the term *mood*, and the skillful examiner must ask for the feelings of the person in a number of creative ways. If the patient cannot describe her emotional state in her own words, the examiner must explicitly ask questions to elicit the prevailing mood. Open-ended questions should be offered first, such as “How have you been feeling in the last few days?” or “How have you been feeling lately?” or “How do you feel right now?” Follow-up questions are required to determine whether the mood described by the patient at the time of examination has changed since her injury. For instance, is the mood more intense now than prior to the injury, less intense than prior to the injury, or the same? The term *depression* has variable meanings to different people. If the patient uses a term such as depression or sadness, the examiner should ask follow-up questions to determine the intensity of the feelings and, again, whether there has been a change in the feelings since the injury. As we shall see later

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**TABLE 4.10**  
**Descriptors of Affect/Mood**

Dysphoric:	sad, hopeless, grieving, despondent, distraught
Euthymic:	well feeling, comfortable, happy, friendly, pleasant
Euphoric:	elated, ecstatic, hyperthymic, giddy
Apathetic:	flat, bland, dull, lifeless, nonspontaneous
Angry:	irritable, argumentative, irate, belligerent, confrontational
Apprehensive:	angry, fearful, scared, worried, nervous, frightened

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in this text, specific standardized measurements of mood can be made using instruments, such as the *Minnesota Multiphasic Personality Inventory-2* or the *Personality Assessment Inventory*, which lie among other instruments available for assessing overall mood function.

One of the most difficult delineations for the examiner of a traumatically brain-injured patient is to distinguish alterations of mood from changes in outward behavior, such as apathy, somnolence, and the fatigue syndromes, that often accompany brain injury. As noted previously in [Chapter 2](#), hypersomnolence syndromes are frequently seen following traumatic brain injury, and the apathetic disorders of frontal lobe dysfunction are not uncommon. Very careful inquiry of the patient's mood state must be made further in order to differentiate dysfunction of brain stem or cerebral drive from emotionally based alterations of mood.

In the poorly educated or severely brain-injured patient, describing mood may be particularly difficult. Psychiatrists have previously coined the term *alexithymia* to describe the patient who cannot assign descriptors for emotions.<sup>2</sup> Many patients are unable to describe their emotions even in their own everyday language. Furthermore, the brain-injured patient who has sustained an alteration of language or prosody may demonstrate significant difficulty describing his mood. A particularly challenging evaluation of mood occurs in the patient who has sustained the disinhibited frontal lobe disorder described in [Chapter 2](#). This individual may have an outward appearance of euphoria, but the subjective feelings of euphoria and elation are not present. The outward manifestation of affect in this case is very incongruent with the patient's perceived feelings. In fact, the disinhibited patient may feel agitated or dysphoric while outwardly displaying an affect consistent with euphoria and irritability. This has often been termed *pseudoeuphoria*, indicating that the outward expression of affect in these brain-injured patients does not match congruently with an inward feeling of elation. [Table 4.10](#) describes clusters used to describe affect (observable) and mood (internal and subjective) in brain-injured patients.

Whereas mood is similar to the carrier signal of radio waves used for transmission of electromagnetic signals, affect is analogous to the moment-to-moment changes in amplitude of the signal transmitted over the carrier wave. Affect is conveyed to the examiner by the output systems of the brain modulated by the emotional tone of the brain. For instance, with language, the carrier wave is the symbolic aspects of the language, whereas prosody (music or melody of language) is the alterations of mood content expressed with changes of voice inflection, voice emphasis, body language, motor activity, hand gestures, etc. Thus, affect can be conveyed by the tone of the voice, movements of the hands or feet, muscles of facial expression, motor activity level, and posturing. The examiner's own right brain posterior language decoding systems allow her to be empathetic and feel sad herself while examining a depressed patient or feel concern while examining the disinhibited patient. A sense of uneasiness may be felt by the examiner in those patients who are hostile, suspicious, or paranoid. These subtle detector systems are part of all humans, and the examiner is advised to pay careful attention to them. Many messages are expressed emotionally rather than verbally. If a brain-injured patient is experiencing a dysfunction of modulating or regulating systems, the examiner's emotional detector systems may be acutely sensitive to the expressed affect. Affect is often described within five basic parameters: (1) appropriateness, (2)



intensity, (3) mobility, (4) range, and (5) reactivity.<sup>2</sup> Appropriateness helps the examiner determine whether the affect is congruent or incongruent with the mood. The intensity level of affect enables the examiner to determine whether the person is apathetic. The mobility level of affect is often described as labile or constricted. The range of affect may be reduced in patients with alterations of affective drive systems. The reactivity of affect can vary from hyperreactive to nonreactive or nonresponsive.

## THOUGHT PROCESSING, CONTENT, AND PERCEPTION

The reader is referred to two excellent classic monographs providing descriptors of thought processing, content, and perception beyond what can be offered in this text. Lishman<sup>39</sup> and Fish<sup>40</sup> have eloquently described the psychiatric parameters of thought. From a neuropsychiatric standpoint, the evaluation of thought in a traumatic brain injury examination is somewhat more constricted than would be performed in the overall psychiatric examination of patients presenting with specific psychiatric disorders not related to traumatic brain injury. The ability to think symbolically is fundamental to the human being. Obviously, a traumatic brain injury may alter the ability to think due to structural or functional changes in brain processing. In the classical psychiatric mental status examination, thought is assessed for concreteness by proverb interpretation or interpretation of similarities. Insight and judgment is inferred by the interview process and questioning. More formal assessment of these abilities are described further in the cognitive and behavioral assessment sections of this text (see [Chapters 6 and 7](#)).

By allowing patients to speak in an open-ended fashion, the examiner is able to observe the style of thought, process of thinking, and determine the content of the thought. Simply put, by listening to the patient, the examiner wants to determine if the patient can go from point A to point B within the conversation. This requires not only intact cerebral systems directed at thinking, but also the ability to self-monitor one's language while speaking. Numerous descriptors of the thinking process have been offered in psychiatric texts. [Table 4.11](#) outlines some of the abnormalities that may be detected by the examiner, and these terms are described next.

Perseveration can occur with disruptions of working memory and recent memory. The brain-injured patient is unable to self-monitor what he has told others and may repeat themes. It is not unusual for a brain-injured patient to tell his family what he did 30 min earlier and continue to repeat that same theme. For more severe brain injuries, motor perseveration can also occur. As noted previously, when a person attempts to draw a clock, he may perseverate by writing "1" and failing to put the numbers in the appropriate positions around the clock.

A simple way to characterize circumstantiality is to think of traveling the shortest route from Louisville to Chicago. The circumstantial highway route might take a person from Louisville through West Virginia, Pennsylvania, Ohio, Indiana, and eventually to Chicago, rather than the quicker route of going straight north through Indiana and entering from the west into Illinois and then to Chicago. The circumstantial patient is able to go from point A to point B in the conversation, but due to self-monitoring deficits, the individual is overinclusive and overly detailed. Often, it is necessary during the interview to structure the patient so she can stay on topic, rather than advising

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**TABLE 4.11**  
**Thought Processing Defects**

Perseveration	Neologisms
Circumstantiality	Echolalia
Loose associations	Clang associations
Tangentiality	Thought blocking
Flight of ideas	<i>Witzelsucht</i>

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the examiner of all the physicians she has seen, all the trials and tribulations she has undertaken, etc., when merely a simple question was asked, “How do you come to be here today?” Circumstantiality is a very frequent outcome of traumatic brain injury, particularly when frontal systems are damaged.

Loose associations are also fairly common in some brain-injured patients, but far less so than circumstantial thinking. Loose associations are much more common in psychotic patients, particularly those with schizophrenia. In loose associations, words are intact, but the syntactical associations within the paragraph do not connect well and logical meaning and connection are lost. Tangentiality requires careful listening on the part of the examiner to detect. Initially, conversation with tangential patients seems to be going well. However, the interviewer may suddenly realize that the topic has taken a path different than the goal of the original question. Instead of going from point A to point B in the conversation, the patient skips like a rock sailing off the top of a pond onto the bank and picks up an unrelated topic. Flight of ideas is a form of tangentiality wherein the disordered thought occurs very quickly and frequently and the tangents occur every one or two sentences or so, instead of paragraph to paragraph. Flight of ideas is, of course, more commonly seen in manic patients, but it also can occur in the orbitofrontal syndrome and other disinhibition disorders following frontal traumatic brain injury. It again represents an inability on the part of the patient to self-monitor where he is in his discourse.

Neologisms are commonly seen in either psychotic patients or patients with advanced dementia.<sup>1</sup> These are novel, idiosyncratic words and are not found commonly in traumatically brain-injured patients. They often are associated with the classical aphasias following stroke syndromes. Idiosyncratic words frequently sound elaborate and plausible. Neologisms often are associated with delusions in psychotic patients, but rarely so in brain-injured patients. Patients demonstrating echolalia repeat questions or statements made by the examiner. Sometimes, traumatically brain-injured patients will repeat the question to the examiner, as their working memory may be impaired and they must repeat or echo to catch the phrase, if you will, in order to keep it in storage long enough to answer the question. This is a different phenomenon than the echolalia often seen in manic patients. Echolalia is much more common in schizophrenia and mania, is often associated with catatonia, and occurs far less so in brain injury, but it does present in some patients with frontal dementias.<sup>2</sup>

Clang associations are a form of phonemic distortion. The phonemes of words are connected by sound rather than by meaning, and they have no semantic importance. These oral expressions can occur following traumatic brain injury, and in psychiatric medicine, they are often referred to as clang associations, whereas behavioral and classical neurologists may refer to these as phonemic paraphasias. Thought blocking is relatively rare and generally not associated with traumatic brain injury. This is observed most frequently in the psychosis of schizophrenia, but it is seen also in some delirious patients in the acute care setting. A thought is lost in midsentence as a disorder of language monitoring; moreover, the patient cannot maintain the phrase or sentence in working memory long enough to properly process it within the language output systems. *Witzelsucht* is a facetious punning style that some patients exhibit in association with the disinhibited syndrome of infraorbital brain injury. These jocular patients can be quite playful, and their thought processing deficit can be misidentified as hypomania.<sup>28</sup> Some older texts also call this phenomenon *moria*.<sup>40</sup>

The content of thought is particularly important for the examiner to determine when assessing mood or anxiety in posttraumatic brain disorders. Traumatized patients may have recurring intrusive preoccupations with sounds, images, and other stimuli that remind them of the accident. As noted in [Chapter 2](#), many brain-injured patients may develop a posttraumatic stress disorder. As Harry Stack Sullivan said many years ago, one must “listen with a third ear” when assessing patients. This important admonition remains valid today. The examiner should pay special attention to the opening minute of the examination and to any unstructured moments throughout the interview.<sup>2</sup> The unstructured portion of the interview allows one to gauge the processes of thinking and also to assess the themes that are important to the patient. For instance, the patient’s thought may be

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**TABLE 4.12**  
**Perceptual Distortions**

<b>Form</b>	<b>Distortion</b>
Hallucination	Perceptual experience in the mind without a sensory stimulus
Illusion	Sensory misinterpretation of external stimuli
Derealization	The external environment is unreal
Depersonalization	One's self is unfamiliar
Autoscopy	A hallucination of seeing oneself
<i>Déjà vu</i>	Having previously lived the present setting
<i>Jamais vu</i>	Current previously known setting is not familiar

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replete with themes of anger, guilt, diminished self-esteem, fear of intimacy, and desire for closeness. Brain-injured patients often see themselves as different from others and not capable of being loved. Moreover, the patient with an organic neglect or denial syndrome generally will demonstrate a large discrepancy between the content of her thinking and the problems within her observed behavior. Open-ended questions without structure are more fruitful in gaining the content of thinking. For instance, the examiner might ask, “What kinds of problems have you been having lately?” or “Tell me something about yourself.” If the patient is too disorganized in thinking or too language impaired to provide useful information, then the examiner will have to move on to a more structured form of interview. If the examiner learns of specific problem areas in the thinking, these will require interview follow-up. It is particularly important for the clinician to explore delusional content, homicidal ideas, paranoid themes, phobic statements, preoccupation with traumas or healing, ruminations, and suicidal ideas. If any of these themes are discovered, then it is paramount for the examiner to explore the level of distress these themes may cause the patient. In a person who has suspended reality, abnormal ideas may cause little distress, whereas some brain-injured persons may be so worried and focused upon the aversive content of their thinking that they cannot maintain a goal direction for rehabilitation. As we shall see later in this text, the level of disorganization and the level of communication difficulty are inversely proportional to how the brain-injured person will function postrecovery.

By definition, delusions are not interpreted by the patient as being false ideas. Moreover, patients with psychotic disorders usually have impaired insight. Whereas loss of insight indicates very substantial mental disorder, partial insight is a positive prognostic sign following brain injury. [Table 4.12](#) describes the perceptual disturbances that can be seen in patients with altered cognitive processing. As noted in [Chapter 2](#), psychosis is a possible outcome following traumatic brain injury. However, insight is often impaired and may be associated with neglect syndromes. Critchley has described a wide range of organic neglect syndromes,<sup>41</sup> and Prigatano and Schacter have further provided a more modern review of deficits of awareness following brain injury.<sup>42</sup> Substantial perceptual processing deficits associated with parietal lobe or thalamic injury can lead to numerous perceptual distortions and even hallucinations. Temporal lobe injury can cause *déjà vu* or *jamais vu*.

Hallucinations can occur in any sensory modality. They are not very common following head injury unless there has been some substantial injury to the limbic or deep subcortical brain system. Hallucinations can be auditory, visual, tactile, olfactory, or gustatory (taste). These are perceptual experiences in the mind or consciousness of the patient without a sensory input. On the other hand, illusions are sensory misinterpretations of real stimuli. For instance, a visual image is misinterpreted to be an object that it is not. Autoscopy is the hallucinatory experience of seeing oneself. Its best common description is that often reported when patients describe “near-death experiences.” *Déjà vu* is the perception of having previously seen or lived in the current setting. It is a sense that one has “been here before.” On the other hand, *jamais vu* is just the opposite; that is, the present familiar environment seems strange and alien, as if one has not been there previously.

Visual hallucinations are more common following traumatic brain injury than auditory hallucinations unless the traumatically brain-injured person has a prior history of schizophrenia or other psychosis. Visual hallucinations, of course, are much more likely to occur if there is damage to the visual system, particularly the heteromodal processing centers in the vicinity of the calcarine fissure. Patients with cortical blindness due to bilateral occipital lesions may confabulate the description of what they cannot see (Anton's syndrome). This is in actuality a type of visual neglect syndrome.<sup>1</sup> Olfactory hallucinations are relatively rare but can occur with the frontal injuries commonly associated with traumatic brain injury, as the anterior brain structures are more likely to be injured than the more posterior structures. Olfactory and gustatory hallucinations are most frequently encountered following temporal lobe injury, which in turn may lead to seizures in the uncus or entorhinal areas. Somatic (tactile or haptic) hallucinations are rarely, if ever, seen following traumatic brain injury. *Déjà vu* and *jamaïs vu* not only occur following parietal lobe injury, but are also frequently encountered in temporal lobe injuries leading to posttraumatic seizure disorders. Derealization and depersonalization are most likely to be seen in traumatically brain-injured patients in the acute care setting, and these may accompany delirium or encephalopathy following trauma. If present in the acute care setting, these generally do not persist into the rehabilitation period or chronic phase of the brain injury disorder. It should be fairly obvious that, within the context of a brain injury mental examination, these perceptual disturbances must be explored through the interview process, as there is no standardized means to measure perceptual distortions within a face-to-face examination.

## RISK TO SELF OR OTHERS

A careful review of the world medical literature will not find that traumatic brain injury in the acute phase is significantly related to the onset of suicidal ideas or even suicidal attempts. On the other hand, depressed people may become traumatically brain-injured due to impulsive acts, suicidal acts, or by placing themselves in harm's way. Even this outcome seems to be fairly low in likelihood, and where specific instances of motor vehicle crashes thought to be suicide have been studied, they have failed to demonstrate a convincing suicidal link to the automobile crash.<sup>43</sup> It is possible that there is a link with posttraumatic seizures between psychopathology and suicidal intent. Psychopathology is seen in persons with posttraumatic temporal lobe seizure disorders, and there is evidence of an increase in impulsiveness, irritability, emotional lability, paranoia, and other behaviors that may have a negative impact upon a person's intent at self-preservation.<sup>44</sup> However, in the chronic phase of traumatic brain injury, suicide risk increases and covaries with the level of depression.<sup>101,102</sup> Clearly, questions about suicidal intent should be asked of every person who has a depression within the context of a traumatic brain injury examination. These are best explored by skillful and compassionate interview techniques.

## MENTAL SCREENING EXAMINATION

A simple face-to-face approach<sup>45</sup> to the neuropsychiatric examination of the adult traumatically brain-injured patient is provided in [Table 4.13](#). A fundamental test of concentration is to ask the patient to count backward from 20 to 1. Any sequence can be used, and Trzepacz and Baker have suggested counting backward starting at 65 and stopping at 49.<sup>2</sup> They point out that this is a good test for the elderly, as serial 7s may be too sensitive to the normal effects of aging. However, serial 7s in persons under 60 or 65 years of age is very sensitive in detecting impairment of working memory and vigilance. The test is based upon parallel tracking and maintaining two operations in the mind at once. After one subtracts 7 from 100, 93 is now in mind, and the person must keep 93 in mind while he again subtracts 7 to produce 86. This double tracking is a sensitive test of working memory.<sup>45</sup> Strub and Black test short-term verbal memory with four simple words: brown, tulip, eyedropper, and honesty. They picked these words in particular for their semantic and phonemic

**TABLE 4.13****Face-to-Face Neuropsychiatric Screening Methods for Trauma-Induced Brain Injury in Adults**

Domain	Task	Poor Performance Significance
Attention	“Count from 20 to 1 backward.” Serial 7s: “Subtract 7 from 100, then 7 from that answer, and continue.”	Concentration impairment <sup>2</sup> Impairment of working memory <sup>2</sup>
Memory	Short-term verbal memory: “Remember <i>brown</i> , <i>tulip</i> , <i>eyedropper</i> , and <i>honesty</i> .” Short-term visual memory: “Copy these three shapes and remember them [square, triangle, and circle].” Evaluate orientation to person, place, or time. Past memory: “Who is the president? Which country bombed Pearl Harbor? <sup>3</sup> In which city was the World Trade Center?”	Less than 3 words after 10 min: impaired frontosubcortical function of verbal memory <sup>1</sup> Two or less drawn after 3 min is impaired. <sup>35</sup> Normal is perfect responses or off by 1 day on date. Sensitive to low educational level. <sup>46</sup>
Language	Ask for names of common objects in visual space. Repeat: “Methodist Episcopal; the little boy went home; the fat, short boy dropped the china vase.”	Left perisylvian damage if attention is normal. <sup>1</sup> If intact, language dysfunction outside perisylvian area. If impaired, Broca’s, Wernicke’s, or conduction aphasia. <sup>1,22</sup>
Visuospatial	“Copy two intersecting pentagons.” “Draw a clock and put the numbers in place. Set the time for 3 o’clock.”	If impaired, right hemisphere damage. <sup>34</sup> If numbers skewed to right or left, check for visual neglect. Distortion of numbers may represent right hemisphere damage. <sup>78,98</sup>
Executive	Response inhibition: “Tap twice each time I tap once. Now when I tap twice, you do not tap at all.” Frontal lobe word generator: “Say all the words you can think of that start with <i>S</i> [in 1 min], but no people’s names, cities, or places.”	If impaired, orbitofrontal damage. <sup>45</sup> If impaired, dorsolateral frontal lobe or semantic memory system. <sup>45</sup>

diversity to avoid memory cues.<sup>1</sup> Short-term visual memory can be screened by asking the patient to copy simple figures that all persons learn in preschool and elementary school. Asking the patient to copy a square, triangle, and circle and then redraw these after 3 min is a sensitive screening test. Obviously, only the most significantly impaired person will fail this test. On the other hand, since the test is so easy to pass, it is not a useful measure of subtle visual memory loss. By checking orientation, the examiner is actually measuring how the person monitors and incidentally records time (episodic memory). When we arise each morning, we must reorient ourselves to a new day and monitor our place and time throughout the day. We are required to correct for the month every 28 to 31 days, and we must correct for the year annually. We use episodic memory (autobiographic recording) to accomplish orientation. As the person being examined came to the examiner’s office, she had to geographically orient herself and make the obvious connection that she had physically gone from point A to point B and topographically changed location. Only the most severely impaired will not be oriented to person. Performance on orientation tasks correlates with educational level. College graduates performing poorly will miss the day by 1, whereas persons without a high school education may miss by 2 or even 3 days, even if functioning normally. Approximately 8% of normal uneducated individuals may incorrectly identify the month.<sup>46</sup>

In screening language, it is very easy to simply ask for names of common objects in the person’s visual space. Physicians for decades have asked persons what is the name of a watch, eye, pencil, clock, etc. Anomias are common following traumatic brain injury, and if associated with aphasia, they suggest left perisylvian damage, assuming that attention is normal.<sup>49</sup> The patient should be asked to repeat words and phrases. As noted previously, if repetition is intact, language dysfunction

lies outside the perisylvian area. If language is impaired within the context of aphasia, the patient should be further evaluated to determine if she has an anterior or posterior aphasia, or Broca's or Wernicke's aphasia, respectively. Conduction aphasia must also be considered.<sup>1</sup>

A simple screening test for visuospatial function is accomplished by asking the person to copy two intersecting pentagons. This is a widely used screening technique formalized within Folstein et al.'s examination.<sup>35</sup> The right nondominant hemisphere usually is preferential for this type of visuospatial task, and if impaired, it suggests damage within the right cerebral hemisphere. Asking the patient to draw a clock and put the numbers in their proper places is a useful technique for determining if neglect or visual field defects are present.<sup>98</sup> Moreover, as discussed previously, written repetition of numbers will demonstrate motor perseveration, and structural distortion of numbers may represent visuospatial defects from right hemisphere damage.

Executive function can be screened quickly by evaluating response inhibition and the ability to generate words. [Table 4.13](#) demonstrates an easy way to measure response inhibition. This is commonly seen following orbitofrontal damage, and orbitofrontal impairment is frequently seen following traumatic closed-head injury. The frontal language systems also provide word-generating capabilities. The *Controlled Oral Word Association Test* or *FAS Test* is often used by neuropsychologists to determine word-generating capabilities.<sup>3</sup> By asking the patient to generate as many words as possible that start with the letter *F*, *A*, or *S*, the dorsolateral frontal cortex or semantic memory system can be screened.<sup>1,45</sup>

## THE ADULT NEUROLOGICAL EXAMINATION

A significant and important portion of the neuropsychiatric examination includes a physical neurological examination. The extent of any residual deficits varies widely from patient to patient and depends on the nature of the trauma and the localization of the brain injury. Open-head wounds or penetrating brain injuries (e.g., depressed skull fracture or gunshot wound) most often cause a focal brain injury under the site of impact due to skull fracture, brain contusion, laceration, hemorrhage, or traumatic necrosis of underlying brain tissue.<sup>47</sup> In closed-head injury due to acceleration–deceleration as noted in [Chapter 1](#), insults to the brain occur primarily to the frontal and temporal poles and occasionally occipital lobes or posterior parietal areas. Diffuse axonal injury is a frequent outcome of acceleration–decelerations of significant velocity and occurs as a result of axonal disruption. This often leads to injury within the subcortical white matter.<sup>48</sup> The brain structures most likely to demonstrate injury following this type of trauma are the corpus callosum (at the splenium or genu), superior cerebellar peduncles, basal ganglia, and the periventricular white matter.<sup>5</sup> [Chapter 1](#) points out that the brain also may be damaged as a result of secondary injury after head trauma as a result of complications of the injury, including edema, hypoxia, hypotension, brain shift herniation, and compensatory hydrocephalus.

While the neurological examination is a key element in the evaluation of the traumatically brain-injured patient, the focus of the examination varies depending on the stage of the patient within recovery. Clinicians providing neuropsychiatric cognitive examination of traumatically brain-injured patients will rarely be asked to examine trauma patients in the acute care setting. That is best left to neurosurgeons and neurologists. On the other hand, those clinicians involved in rehabilitation, such as physiatrists, or postrecovery cognitive assessment, such as psychiatrists, neurologists, and psychologists, are focusing upon the specific physical, neurologic, cognitive, and behavioral deficits that will potentially limit the patient from a functional standpoint or that are of importance in assessment of damages that may be significant to the patient in a legal setting. Hemiparesis may affect the patient's ability to perform ordinary activities of daily living without help. Spasticity may impede locomotion and the use of wheelchairs and rehabilitative devices. Thus, this section focuses upon identifying neurological deficits that are common in traumatically brain-injured patients and functional impairments, particularly as they relate to rehabilitation or

postrecovery function. If more complete assessment beyond the limits of the neuropsychiatric examination is required, the patient should be referred to an appropriate neurologist, neurosurgeon, or psychiatrist for evaluation, and then that information should be made available to the neuropsychiatric examiner. Moreover, while a neurologist ordinarily would perform a simple general screening mental status examination, that has been covered before in great detail and will not be included as part of the neurological examination in this text.

## **CRANIAL NERVE EXAMINATION**

### **Cranial Nerve I**

Traumatically brain-injured patients who have suffered a loss of consciousness may have olfactory dysfunction as high as 20%.<sup>50</sup> In general, olfactory dysfunction occurs in approximately 7% of patients sustaining traumatic brain injury.<sup>10</sup> In mild head injury, cranial nerve I is the most commonly affected cranial nerve. Anosmia is usually caused by frontal or occipital blows, and the clinician will have to consider whether a fracture has occurred through the cribriform plates. As frontal brain parts are contused or slide forward within the cranial vault, the olfactory epithelium to the entorhinal cortex may be affected.<sup>51</sup> If the patient can smell but has a distortion of the normal sense of smell (parosmia), injury to the orbitofrontal and temporal lobes may have occurred.<sup>52</sup> Olfactory function supplies not only the primary sense of smell, but also part of the pleasure of taste. Food substances in the mouth send aromatic molecules upward through the nasal passages to the olfactory apparatus, and this leads in part to the appreciation of taste. Functional impairment of the olfactory apparatus can be dangerous. If the person is unable to smell smoke, gas, or other noxious substances, his life could be at risk.<sup>53</sup>

The examination of smell is best accomplished by using common environmental odors. The use of essential oils of peppermint and anise may work nicely. Peppermint has been smelled by virtually everyone, and anise is a frequent component of licorice and other candy substances often eaten by children. Examination of smell should not involve noxious stimuli such as ammonia or substances containing high amounts of alcohol. Strong chemicals or alcohols will stimulate the trigeminal nerve within the mucous membranes of the nose rather than the olfactory nerve, and the examiner will be unable to distinguish whether the patient can appreciate odors.

### **Cranial Nerve II**

Of all patients who sustain a traumatic brain injury, approximately 3% will demonstrate a persistent visual field defect, impaired visual acuity, or blindness.<sup>54</sup> The optic nerve or anterior visual pathways are affected in approximately 5% of persons who sustain a traumatic brain injury.<sup>55</sup> Since most of the traumatic brain injuries are frontal injuries, the optic nerve and its pathways may be injured due to bone fractures, shearing forces, stretching, contusion, or loss of blood supply.<sup>56</sup> Depending upon the location of the lesion, the visual deficit may include monocular blindness due to optic nerve injury, bitemporal hemianopia due to ischemia of the optic chiasm, homonymous hemianopia due to injury of the optic radiations, and cortical blindness due to an occipital lesion in the calcarine cortex. Occipital brain lesions are more common after head injury in children than adults, but they are usually transient.<sup>20</sup>

Examination of vision is performed by confrontational testing while standing directly in front of the patient. If there is a unilateral optic nerve injury, neither the ipsilateral nor the contralateral pupil will be constricted when light is directed into the injured eye. On the other hand, both pupils will constrict when light is directed into the unaffected eye. The swinging flashlight test can be used to measure pupillary light response if the lesion is prechiasmatic. By shining a light in one eye and swinging it back and forth to the other eye, the pupil on the injured side will dilate as the light is swung to that eye (Marcus–Gunn phenomenon). If the optic nerve is atrophied, during

fundusoscopic examination the clinician will note that the optic disc is pale. If there is no optic nerve or prechiasmatic injury, visual acuity can be tested using the handheld Snellen acuity chart or a near-vision reading card. If these are not available, the person can be asked to read simple materials such as a newspaper.<sup>56</sup>

Visual fields are also assessed by confrontational testing. The neurological terms used for visual field descriptors are confusing, and the reader will find some texts describing visual field cuts as hemianopsia, while other texts will call them hemianopia. These terms are equivalent. When testing visual acuity, the patient should wear his prescription glasses if he has them. This is because poor visual acuity caused by retinal or optic pathway dysfunction cannot be corrected by eyeglasses. If visual acuity is corrected by eyeglasses, the abnormality is generally in the ocular lens or other parts of the refraction system and not in the visual pathway.

When assessing patients for visual field defects, it is simple to remember that if a visual defect is monocular, it is in the eye or prechiasmatic. If the visual defect is bitemporal, the lesion is in the optic chiasm. Distal to the optic chiasm, a lesion in the optic tract will produce either left or right hemianopia. Lesions distal to the lateral geniculate body will affect either the inferior or superior radiations, and thus will produce a superior or inferior quadrantanopia. Lesions within the occipital lobe visual processing system will cause impairments ranging from alterations of visual processing and color recognition to cortical blindness.

### **Cranial Nerves III, IV, and VI**

During traumatic head injury, the oculomotor, trochlear, or abducens nerve is injured in 2 to 8% of patients. The most common causes of injury to these nerves result from orbital wall fractures or a fracture in the cavernous sinus due to a basilar skull fracture.<sup>57-59</sup> As noted earlier, brain stem injury may also occur with trauma to the head, and this may in turn directly injure cranial nerve nuclei or their intranuclear pathways.<sup>60</sup> Conjugate horizontal gaze requires a coordination in contractions between one lateral rectus muscle (nerve VI) and the medial rectus muscle of the contralateral or opposite eye (nerve III). The frontal gaze center within the frontal lobe initiates voluntary horizontal conjugate gaze and projects nervous impulses to the contralateral (opposite) pons. When one examines the patient for horizontal conjugate gaze to command, such as when examining for horizontal nystagmus, this function is a response to vestibular input and is under cerebellar control, and thus is an alternative neuroanatomical pathway for conjugate horizontal gaze and differs from that which is initiated voluntarily. However, both voluntary and involuntary horizontal gaze use the pontine visual center for lateral gaze. This center in the pons has several names associated with it, including the paramedian pontine reticular formation (PPRF) and the para-abducens nucleus. Discharges from the horizontal gaze center in the pons permit simultaneous stimulation of the ipsilateral sixth nerve and contralateral third nerve. As a result, conjugate horizontal gaze moves the eyes toward the side of the discharging gaze center. Thus, horizontal gaze to the patient's right is using the discharging gaze center of the right pons. Dysconjugate gaze, as a result of injury to gaze structures, may cause the patient to complain of double vision or diplopia.

Vertical gaze depends upon coordinated contractions of eye muscles innervated by nerves III and IV. These nuclei are innervated by an anatomically different control locus, as the vertical gaze center lies in the roof of the midbrain (the tectum) and not the pons. Paresis of ocular movement may cause functional impairment by interfering with visuomotor tasks. The inability to move the eye upward, inward, or downward, with preserved lateral movement, suggests injury to nerve III. This often is accompanied by an enlarged pupil and a droopy eyelid on the side of the injury. Injury to nerve IV may manifest as the inability to turn the eye inward or move it downward and is often accompanied by head tilt.<sup>61</sup> The inability to move the eye laterally, with other ocular movements preserved, is consistent with an injury to nerve VI.<sup>62</sup>



## **Cranial Nerve V**

A lesion in cranial nerve V occurs in about 3.6% of head-injured patients.<sup>63</sup> The injury is most commonly due to a facial fracture and involves any or all branches of the trigeminal nerve. Rarely, the trigeminal nerve may be injured as a result of brain stem trauma or following a basilar skull fracture into the petrous bone.<sup>64</sup> If the sensory branches of nerve V are injured, hemianesthesia in the face will be found in one of the three branches. Injury to the ophthalmic branch causes corneal anesthesia, and injury to the maxillary or mandibular branches will produce anesthesia in the mid-lower face or the lower face. Injury to the motor branch of the trigeminal nerve produces a weakness in the masseter, temporalis, and pterygoid muscles.

Assessment of cranial nerve V is fairly simple. The corneal reflex will test the sensory ophthalmic branch of the trigeminal nerve, and facial sensation over the lateral maxillary and mandibular areas can be tested with a cotton swab. Motor function can be tested by vigorous clenching of the jaw by the patient to measure masseter and temporalis power. The pterygoid muscle strength can be assessed by asking the patient to move her jaw laterally. With a trigeminal nerve injury, the jaw will deviate toward the paralyzed side.

## **Cranial Nerve VII**

The facial nerve is injured in approximately 3% of head-injured patients. This usually results from a fracture of the temporal bone.<sup>10</sup> Facial nerve injury results in weakness of the muscles of the upper and lower face on the side of the injury. If the corticobulbar pathway is affected as a result of frontal lobe injury, injury to the internal capsule, or injury to the upper brain stem, a facial weakness will be present on the same side as the lesion, but the upper facial muscles will be spared. Facial nerve function is assessed by asking the patient to grin, purse her lips, raise her eyebrows or forehead, and tightly close the eyes.

The sensory portion of nerve VII carries taste sensation from the anterior two-thirds of the tongue. This pathway may be tested if needed by applying a dilute salt or sugar solution to the anterior portion of each side of the tongue. The patient should be instructed to remain with the tongue protruded so that the solutions do not mix from side to side. The mouth should be rinsed with water between applications of solution. If the sensory portion of nerve VII is intact, the patient will normally be able to identify these fundamental tastes. Aromatic substances with tastes that depend in part upon olfaction should not be used. Thus, taste should not be tested with aromatic oils or herbs. Moreover, it should be remembered that facial nerve damage resulting in paresis of the ipsilateral upper and lower facial muscles may or may not be accompanied by a loss of taste sensation.

## **Cranial Nerve VIII**

Hearing loss frequently accompanies traumatic head injury, and the incidence ranges from 18 to 56% of head trauma patients.<sup>65,66</sup> The cause of injury usually follows harm to the inner ear structures. The most common injury is a longitudinal fracture of the temporal bone caused by a lateral blow to the head, which results in a conductive hearing loss due to dislocation and disruption of the ossicles.<sup>66</sup> The two special sensory functions of nerve VIII (acoustic nerve) are auditory (cochlear division) and labyrinthine (vestibular division). The cochlear nerve transmits auditory impulses from the middle and inner ear mechanisms to the superior temporal gyri of both cerebral hemispheres (Brodmann's areas 41 and 42). As hearing is represented bilaterally on the cortex, unilateral lesions of the brain stem or cerebral hemispheres will not cause hearing impairment. However, transverse fractures of the temporal bone caused by occipital or frontal blows deforming the temporal bone cortex and splitting it cause sensorineural hearing loss, vertigo, and disequilibrium due to direct injury to either the acoustic branch of nerve VIII or the cochlea or labyrinth structures in the inner ear.<sup>10</sup> In those patients who sustain a brain stem contusion, the auditory vestibular nuclei may be impaired. Generally, the examiner will not be able to detect significant

functional impairment, as the deficit generally occurs unilaterally and the bilateral representation of hearing to some degree protects the patient's auditory ability in these instances. The more common outcome of head injury is impairment of vestibular function. This leads to dizziness or impairment of balance and coordination.

During examination of nerve VIII, hearing is tested initially by whispering into each of the patient's ears while covering the other or rubbing one's index finger and thumb together while covering the nontested ear. Air conduction vs. bone conduction is assessed by the Rinné and Weber tests. In the Rinné test, the vibrating tuning fork is held first against the mastoid process. When the sound is no longer heard by bone conduction, air conduction is tested by holding the tines outside the auditory canal. In sensorineural hearing loss, air conduction usually outlasts bone conduction. In conductive deafness, bone conduction is superior. A vibrating tuning fork is placed at the center of the forehead during the Weber test, and the patient reports whether the sound appears to originate from the right, left, or center of the head. If the sound lateralizes to either side during the Weber test, this is abnormal and indicates that bone conduction rather than air conduction is transmitting the sound. Sound lateralizes away from the side of sensorineural hearing loss because the acoustic nerve cannot detect the impulses and the sound lateralizes to the good ear. If the patient has conductive hearing loss, the auditory apparatus responds to bone conduction with less competition from external sound, and the patient will report hearing the sound better toward the side of the conductive hearing loss.

The presence of direction-fixed horizontal nystagmus usually suggests a unilateral injury to the vestibular apparatus. As noted previously, vertical nystagmus usually results from brain stem injury. However, nystagmus may also occur as a consequence of sedative-hypnotic medications, anticonvulsant medications, alcohol, and other specific medications. If vestibular injury is suspected, it is best to seek consultation from an otolaryngologist or otoneurologist.

## **Cranial Nerves IX and X**

These nerves are generally tested together, as clinical separation is difficult, if not impossible. The glossopharyngeal (nerve IX) and vagus (nerve X) are only rarely affected in traumatic head injury. The most common cause of injury to these structures is as a result of basilar skull fracture, which extends into the foramen magnum.<sup>63</sup> Nerve IX carries laryngeal and pharyngeal sensory function, and nerve X transmits primarily motor function of the same structures. An injury to these nerves generally results in an impaired ability to phonate, such as the letter *E*, and impaired swallowing. Nerve IX carries taste for the posterior one-third of the tongue and receives other sensation from the oropharynx. Nerve X supplies the motor systems necessary to produce the gag reflex once a sensation is carried through nerve IX. Thus, the gag reflex is composed of a reflex arc between cranial nerves IX and X.

To assess function of these two nerves, the examiner should listen to spontaneous speech during casual conversation. The patient may be asked to repeat syllables that require lingual (la), labial (pa), and guttural (ga) speech control. If a patient has cerebellar dysfunction instead of injury to nerves IX and X, she generally will demonstrate irregularities in the rhythm of speech similar to those in dystaxia, but her ability to form syllables should largely be intact. Moreover, injury to nerves IX and X should not be confused with dysphasic patients, as the patient with brain stem injury to these cranial nerves will be able to provide full meaning when speaking and verbal comprehension will be normal. Furthermore, a patient with injury to cranial nerves IX and X can write without language errors. For instance, the aphasic patient, when directed "Please raise your right hand and touch your right ear," would be unable either to comprehend or to comply with the request. A patient with injury to nerves IX and X would completely understand the command and be able to execute it.

In testing the gag reflex, with injury to nerves IX and X, the reflex is diminished or absent on the side of the nerve injury. Moreover, the palate and uvula may be deviated to the opposite side.

If there is an upper motor neuron lesion above the brain stem nuclei of cranial nerves IX and X, the gag reflex may be pathologically brisk and the patient may retch or even vomit. This is sometimes seen as a consequence of extensive injury to the frontal lobes or deep white matter structures. In this case, usually there is an associated pseudobulbar palsy consisting of dysarthria, dysphasia, and emotional lability.

### **Cranial Nerve XI**

The spinal accessory nerve (nerve XI) supplies motor function to the ipsilateral sternocleidomastoid and trapezius muscles. Only rarely is this nerve injured in traumatic brain injury, and that usually is in association with a basilar skull fracture. On examination, the spinal accessory nerve function is assessed by testing neck rotation. One must remember the rule of opposites here. When the right sternocleidomastoid muscle (R. SCM) is activated, the head turns to the left, and of course, the opposite holds for activating the left sternocleidomastoid muscle (L. SCM). Thus, if you ask the patient to rotate his head to the right and push upon your fist, you are testing the left sternocleidomastoid muscle. On the other hand, weakness of the trapezius muscle will be demonstrated if the patient has difficulty shrugging his shoulder on the ipsilateral side of the lesion.

### **Cranial Nerve XII**

The hypoglossal nerve (nerve XII) carries motor fibers to the muscles of the tongue. It is the only somatic motor nucleus located primarily in the medulla. It rarely is affected in patients sustaining traumatic injury to the head, and when injury occurs, it usually is the result of a basilar skull fracture or injury to the alanto-occipital region.<sup>67</sup> Nerve XII is tested by having the patient stick out his tongue. If the hypoglossal nerve has been injured, the tongue will deviate to the same side as the nerve injury. Fractures of the occipital condyle or bullet wounds in this anatomical area may cause a Collet–Sicard syndrome following injuries to nerves IX through XII.<sup>100</sup> [Table 4.14](#) offers a quick guide to traumatically induced cranial nerve injuries.

## **MOTOR EXAMINATION**

In the neuropsychiatric examination of the brain-injured patient, muscular atrophy usually occurs as a result of an immobilization syndrome following prolonged coma or inability to move. Focal muscle atrophy is invariably associated with lower motor neuron (LMN) injury and should alert the examiner to possible peripheral nerve injury or radiculopathy (nerve root injury). It is not expected that the neuropsychiatric physical examination will be as thorough as that provided by a neurologist. However, general observation will reveal the muscle bulk of the patient. Focal atrophy can be discerned by comparing the circumference of the limb in question, and measurements around the biceps, quadriceps, or gastrocnemius may be useful for side-to-side comparisons. Traumatic brain injury often is associated with severe trauma to the body, and the median, ulnar, radial, and sciatic nerve group may be injured as a result of skeletal injury or focal trauma. Brachial plexus or cervical radiculopathies are not uncommon if the patient is thrown around in an accident sufficient to stretch cervical, thoracic, or lumbar nerve roots.

### **Muscle Tone**

Spasticity is the most common abnormality of muscle tone seen in traumatically brain-injured patients. With spasticity, when the examiner passively moves an extremity through its range of motion, a velocity-dependent increase in resistance may be noted. Spasticity predominantly affects the flexor groups in the upper extremities and the extensor groups in the lower extremities. Associated neurological signs are seen with spasticity. These usually include muscle weakness, hyperreflexia, and a positive Babinski sign in the upward (extensor) direction. Spasticity is noted

**TABLE 4.14**  
**Traumatic Cranial Nerve Injuries**

Nerve	Usual Cause of Trauma	Clinical Testing
I	Frontal blows, fracture of the cribriform plate, contusion of the entorhinal cortex	Nonirritating stimuli such as anise or peppermint oils
II	Fractures of orbital bones, shearing forces, mechanical stretching, contusions, or vascular injury	Pupillary light response, fundoscopic examination, visual field testing, and measurement of visual acuity
III, IV, and VI	Fractures of the orbital walls, basilar skull fracture extending into cavernous sinus	Eye tracking right, left (nerve VI); up, down (nerve III); in and down (nerve IV); in and up (nerve III); raise eyelid, pupillary light response (nerve III)
V	Facial fractures; rarely brain stem injury or petrous bone fracture	Corneal reflex; sensation of lateral face, gums, inner cheek (sensory limb); masseter, pterygoid, temporalis strength testing (motor limb)
VII	Temporal bone fractures, brain stem trauma to nerve nucleus (lower motor neuron); injury to frontal lobe or internal capsule (upper motor neuron)	Squeeze eyes closed, raise eyebrows, purse lips, grin (UMN lesion spares forehead raising); sensory arm tested with sweet or salt solution to anterior tongue
VIII	Longitudinal fracture of temporal bone, transverse fracture of temporal bone, petrous bone concussion	Hearing check, Weber and Rin� tests, check for horizontal nystagmus, ice-water caloric test
IX and X	Basilar skull fracture extending into foramen magnum	Test gag reflex; repeat <i>la</i> , <i>pa</i> , and <i>ga</i> ; examine uvular and palatal movement
XI	Basilar skull fracture	Turn head to right (L. SCM) and left (R. SCM) against force; raise shoulder toward ears (trapezius)
XII	Basilar skull fracture or alanto-occipital injury	Protrude tongue; deviation is to the side of the injury; atrophy to side of injury

*Note:* UMN = upper motor neuron; L. SCM = left sternocleidomastoid muscle; R. SCM = right sternocleidomastoid muscle.

by increasing the velocity of the movements of the extremity. Rigidity, on the other hand, is also a resistance to passive muscular movement, but it has no relationship to velocity. It is found most prominently in the flexor muscle groups of the upper and lower extremities when it is present. Cogwheel rigidity is a ratchet-like resistance noted during passive movement of the extremities. It is commonly not present following traumatic brain injury unless there has been direct injury to the basal ganglia. Neurologically, this is usually seen as a consequence of cerebral anoxia or, of course, as a side effect of neuroleptic medications. Bilateral frontal lobe injury often results in paratonia. Bilateral frontal lobe contusion is not unusual in traumatically induced brain injury, and during passive movement of the extremities, the patient may be unable to voluntarily relax her muscles when asked to do so. Hypotonia, diminished muscle tone, is generally not a consequence of traumatic cerebral injury. However, it is seen following hypoxic birth injury or traumatic damage or injury to the cerebellum.

Spasticity has significant negative implications for the rehabilitation of traumatically brain-injured patients. Spasticity in the affected limb may impede mobility and transferability. Upper-extremity spasticity may affect the patient's ability to perform daily care activities. Spasticity of the neck or head can lead to difficulties with feeding, and spasticity of the pharyngeal and laryngeal muscles may interfere with oral communication, swallowing, and even breathing. If tone is increased in the trunk muscles, the patient may experience problems positioning herself in bed, in a wheelchair, or during standing and attempts at ambulation. Spasticity associated with paresis may result in joint contractures of the affected extremity. These are most likely to be seen in the wrist, elbow, knee, or ankle. The examination of muscle tone occurs with the patient fully relaxed. Passive movements of the upper and lower extremities are elicited. Flexing and extending the wrist, elbow,

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**TABLE 4.15**  
**Signs of UMN vs. LMN Lesions**

UMN	LMN
Hyperreflexia	Hyporeflexia
Spasticity	Flaccidity
Babinski sign	Atrophy
Clonus	Fasciculations
Weakness	Weakness

*Note:* UMN = upper motor neuron; LMN = lower motor neuron.

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shoulder, knee, or hip may elicit abnormalities of tone. If range of motion is limited, the examiner should consider contracture or a heterotopic overgrowth of bone in the affected joint region leading to ossification.<sup>68</sup>

### **Muscle Strength**

The two most common patterns of muscle weakness following traumatic brain injury are hemiparesis and quadriparesis due to injury to the corticospinal tracts coursing through the cerebral hemispheres or within the brain stem. With an upper motor neuron (UMN) lesion of this type, weakness is usually accompanied by spasticity and hyperreflexia. If the muscle weakness is focal, the examiner should be suspicious of a superimposed nerve root injury (radiculopathy) or peripheral nerve injury.<sup>54</sup>

Muscle weakness not only causes obvious functional limitations, but also may lead to significant neuropsychiatric morbidity. Depression is not uncommon, depending on the distribution and severity of the weakness. Patients with severe quadriparesis often are unable to roll themselves in bed without assistance and cannot perform even simple daily care activities. A patient with hemiparesis usually has less physical restraint than the quadriparesis patient; however, he often has difficulty with ambulation, transfers, and daily care activity. [Table 4.15](#) distinguishes the five signs of upper motor neuron lesions from the five signs of LMN lesions commonly encountered in the clinical examination of muscle function. It should be noted that of the five notable signs, only weakness is seen in both UMN and LMN lesions.

### **ABNORMAL INVOLUNTARY MOVEMENTS**

Either traumatic brain injury or dopamine-active medications commonly cause abnormal involuntary movements (AIMS) following trauma. Traumatic brain injury, particularly injury to the basal ganglia, can produce dystonia, dyskinesia, choreoathetosis, ballismus, myoclonus, asterixis, or Parkinsonism.<sup>69-80</sup> Dystonia is an involuntary sustained contraction of both agonist and antagonist muscles. It may cause repetitive, twisting movements or abnormal postures.<sup>69,70</sup> The psychiatrist or neurologist will be familiar with this disorder, as it frequently is caused by high-potency neuroleptic medicines such as haloperidol or fluphenazine. Dystonia generally has two causes following brain trauma: injury to the basal ganglia or as a side effect of neuroleptic medications. Dyskinesias are stereotyped, automatic movements of the limbs or oral-facial muscles, and they may also result from injury to the basal ganglia or from neuroleptic medication side effects. Choreoathetosis (*choreo* = dance, *athetosis* = wormy or writhing) is a slow spasmodic involuntary writhing or dancing movement of the limbs or face muscles. It is commonly seen as a side effect of neuroleptic medications, adrenergic medications, or anticonvulsants. It also is reported as an outcome from traumatic injury to the basal ganglia.<sup>71</sup> Ballismus is a violent flinging of the upper extremity, usually at the proximal shoulder, and generally is an outcome of injury to the subthalamic nucleus.<sup>10</sup> Tremor

is a frequent outcome as a medication side effect, but it has also been reported as a consequence of head injury. In the traumatically brain-injured patient, it is most frequently seen as a postural tremor and it may involve the head, upper extremities, or legs.<sup>72</sup>

Myoclonus is a shock-like or brief contraction of voluntary muscles. It can occur throughout the whole body, but it is found generally in a group of muscles. It is sometimes induced by an auditory stimulus, such as a loud noise or clap of the hands. It has been reported as an outcome of traumatic brain injury, and it usually is associated with cerebellar, basal ganglia, or pyramidal signs.<sup>73</sup> It is a common side effect from dopaminergic medications used in the treatment of Parkinsonism, and it often is an outcome of hypoxic brain injury. Asterixis is an involuntary lapse of posture or a flapping of the hands. It is most likely to be detected as a wrist flap, and physicians are aware of this as an outcome of hepatic failure. However, it also has been reported as an outcome of thalamic, internal capsule, midbrain, or parietal cortex injury.<sup>74–77</sup> Posttraumatic Parkinsonism has been described as a result of traumatic brain injury,<sup>78</sup> and most readers will be familiar with the posttraumatic Parkinsonism present in a former world-famous heavyweight boxer.

Examination of the patient to detect AIMS is primarily visual. However, choreoathetotic movements of the hands and face often can be detected by activating movements. Having the patient walk down the hall may activate choreoathetotic movements in the fingers and wrists. With the patient sitting in front of the examiner, the patient can be asked to tap her hand rapidly on her thigh, and while the examiner observes the mouth parts or the contralateral hand, dyskinetic movements may become manifest.

## SENSORY EXAMINATION

In orienting oneself for this portion of the neurological examination, it should be remembered that the sensory dermatomes are mapped over the human body. Eight upper-body dermatomes and six lower-body dermatomes are noted in [Table 4.16](#). Remember that the C1 dermatome does not exist, and the first clinical dermatome is C2, found at the occiput. The T4 dermatome marks the nipple line, and the T10 dermatome marks the umbilicus. Sensory perception is often dysfunctional in patients following traumatic brain injury. The sensory deficits may be of little consequence to the patient and are generally masked or outweighed by impairment in motor and cognitive systems.

An injury to one of the thalami causes an impairment of all sensory modalities on the opposite side of the face and body, whereas in parietal lobe injuries, sensory loss affects localization of the site of sensory stimulation. However, pain and temperature sensation are preserved following parietal lobe injuries. In addition to inability to localize the sensory input following parietal lobe injuries, the examiner will also find an impairment of stereognosis (the ability to manipulate shapes with the hand and identify them by tactile sensations). Joint-position sense is also impaired by parietal lobe injuries, as is graphesthesia (the ability to recognize figures written on the skin while

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**TABLE 4.16**  
**Sensory Dermatomal Patterns**

Upper Body		Lower Body	
C1	Does not exist	L1	Groin
C2	Occipital area	L2	Lateral thigh
C4	Above collarbone	L3	Medial thigh
C6	Thumb	L4	Medial leg
C7	Middle fingers	L5	Lateral leg, big toe
C8	Little finger	S1	Little toe, sole of foot
T4	Nipple line		
T10	Umbilicus		

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blindfolded or with eyes closed). In nondominant or right hemisphere injuries, sensory neglect is often apparent in the left hemisphere.

Assessment of the primary sensory modalities is easily accomplished by face-to-face neurological examination. Examination should include determination of sensation to pain, light touch, vibration, and joint-position sense. Once it has been established that the primary sensory modalities are intact, one can then check higher cortical sensory functions subserved by the parietal lobe. These include graphesthesia, stereognosis, and locating a sensory stimulus. Patients who have a neglect syndrome will be easily identified at this point, as they will be able to detect a stimulus such as a pinprick on either limb when the limb is tested individually, but they will neglect the affected side when the limbs are touched simultaneously. The examiner should ask that the patient's eyes be closed during this portion of the examination to disallow visual cues.

## REFLEXES

As has been noted in [Table 4.15](#), examination of the reflexes is very important in order to determine whether there is an upper or lower motor neuron lesion and to find lateralizing signs. Hyperreflexia is a consequence of injury to the upper motor neurons, whereas injury to the lower motor neurons causes hyporeflexia. Spasticity covaries with the hyperreflexia, whereas flaccidity generally accompanies hyporeflexia. The tendon stretch reflex tests the sensory–motor arc at the spinal cord level of the specific reflex. For instance, the biceps reflex tests the integrity of the C5–C6 spinal cord level. A hyperreflexic biceps tendon, in association with spasticity, would point to an upper motor neuron lesion in the contralateral brain or brain stem. The upper motor neuron pathway crosses the midline primarily in the pyramidal decussation of the lower medulla, which is immediately above the foramen magnum in a normal person. The first synapse in the direct corticospinal pathway from brain to spinal cord is in the anterior horn of the spinal cord. [Table 4.17](#) delineates the muscles associated with spinal nerve roots and the reflex that will test a particular nerve root. During examination, the reflex is elicited with a brisk tap from a reflex hammer over the tendon. Neurologists generally grade the level of the reflex, but for purposes of neuropsychiatric screening, that probably is not necessary; the important analysis is whether the reflexes are symmetrical from right side to left side and whether there is evidence of hyperreflexia or hyporeflexia.

The reflexes clearly help localize the site of a traumatic brain injury. A hyperactive reflex is consistent with an injury to the corticospinal tract, and one should find associated muscle weakness ([Table 4.15](#)) and possibly an upgoing large toe upon stroking the sole of the foot (Babinski sign). Hypoactive reflexes are seen often with injury of the lower motor neuron. Focal hyporeflexia in one nerve root system should alert the examiner to injury in a spinal root, plexus, or peripheral nerve. Diffuse hyporeflexia is seen following cerebellar injury, but it also is common in the

**TABLE 4.17**  
**Muscle Stretch Reflexes**

Roots	Muscles	Actions	Reflexes
Nerve V	Masseter	Clench jaw	Jaw reflex
C5	Deltoid	Abduct shoulder	—
C5–C6	Biceps	Flex elbow	Biceps reflex
C5–C6	Brachioradialis	Flex elbow	Brachioradialis reflex
C7	Triceps	Extend elbow	Triceps reflex
C8	Intrinsic hand	Abduct/adduct fingers	—
L3–L4	Quadriceps	Extend lower leg	Patellar reflex
L4–L5	Anterior tibial	Dorsiflex foot and big toe	—
S1	Gastrocnemius	Plantarflex foot	Achilles reflex

peripheral neuropathy often associated with hypothyroidism, diabetes mellitus, alcoholism, or renal disease. As noted previously, a hyperactive jaw jerk suggests bilateral corticospinal tract injury above the level of the middle pons.

## **COORDINATION: CEREBELLAR**

Coordination is controlled by various brain and peripheral nervous system structures. These include the corticospinal tracts, the basal ganglia, the cerebellum, and the sensory pathways. The most important area of the brain that contributes to coordination is the cerebellum. Before one can attribute incoordination to cerebellar dysfunction, it must be determined that the other four systems contributing to coordination are intact. Therefore, vision must be intact to coordinate movement; the motor system must be intact enough to provide strength sufficient to perform a task; proprioceptive sensation must be intact for the person to detect the attitude of his limbs in space; and the vestibular system must be intact so that the patient can integrate rotational movement and position in space.<sup>79</sup>

Cerebellar injury may result in either limb or truncal ataxia. The patient may be unable to gauge distance (dysmetria), and inability to do so will result in the patient overshooting or undershooting an intended target with his hand or foot. Cerebellar injury also may result in impairment of rapid alternating movements (dysdiadochokinesia) or in a reduction in speed and skill while performing complex movements (dyssynergia). Lastly, cerebellar injury can cause intention tremor.<sup>80</sup> The vermis is the most important part of the cerebellum for control of leg coordination. On the other hand, the cerebellar hemisphere is the most important structure for arm and hand coordination. The three major signs that suggest cerebellar incoordination are dysmetria, intention tremor, and dystaxia. Sensory pathway lesions involving the posterior columns will cause dystaxia due to an impairment of proprioceptive sensation, but they will not result in dysmetria of the toe when pointing to an object.

Examination of upper-extremity coordination is fairly simple. The examiner should ask the patient to alternately touch her nose and then the examiner's finger, which is placed at an arm's length from the patient. Intention tremor can be detected as a fine rhythmic, regular movement of the outstretched finger that intensifies as the patient attempts to touch the examiner's finger or hand. This is different from dysmetria, which is "past-pointing," a jerky irregular movement and overshooting of the patient's arm or finger when she tries to touch the examiner's hand or finger target. Should dysmetria or intention tremor be present, the patient can be asked to produce handwriting. Intention tremor will affect the smoothness and accuracy of the handwriting movements, whereas dysmetria may result in the patient being unable to maintain handwriting upon a straight line. To test for dysdiadochokinesia, the patient should be sitting comfortably in front of the examiner. The examiner should then ask the patient to place her right hand on her knee. Alternatively, the patient can place the palm of her hand on a table. The examiner should then demonstrate how to rapidly turn his hand palm up and then palm down and ask the patient to repeat the maneuver, first with the right hand and then with the left. Another simple measure of dysdiadochokinesia is to ask the patient to alternately touch fingers 2 through 5 with the thumb in rapid succession. The speed, rhythm, and smoothness of the movement should be assessed, as well as the accuracy of point-to-point contact.

Lower-extremity coordination may be assessed with the patient sitting on the examination table in front of the clinician or in a chair facing the examiner. The patient is asked to touch his heel to his opposite knee and then slide his heel up and down his lower leg. Smoothness and accuracy are again assessed. If this is not practical for the patient, for instance, due to hip dysfunction, the patient can be asked to draw a figure eight or circle in the air with his large toe. Dysdiadochokinesia of the foot can be assessed by asking the patient to rapidly tap his foot on the floor. Dystaxia is best tested by observing the patient while walking. One can ask the patient to perform "the drunk test" by placing the feet heel to toe. However, the examiner should exercise caution in asking significantly weak patients or elderly patients to perform this maneuver, as they may fall.



**TABLE 4.18**  
**Examination of Coordination**

Defect	Maneuver
Dysmetria	Finger-to-nose test, toe-to-finger test
Intention tremor	Same as above, handwriting analysis
Dyssynergia	Thumb-to-fingers in rapid succession
Dysdiadochokinesia	Rapid supination–pronation of hand, rapid tapping of toe upon floor
Dystaxia	Heel-to-toe walking, observe gait and turns
Romberg sign	Stand with heels together, arms stretched forward, close eyes, maintain posture

Persons who have a true sensory loss in both feet due to neuropathy or other cause will be unable to maintain their posture during the Romberg maneuver. Also, this will be found in patients who have injury to the posterior columns of their spinal cord from trauma, multiple sclerosis, syphilis, or vitamin B-12 deficiency. The Romberg sign is easily elicited by having the patient stand in front of the examiner, stretch her arms at 90° forward from her body, and close her eyes; the clinician then asks the patient to maintain her balance. Be prepared to catch her if necessary. When the patient closes her eyes, the ability to visually compensate for body position in space is lost, and if the posterior columns cannot transmit sensory information from the feet or if the patient cannot feel the floor with her feet, she may fall. [Table 4.18](#) describes the simple maneuvers for evaluating coordination.

## POSTURE AND GAIT

Many of the relevant examination techniques have been covered previously. However, since traumatic brain injury frequently impairs the motor and sensory systems, posture, balance, and gait are often impaired. Observation of gait is simple: merely ask the patient to walk, if he can. A patient with spastic hemiparesis may have difficulty standing because of trunk instability or may have a limp on the affected side due to weakness in the leg. Weak hip flexors and ankle dorsiflexors will cause an impaired swing-through of the leg, and the toe will inadequately clear during the swing phase of the gait. To compensate, the patient may swing the affected leg away from the body in a circumduction arc. The patient with a hemiparetic arm may hold it in a flexed posture as he walks. Parkinsonian dysfunction will reduce arm swing and stride length. The Parkinsonian patient also may have a shuffling of the feet associated with stooped posture. These findings are pathognomonic of basal ganglia dysfunction. If the patient has proprioceptive deficits in the foot, he may have difficulty placing the foot and maintaining balance.

The patient should be observed as she stands or sits. This will give an index of hip strength and static balance. By asking the patient to stand with her feet together and arms outstretched, one can further assess static balance. While walking, the gait should be assessed to determine if the patient's head and trunk are in the proper position and whether the arm swing is normal and symmetrical. Assessment of posture and gait should be coupled with the examination of coordination.

## THE CHILD MENTAL EXAMINATION

The mental status examination may be conducted at the beginning or end of the pediatric neuropsychiatric examination. Oftentimes, the child's neurological examination provides helpful data on the mental status, for example: Can the child pay attention to the examiner? Does the child follow the examiner's simple directions? Is the child impulsive? Does the examiner have to repeat questions? How does the child respond socially?<sup>81</sup> Obviously, in assessing the child, if the child is a competent historian, the chief informant is the child. In examination of the minor child, while the

child's information is very important, the parent or guardian generally represents the child. The child's psychiatric interview is thus more complex than that of the adult. The examiner must take into account the child's age, level of cognitive development, and willingness to discuss problems.<sup>82</sup> While some experts may examine children younger than 3 years to determine cognitive capacity, cognitive examinations of children younger than 3 are difficult, if not impossible, to complete with objective data. Since standardized neuropsychological test instruments exist for children 3 years old and above, it is probably best to wait until the brain-injured child is age 3 to assess cognitive deficits objectively.

Children ages 3 to 6 years can usually provide the examiner with correct information if the questions are framed in a manner consistent with their levels of development.<sup>81</sup> However, as we will see later in this text, the examiner must use care and not make assumptions about the validity of a child's report in situations where child abuse issues or litigation may be involved. Younger children are suggestible, and they may merely repeat information given to them by a hostile or litigious parent.<sup>83,84</sup> The neuropsychiatric mental examination of a very young child is essentially a neurodevelopmental examination. The Folstein et al. Mini-Mental State Examination has been adapted for use with children by Ouvier et al.,<sup>85</sup> and Weinberg et al.<sup>86</sup> have developed the *Symbol Language Battery* for use in the physician's office to screen child cognition.

## ATTENTION

Attention can be evaluated in the young child by observing the youngster's ability to attend to the examiner or to listen to the topic of discussion. The degree to which the child jumps from one activity to another or needs restructuring and physical limitations is an important marker of poor attention. If the child is easily distracted by noises outside the examination area or quickly drawn to objects in the room and is unable to resist grabbing the objects, then it is fairly obvious that the child's attention is impaired. For a child greater than age 8 years, attention can be assessed by having the youngster count from 1 to 20. If vigilance is assessed, generally children over age 9 can perform serial 7s or spell *world* backward.

## SPEECH AND LANGUAGE

The evaluation of speech and language, of course, depends upon the age of the child and the development of language appropriate to the child's age. As noted earlier with the adult mental examination, the examination of a child's language is no different. The examiner must listen to the articulation, inflection, and rhythm and fluency of the child's speech. Analysis of language is based on whether the child speaks with idiosyncratic aspects and if the vocabulary and syntax are correct. With a young child, it is important to note whether there is misuse of pronouns and gender. A judgment can be made about the overall intelligence of the child based on how the language is produced and whether it is appropriate to the child's age. Can the child tell a small story or a joke (narrative discourse)? The nonverbal aspects of language are evaluated in the child in the same way as they are in the adult. Does the child have appropriate facial expression, speech melody, and intonation and make eye contact with the examiner? If the child appears to have a formal language disorder, it may be necessary to consult with a speech and language pathologist for more definitive evaluation. The important speech and language milestones of the child below 7 years are noted in [Table 4.19](#).

## MEMORY AND ORIENTATION

Children ages 3 to 7 years are able to answer general orientation questions.<sup>87</sup> For instance, a child within this age group is able to give his first and last name and tell how old he is. He generally knows the month and day of his birthday and the city where he resides. He is able to relate his

**TABLE 4.19**  
**Important Childhood Speech and Language Milestones**

Receptive Language	Age	Expressive Language
Turns to sound of bell	6 months	Cries, laughs, babbles
Waves bye-bye	9 months	Imitates sounds and makes dental sounds during play (e.g., “da-da”)
Knows meaning of “no” and “don’t touch”	12 months	Uses 1 or 2 words (e.g., “da-da,” “mama,” “bye”)
Responds to “come here”	15 months	Uses jargon (speechlike babbling during play)
Points to nose, eyes, hair	18 months	Uses 8–10 words (one third are nouns) Puts 2 words together (e.g., “more cookie”) Repeats requests
Points to a few named objects	24 months	Asks 1- to 2-word questions (e.g., “Where kitty?”)
Obeys simple commands		
Repeats 2 numbers	30 months	Uses “I,” “you,” “me”
Can identify by name “What barks?” and “What blows?”		Names objects Uses 3-word simple sentences
Responds to prepositions <i>on</i> and <i>under</i>	3 years	Masters consonants <i>b</i> , <i>p</i> , and <i>m</i>
Responds to prepositions <i>in</i> , <i>out</i> , <i>behind</i> , and <i>in front of</i>	4 years	Speaks in 3- to 4-word sentences Uses future and past tenses Masters consonants <i>d</i> , <i>t</i> , <i>g</i> , and <i>k</i>
Can repeat a 7-word sentence	6.5 years	Masters <i>th</i> sound Uses 6- to 7-word sentences Says numbers up to 30s

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father’s and his mother’s names. If in school, he can generally name his school and grade level. Most children within this age group will be able to tell the examiner their present location, for instance, in a hospital or doctor’s office. They are also able to state whether it is daytime or nighttime.<sup>81,82</sup>

Children ages 8 to 15 years are oriented more specifically in time. Children in this age group will be able to tell the examiner the current time and day of the week. They know the day of the month and the name of the month. They also are able to give the year, unlike most children in the 3- to 7-year age group.<sup>81,82</sup> A child younger than 8 years can usually learn and repeat three simple objects if he is given sufficient rehearsals to learn all three. For instance, the child can be asked to remember *ball*, *cup*, and *doll*. Most normal children will remember two or three of these objects. Visual memory can be tested by hiding three objects as the child watches. Five minutes later, the child can be asked to retrieve the objects, and even the younger child should be able to do so unless visual memory is impaired. Remote memory can be evaluated by asking even a young child to relate her favorite television show or, if in preschool or school, her teacher’s name.

## **VISUOSPATIAL AND CONSTRUCTIONAL ABILITY**

Pencil-and-paper tests can be used to assess visuospatial abilities in young children. A 3-year-old child should be able to copy a circle drawn by the examiner in front of the child. A 4-year-old should be able to copy an X and a + symbol. A 5-year-old can copy a triangle, and a 6-year-old can copy a square. Children older than age 7 generally can copy intersecting diamonds.<sup>81</sup> Most children ages 9 or older should be able to draw a clock, place the numbers in the appropriate locations, and draw hands to four o’clock.

## EXECUTIVE FUNCTION

Deficits in executive function occur very frequently after childhood closed-head injuries and other traumatic brain injuries. However, studies of children with executive dysfunctions are particularly lacking since frontal lobe function continues to develop in humans until about age 25. It is thus understandable that there would be limited markers for executive function in the young child. When examining children who have sustained brain trauma, various tasks may assess executive function, but these are probably outside the scope of a face-to-face mental examination. These tests would include the *Tower of London*, which measures planning skills; the *Controlled Oral Word Association Test*, which measures verbal fluency; and the *Wisconsin Card Sorting Test*, which measures concept formation and mental flexibility.<sup>88</sup>

## AFFECT AND MOOD

Stability of moods normally is evident in children by age 2 or 3. Usually at this point there is a diminishment of crying and separation anxiety. Most children by preschool age have learned not to show anger or to be abusive to others. The child can be asked very simple questions, as outlined by Weinberg and others.<sup>99</sup> Simple questions include:

- Can I ask you some very personal important questions?
- Are you having mostly good, mixed, or bad days in your feelings?
- Is it so bad you sneak off to your room and cry?
- Are you able to have fun when you feel badly?
- Have you been thinking about dying or wish that you were dead?
- Would you like to leave and go to heaven?

Also, as noted, the child may be irritable or assaultive. It may have been noted that he spoke of death. In young children in particular, depression and sadness may manifest as gastrointestinal complaints.

## THOUGHT PROCESSING, CONTENT, AND PERCEPTION

Determining thought content in the child is difficult at best. The problem of assessing thought disorder in children has been addressed by others.<sup>103,104</sup> Children can develop delusions and even hallucinations. Detecting this in very young children is difficult, but Caplan et al.<sup>105</sup> has developed an instrument that reliably and validly measures illogical thinking and loose associations in children. The development cutoff age is 7 years in nonschizophrenic children. The *Kiddie Formal Thought Disorder Rating Scale* (K-FTDS) is useful to assist the neuropsychiatric examiner if consideration is given that children between 3 and 7 years are demonstrating formal thought disorders. [Table 4.20](#) outlines a face-to-face neuropsychiatric screening method for the child who has sustained brain trauma.

## THE CHILD NEUROLOGICAL EXAMINATION

When examining the child who may have been traumatized and spent many days in a hospital, it is useful to keep a few simple pediatric pearls in mind. In younger children, the neurological examination will be a catch-as-catch-can procedure. A considerable amount of information can be obtained merely by observing the youngster play or interact with her parents. The dominant handedness of the child or the presence of cerebellar deficits, hemiparesis, or even visual field defects may become apparent with this approach. It is best not to wear a white coat, as children equate this with injections or immunizations. For the 3- to 7-year-old child, a few small items are useful for the examination, for example, a tennis ball, small toys, a small car or truck that can be

**TABLE 4.20**  
**Face-to-Face Neuropsychiatric Screening Methods for Brain-Traumatized Children**  
**Ages 8–15 Years**

Domain	Task
Orientation	“What is the year, the season, the date, the day, and the month?”
Attention	“Count from 20 to 1 backward.”
Vigilance	“Subtract 7 from 100; now subtract 7 from that answer; keep subtracting 7 from each answer.”
Memory	“Repeat after me: <i>ball, cup, doll</i> . Repeat them again. Now I want you to remember these. I will ask you to repeat them later.” “I’m going to hide these objects here in the room [3 common items]. Watch me and then I will see if you can find them.”
Language	Point to a pen, your watch, your nose. Ask the child to name each one. “Repeat: no ifs, ands, or buts.” “Take this paper, fold it in half, carry it across the room, and place it on the desk.” Show a paper or card with large print: “CLOSE YOUR EYES.”
Visuospatial	Have the child copy two intersecting diamonds. Ask the child to draw a clock, place numbers on the clock, and place hands at 4 o’clock.

used to assess fine motor coordination, a bell, and a bright or shiny object that will attract the child’s attention.

When examining a 3- or 4-year-old child, it is best to have the child seated in his mother’s or father’s lap and to talk to the child while facing him. Defer touching the child until some degree of rapport as been established both with the parent and the child. For a 3- or 4-year-old child, handing him a toy or a bright object may improve the development of rapport. Patience is required because most young children, once frightened, are difficult to reassure and the examination may not proceed well.

### APPEARANCE

The general appearance of the child is carefully noted, particularly her facial configuration and the presence of any dysmorphic features or structural alterations of the face. Cutaneous lesions are clues to the presence of phakomatoses. These include lesions such as *café au lait*, angiomas, facial pigmentations, etc. Some pediatric neurologists take particular note of the location of the hair whorl, as abnormalities of whorl patterns may indicate the presence of a cerebral malformation.<sup>89</sup> The neuropsychiatric examiner is clearly not expected to be an expert pediatrician or pediatric neurologist. If unusual facial features are found, a consultation may be required, as clearly what may appear to be cognitive changes from a traumatic head injury may in fact have a contributing factor or causation from a congenital or genetic disorder.

The general appearance of the skull can suggest the presence of macrocephaly, microcephaly, or craniosynostosis. Prominence of the venous pattern over the scalp might accompany increased intracranial pressure. Biparietal enlargement suggests the presence of subdural hematomas and, in certain situations, should raise the suspicion of child abuse. Palpation of the skull can disclose ridging of the sutures as occurs in craniosynostosis. The head circumference of the child should be measured and compared with a standard international and interracial head circumference graph.<sup>90</sup> One may review most standard textbooks of pediatrics for this information.

### CRANIAL NERVES

As with the adult, a child may lose olfactory nerve function due to infraorbital or temporal lobe brain trauma or a fracture through the cribriform plate. Olfactory sensation is not functional in a

newborn, but is present by at least 5 to 7 months of age. By the time an accurate brain injury examination of a child can be made at age 3, full olfactory function should be present. However, a newborn will respond to inhalation of irritants such as ammonia or vinegar, as this is transmitted by nerve V. Even a child born without an olfactory apparatus will respond to irritation of nerve V.<sup>91</sup>

The optic nerve in the child can be injured in the same manner as the adult's optic nerve. The macular light reflex is absent until approximately 4 months of age, but clearly by age 3, the child will have a physiological reflex. Visual acuity can be tested in the older child by standard means. In the 3- or 4-year-old, approximation of visual acuity can be obtained by observing him or her at play and by offering toys of various sizes into the visual field. In a very small child or a child who is severely injured, the blink reflex, closure of the eyelids when an object is suddenly moved toward the eyes, may be used to determine the presence of functional vision. This reflex is absent in the newborn and does not appear until approximately 3 to 4 months of age. It is present in about half of normal 5-month-olds, but certainly by age 1, all normal children should have a physiological blink reflex.<sup>92</sup>

Nerves III, IV, and VI are evaluated after first noting the position of the child's eyes at rest. Observation of the points of reflection of light from the illuminating instrument will assist the examiner in detecting nonparallel alignment of the eyes. Paralysis of nerve III results in a lateral and slightly downward deviation of the affected eye. If nerve VI is paralyzed, a medial deviation of the affected eye will be noted. Paralysis of nerve IV produces little eye position change at rest. Eye movements are examined by having the very young child visually follow a shiny object. The mother should hold the child's head to prevent rotation. If the young child will permit the examiner to do so, each eye should be examined separately while the other one is kept covered. Sometimes, the child is able to assist with this, and at other times, the parent may be asked to assist. There should be no difficulty detecting abnormalities in a young child, as eye excursion is completely developed in all directions by about 4 months of age. Eye movements directed toward a sound appear at about 5 months of age, and depth perception is present at 2 to 4 months of age.<sup>93</sup> In a palsy of nerve VI, failure of the affected eye to move laterally should be readily demonstrable. For a pure nerve III palsy, the defective eye will appear outwardly and downwardly displaced. Lateral movement will be defective. If nerve IV is palsied, the eye fails to move down and in. This defect is often accompanied by head tilt.

A simple test for the motor component of nerve V is performed by asking the child to demonstrate how to chew gum. If the child seems to fully comprehend this instruction, the examiner can chew appropriately in front of the child so that the child can attempt to mimic the examiner's movements. In a unilateral lesion of the trigeminal nerve, the jaw will deviate to the paralyzed side, and there should be atrophy of the temporalis muscle present some months after the injury. An upper motor neuron lesion above the level of the pons will result in an exaggerated jaw jerk. The sensory branch of the trigeminal nerve is tested by the corneal reflex and lateral facial sensation.

Injury to nerve VII should result in facial asymmetry. As noted previously, if the facial nucleus and branches distal to this site are injured, lower motor neuron weakness in which both upper and lower parts of the face are paralyzed will be present. Normal wrinkling of the forehead cannot be performed, the eyebrows cannot be elevated, and the affected eye cannot be closed. Weakness of the face will be obvious on observation, and the asymmetry should be accentuated when the child laughs or cries. Recall that facial weakness due to an upper motor neuron lesion above the facial nucleus or in the cerebral structures will spare the upper face musculature. The upper facial motor neurons receive little direct cortical input, whereas the lower facial neurons apparently do.<sup>94</sup> The sensory arm of the facial nerve can be tested with a weak salt or sweet solution, as described earlier with adult testing.

Hearing can be tested in the younger child<sup>90</sup> using a tuning fork or a bell. By age 3, all normal children will have the ability to turn the eyes to the direction of the sound, as this becomes evident by 7 to 8 weeks of age, and turning the eyes and head to stimuli appears at about 3 to 4 months of age. If there is a question of hearing loss in the child, audiometric evaluation may be required.

Vestibular function can be assessed by observing for nystagmus. It is not recommended during a neuropsychiatric examination that caloric testing of a young child be performed, and should this be required, consultation with an otolaryngologist is recommended.

Examination of nerves IX and X can be performed during the oral examination. The resting uvula and palate should function during phonation, and a failure to elevate indicates impaired nerve X function. The gag reflex tests both arms of the vagus–glossopharyngeal nerve arc. Measuring taste carried by nerve IX over the posterior part of the tongue is extremely difficult and is not recommended in children. Testing of nerve XI can be accomplished by having the child rotate her head against resistance from the examiner's fist or hand. Most children age 3 and older can mimic shoulder shrugging of the examiner. During examination of the mouth, the resting tongue can be observed for vesiculations. Nerve XII is easy to test in children, as they enjoy sticking their tongue out to mimic the examiner, and a paretic tongue will deviate toward the side of the lesion.

## **MOTOR**

The child's station can be observed at a distance. It is worthwhile to watch the child stand and then ask the youngster to run down the hallway. This enables assessment of running gait. Throwing a tennis ball down the hallway and asking the youngster to retrieve it is an excellent way to observe bilateral motor function, as most children enjoy performing for the examiner. This will provide sufficient information in the younger child to determine muscle strength, and other examinations of strength are merely confirmatory. In the child older than age 5, evaluation of the motor system can be done in a more formal manner. Muscle tone is examined by manipulating the major joints. It is necessary to rule out alterations of tone, particularly in children who may have had a perinatal birth injury and later sustained a traumatic brain injury.

A sensitive test for hypotonia of the upper extremities is to ask the child to raise his hands over his head. The pronator sign will appear in the hand on the hypotonic side as it hyperpronates to palm outward as the arms are raised. The elbow may flex as well. In the lower extremities, weakness of the flexors of the knee can be tested readily by having the child lie on her tummy and asking her to maintain her legs in flexion at right angles to the knee. The weak flexors will not allow her to maintain the leg at a 90° angle.

## **SENSORY**

Sensory examination is almost impossible to assess in a toddler. However, since adequate neuropsychiatric examination of a brain-injured child is difficult to perform before age 3, the examinations of children in this circumstance will focus on age 3 and above. Sensory modalities can be tested in a 3- or 4-year-old if the child is comforted on the parent's lap. Using a tracing wheel is the preferred modality. Pins appear too much like injection needles to a youngster. Likewise, most children can cooperate for vibratory testing if the child is told that it will tickle. Object discrimination can be determined in children older than age 5 by the use of paper clips, coins, or rubber bands.

## **COORDINATION: CEREBELLAR**

The younger child enjoys performing the finger–nose test if the child's attention span will permit it. Coordination can be tested by having the youngster reach for toys and manipulate them. The older child can perform not only finger–nose testing, but also heel–shin testing. The ability to perform rapidly alternating movements (diadochokinesia) can be tested by having the child repeatedly tap the clinician's hand or by having him perform rapid pronation and supination of the hand on the knee. Rapid tapping of the foot on the floor will evaluate diadochokinesia of the foot. The heel-to-shin test is more difficult for children to comprehend than the finger-to-nose test. Children 9 years of age and older generally can perform the heel-to-shin maneuver, but children ages 7 and below may have difficulty with this performance. Observation of the child is best to determine

abnormal involuntary movements, and the procedures used for the adult can be applied here. Athetoid and choreiform movements may activate during walking or by rapidly slapping one's thigh. Dystonic posturing is detected best by observation.

## REFLEXES

The younger the child, the less information that is obtained from deep tendon reflexes. With a child, reflex inequalities are common and less reliable than inequalities of muscle tone in terms of determining the presence of an upper motor neuron lesion.<sup>95</sup> The major deep tendon reflexes are noted in [Table 4.17](#). The Babinski sign is a significant indicator of impaired pyramidal tract function. Some young children cannot tolerate having the sole of their foot stroked, but stimulation of the outer side of the foot is less problematic for these youngsters. The Babinski response in the child is identical to that in the adult, and an extensor plantar response can be distinguished from voluntary withdrawal. Withdrawal is seen after a moment's delay, whereas the extension of the great toe and the fanning of the toes is immediate following stimulation. A Babinski sign is seen normally in the majority of 1-year-old children and in many children up to 2½ years of age. However, by age 3, almost all children will no longer demonstrate a Babinski sign.<sup>95</sup> Clonus is a regular repetitive movement of a joint caused by sudden stretching of the muscle. It is easiest to demonstrate by dorsiflexion of the foot. The examiner can press on the anterior sole of the foot and flex the ankle. Several beats of clonus can be demonstrated in very young children, but a sustained ankle clonus in a child older than age 3 is abnormal and suggests a lesion of the pyramidal tract. It is due to increased reflex excitability.<sup>90</sup> Young children often can perform tandem walking. This will be difficult for a 3- or 4-year-old child, but forward tandem gait is performed successfully in 90% of children 5 years of age or older. Hopping in place on one leg generally is difficult for a 3- or 4-year-old. However, by age 7, 90% of children will be able to hop in place on one leg.<sup>96</sup>

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# 5 The Use of Structural and Functional Imaging in the Neuropsychiatric Assessment of Traumatic Brain Injury

## INTRODUCTION

The management and evaluation of traumatic brain injury have been revolutionized with the advent of structural and, more recently, functional brain imaging. Computed tomography (CT) techniques were developed by the Nobel Prize-winning scientist Godfrey Hounsfield. The first CT imagers were developed by EMI Ltd. of Middlesex, England, and were introduced into clinical practice in 1972. These scanners came into the U.S. during approximately 1972–1973 and now have gone through five generations of development. CT is presently recognized as the first and most important step in evaluating for head and contiguous spine injuries following trauma.<sup>1</sup> In 1946, Bloch and Purcell discovered that when atomic nuclei are placed in a magnetic field, certain properties of structures or tissues can be measured. These scientists received a Nobel Prize for their discoveries in 1952, and their work led to the use of magnetic resonance imaging (MRI) in humans in about 1983.<sup>2,3</sup> MRI has been very much improved in its ability to be used for the evaluation of traumatic brain injury, and it is growing in adaptation in the acute care setting with the advent and refinement of fast imaging techniques and improvements in scanner hardware. The examination time for MRI is no longer a significant limitation in the evaluation of head trauma patients, as it is possible to obtain high-quality T1-weighted scans in 2 to 3 min using standard short repetition time/echo time (TR/TE) techniques.<sup>4</sup>

The evolution of single-photon emission computed tomography (SPECT) imaging of brain trauma patients developed out of methods of studying regional cerebral blood flow (rCBF). Using inhalation or intravenous injection of <sup>133</sup>Xe allowed a distinction between blood flow and gray and white matter to be determined.<sup>5</sup> Recent studies conclude that brain SPECT can be valuable in predicting the neuropsychological behavior of survivors of severe head injury,<sup>6</sup> and SPECT imaging is more sensitive than computed tomography in detecting posttraumatic brain lesions.<sup>7</sup> Positron emission tomography (PET) is rapidly emerging as state of the art for functional imaging of brain metabolism and blood flow. PET studies now are commonly performed following trauma, and a large body of knowledge is emerging regarding the relationship between PET metabolic studies and neuropsychological impairments after diffuse traumatic brain injury.<sup>8</sup> PET is more recently being used to study metabolic recovery following human traumatic brain injury.<sup>9</sup>

Functional magnetic resonance imaging (fMRI) refers to the demonstration of brain function with neuroanatomic localization on a real-time basis. The vast majority of such studies are performed using blood oxygen level-dependent (BOLD) contrast, which requires the detection of very small signal intensity changes. This signal response, detected by MRI, is a result of localized

hemodynamic changes induced by regionally increased neuronal activity associated with performing a defined cognitive task. Clinical fMRI studies are developing wherein functional maps can be generated for individual patients within a busy clinical schedule and reported in a timely fashion. Various paradigms exist for measuring a cognitive task performed by the patient during the fMRI study.<sup>10</sup>

Electroencephalography (EEG) appeared to lose its usefulness for detecting brain trauma following the advent of CT imaging and, later, the advent of MRI. Reports of EEG findings in brain trauma patients date back to the 1940s. More recently, the practical utility of electrophysiological testing with EEG is proving more useful.<sup>11</sup> Lately, the continuous use of EEG monitoring in the neurosurgical ICU is demonstrating that many subclinical seizures, including status epilepticus, are being missed, as often they are not evident clinically.<sup>12</sup> This chapter reviews all these modalities and the relationships they may play within the overall neuropsychiatric evaluation of traumatic brain injury.

## STRUCTURAL IMAGING OF BRAIN TRAUMA

It is important that the neuropsychiatric examiner or other physician develop a professional relationship with radiologists and neuroradiologists. These persons are needed to provide consultation and interpretation of CT and MRI to the neuropsychiatric examiner.

### COMPUTED TOMOGRAPHY

#### Use in the Acute Care Setting

CT remains the primary method for evaluating closed-head injuries. CT has various advantages: it is more widespread, low in cost, and safe, and it has rapid imaging time. CT has eventually replaced skull x-ray as the primary imaging tool in head injury because it provides imaging not only of the brain but also of other soft tissues, as well as the bony calvarium.<sup>13</sup> CT will not show every calvarial fracture, but it will show a sufficient number of depressed ones and reveal basilar skull fractures that planar x-rays do not demonstrate. CT is also the method of choice for demonstrating fractures of the facial bones, including the paranasal sinuses and orbits.

CT's main role in the screening of brain injury is to separate patients into three categories: (1) those with normal intracranial structures, (2) those with focal intraaxial or extraaxial hematomas, and (3) those with a more diffuse pattern of brain injury.<sup>14</sup> However, a cardinal rule to be followed when evaluating acute head injury is that normal findings by computed tomography do not exclude central nervous system injury.<sup>15</sup> In fact, in those patients demonstrating mild cognitive impairment who are triaged in the emergency department, greater age, a *Glasgow Coma Scale* (GCS) score of 14 or 15, and cranial soft tissue injury are risk factors for CT-detected intracranial hemorrhage.<sup>16</sup> It is advised that when an admission CT scan demonstrates evidence of diffuse brain injury, follow-up scans should be performed, because approximately one in six such patients will demonstrate significant CT evolution of injury over time.<sup>17</sup>

With regard to children, the clinical signs of brain injury are poor indicators of intracerebral injury in infants. A substantial fraction of infants with traumatic brain injury will be detected following CT imaging of otherwise asymptomatic infants who have significant scalp bruising. If children are older than 3 months of age, and they have no significant scalp hematoma, in general they may be safely managed without radiographic imaging.<sup>18</sup> Other CT findings in children may lead to the conclusions of probable child abuse. These include interhemispheric falx hemorrhage, subdural hemorrhage, large collections of extraaxial fluid, and edema of the basal ganglia. These findings are discovered significantly more frequently in inflicted pediatric head trauma than in noninflicted trauma.<sup>19</sup> Moreover, a normal neurologic examination and maintenance of consciousness does not preclude significant rates of intracranial injury in pediatric head trauma patients. Neither

loss of consciousness nor mild altered mental status is a sensitive indicator to guide the selection of pediatric patients for CT scanning, contrary to the usual conventions. A liberal policy of CT scanning is now warranted in emergency departments treating pediatric patients following head trauma.<sup>20</sup>

Recent findings within CT emergency room evaluations indicate that motor vehicle air bags are quite dangerous to very young children. As of November 1, 1997, automotive air bag deployments in low-speed collisions had resulted in the deaths of 49 children and the serious injuries of 19 children in the U.S. CT scans reveal that crush injury to the skull predominated in infant victims traveling in rear-facing child safety seats, whereas both cranial and cervical spine trauma occurred in older children traveling restrained, improperly restrained, or unrestrained in the front passenger seat of the vehicle.<sup>21</sup> Thus, the neuropsychiatric examiner, seeing a pediatric patient for evaluation who has been struck in the head by an air bag, should always consider closed-head traumatic brain injury as a possible mechanism of cognitive changes.

The indications for CT of the head after trauma are debated in the medical literature. However, a summary of the published findings notes indications for CT of the head in patients who sustained head trauma. These include:

1. Glasgow Coma Scale of less than 15
2. Clinical signs of basilar skull fracture or depressed skull fracture
3. All penetrating head injuries
4. Anisocoria or fixed and dilated pupils
5. Neurologic deficit, including focal motor paralysis
6. Cranial nerve deficit
7. Abnormal Babinski reflex
8. Known bleeding disorder or patient on anticoagulation medication
9. Loss of consciousness for more than 5 min
10. Anterograde amnesia<sup>13,22</sup>

The role of cranial CT scanning for adult patients with minor head injury is equivocal. The indications for pediatric patients were described in greater detail previously. However, at present, there is no algorithm that has yet been established that can predict all patients who will have a positive CT scan following head trauma. Patients with a GCS score of 15 have a low percentage (< 0.1%) of neurosurgical lesions.<sup>23</sup> Thus, the role of cranial CT scanning for patients with minor head injury remains controversial. [Table 5.1](#) categorizes the appearance of CT imaging of brain trauma.

## Skull Fracture

The incidence of skull fracture increases in relation to the severity of brain injury. However, a skull fracture provides evidence of bone injury from trauma, but it does not necessarily mean that the brain or spinal cord has been injured. MRI does not usually reveal fractures, because the protons of cortical bone are nonmobile during image acquisition. Thus, cortical bone appears as a linear hypointensity or blackness that cannot be discerned from air or cerebral spinal fluid (CSF). CT with bone window settings is now the method of choice for determining the presence of skull fracture, rather than standard planar cranial x-rays. However, when the neuropsychiatric examiner reviews medical records and observes shortly after the time of trauma prior evidence of a skull fracture, it must be remembered that bony injury is significant, not only as a sign of potential brain injury, but also as a pathway for the spread of infection. Moreover, skull fracture often has an associated cranial nerve palsy (see [Chapter 4](#)). If the records indicate that blood is present behind the tympanic membrane without direct ear trauma, or there is evidence of otorrhea or rhinorrhea or evidence of a subcutaneous hematoma around the mastoid process (battle sign), or when bruising around the orbits without direct orbital trauma (raccoon sign) is present, evidence of a basilar skull

**TABLE 5.1**  
**CT and Traumatic Brain Injury**

Lesion	Image Findings
Skull fracture	Calvarial disruption on bone window settings <sup>24</sup>
Contusions	Usually adjacent to anterior and middle cranial fossae, sphenoid wings, and petrous ridges — most frequent in frontal and temporal poles and undersurfaces of frontal lobes; hemorrhagic lesions, high-density; nonhemorrhagic lesions, low-density <sup>14,26,27</sup>
Epidural hematoma	Usually presents as a high-density, biconvex lens; does not cross suture margins; focal iso- or hypodensity consistent with active bleeding or coagulopathy <sup>1,30</sup>
Subdural hematoma	Acute: isodense against gray matter if hemoglobin less than 10–11 g/dl; if not isodense, presents as a crescent-shaped hyperdense collection that conforms to the gyral-sulcal pattern; does not cross the falx <sup>1,31,32</sup> Chronic: fluid usually appears hypodense due to blood product breakdown, but density higher than CSF due to protein content; upon complete breakdown of blood products, fluid may be isodense to brain <sup>1,33,34</sup>
Subarachnoid hemorrhage	Linear hyperdense fluid collection within sulci and fluid cisterns <sup>1,13</sup>
Intraparenchymal hemorrhage	Mostly found in frontal and temporal brain areas; usually hyperdense in appearance; serum from a clot may cause a rim of hypodensity; edema may produce a mass effect; in older lesions, new vessel formation may enhance as a rim with contrast agents; clot resorption may leave a cavity <sup>1,13,24,38,47</sup>
Intraventricular hemorrhage	Focal and diffuse hyperdensity within the ventricles; blood tends to settle in the occipital horns <sup>25,32</sup>
Diffuse axonal injury	Most injuries in lobar white matter at corticomedullary junction of frontal and temporal lobes; also appears at or in the corpus callosum and dorsolateral brain stem; usually appears as small hyperdense bleeds in these areas <sup>1,40–42</sup>
Brain swelling	Obliteration of cerebral sulci and basal cisterns; effacement of gray matter–white matter interface; edematous brain usually hypodense <sup>1,13,25</sup>
Chronic neurodegeneration	Irregular brain surface with hypodensity within parenchyma; overlying cerebrospinal fluid spaces may enlarge; cortical gyri size may diminish with increased ventricular size <sup>1,48</sup>

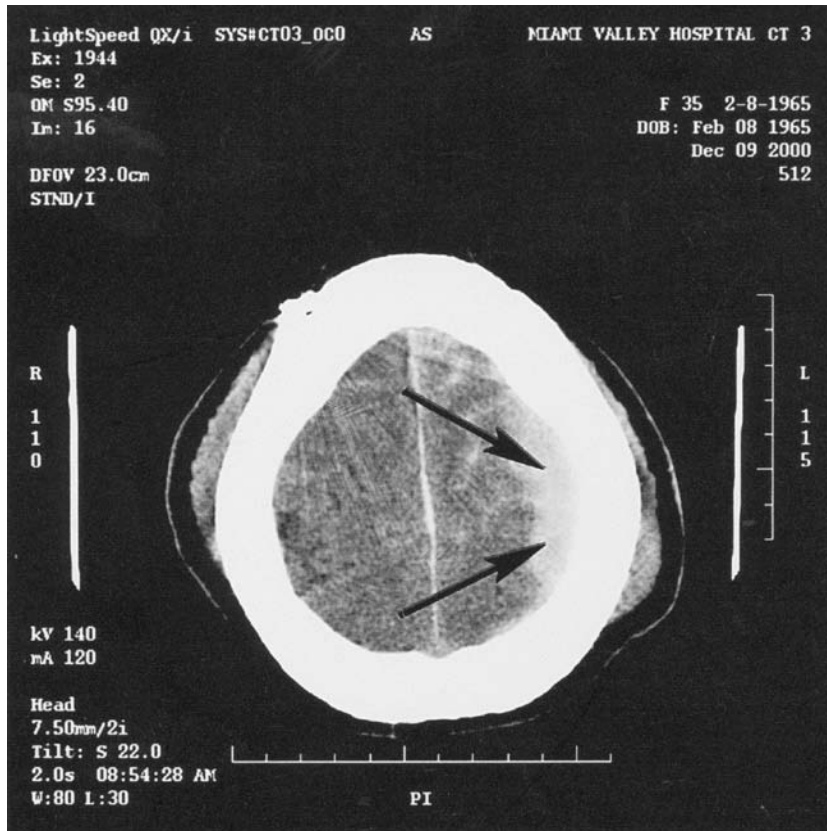
fracture should be sought. Skull x-rays have generally been suboptimal in demonstrating these fractures, but now high-resolution cranial CT with thin sections is the best modality for demonstrating such fractures.<sup>24</sup>

Depressed skull fractures occur when a fracture edge locks under the intact adjacent calvarium or when the skull bone fails to rebound. There is a high incidence of underlying brain injury when this occurs, and often the dura is torn by these fractures. Thus, if these findings are noted, the neuropsychiatry examiner should carefully look for other signs of underlying parenchymal injury and neuropsychological deficit. These fractures occur most often in the parietal and frontal bones, and they are found more frequently in young adults and adolescents than in adults. The depressed fracture is considered clinically significant when the fractured fragment is depressed below the edge of the intact adjacent inner table of the skull or the fracture overlies a major dural venous sinus or the motor cortex. These fractures are also significant if they are associated with dural tears, penetration of the cerebral parenchyma by a foreign object, or the presence of an underlying parenchymal injury.<sup>24</sup>

### Contusions

A brain contusion is a contact injury that results from a cerebral gyrus striking the inner surface of the skull. Thus, it is a bruise on the surface of the brain.<sup>25</sup> The areas of the brain most vulnerable to this type of injury are those adjacent to the floor of the anterior and middle cranial fossae, the sphenoid wings, and the petrous ridges. Therefore, the frontal and temporal poles and the under-





**FIGURE 5.1** CT scan revealing left lateral superior subdural hematoma. Note that the blood products are contiguous and follow the gyral pattern of the brain.

surfaces of the frontal lobes are most commonly involved. Less frequently, the inferior surface of the cerebellum may be bruised. These lesions often appear unrelated to the point of impact.<sup>26</sup>

With CT imaging, contusions may produce high-density (hemorrhagic) or low-density (non-hemorrhagic or hemorrhagic with partial voluming of hemorrhagic elements as a result of necrotic or edematous brain) areas of mass effect. The single most frequent hemorrhagic brain lesion seen on CT is a hemorrhagic contusion. One problem with CT in recognizing small superficial contusions when a thin stripe of high-density cortical blood lies next to high-density bone is that an artifact may obscure the blood in the hemorrhage. Blood on the surface of the brain adjacent to bone may produce a beam-hardening artifact. Contusions of the parietal vertex and inferior temporal lobe may be partially volumed with contiguous bone on the axial CT slice, resulting in an overall bone density that obscures the presence of the contusion. Usually, coronal images are not obtained during CT studies in the emergency department, and as a result, such contusions are frequently missed.<sup>14,27</sup> [Figure 5.2](#) demonstrates bilateral encephalomalacia of the subfrontal cortex 4 months after hemorrhagic contusions of the undersurface of the frontal lobes formed during a vehicular accident.

### Brain Stem Injury

The most common location for acute posttraumatic brain stem injury is the dorsolateral aspect of the upper midbrain. This injury occurs as the brain stem strikes the edge of the tentorium. However, CT imaging is a very poor modality for detecting this type of injury. Only 10% of brain stem injuries are clearly detected on CT, and they are usually associated with diffuse axonal injury.<sup>13,25</sup>

## Extradural (Epidural) Hematoma

The epidural space is a potential space between the cranial periosteum and the inner table of the skull. The dura and periosteum are anatomically inseparable. The potential epidural space is tightly bound at the sutural margin. The dural blood supply lies on the inner table of the skull between the skull and the dura, so that with fracture of the inner table, laceration of a meningeal artery is possible. Not all epidural hematomas are arterial in nature, as some are venous in origin and arise because of a disruption of a major dural venous sinus.<sup>13,28</sup> A skull fracture is found in more than 95% of the cases of epidural hematoma. However, in young children, since the skull is quite plastic, epidural hematoma can occur without fracture. Epidural hematomas occur frequently in the posterior fossa as a result of tearing of dural veins or sinuses. The arterial epidural hematoma often enlarges due to systolic blood pressure. However, the venous epidural hematoma seldom enlarges.<sup>29</sup>

The CT appearance of an epidural hematoma depends on the source of the bleed, the interval between the time of injury and the CT acquisition, the severity of the hemorrhage, and the degree of blood clot organization or breakdown. The vast majority of epidural hematomas have the appearance of a biconvex lens on the CT scan. This high-density extraaxial mass on acute CT scan does not cross suture margins. Vertex epidural hematomas may not be seen on axial CT images unless they have a significant mass effect as a result of pushing the dura onto the brain. An epidural hematoma is usually homogeneously hyperdense on CT. If focal isodensity or hypodensity zones are noted within the hematoma, this usually indicates the presence of active bleeding or a coagulopathy. An irregular hypodense swirl correlates with active bleeding in the majority of cases.<sup>30</sup> [Chapter 1](#) describes further the clinical features of epidural hematomas.

## Subdural Hematoma

The subdural space is a potential space that lies between the dura and the arachnoid membranes. During trauma, the arachnoid may be torn and separated from the dura and associated with tearing of the bridging veins by rapid acceleration or deceleration of the head. Subdural hematomas are classified as either acute or chronic. They do not cross the midline because they are fixed by sites of dural attachment at the falx and tentorium.<sup>31</sup> Subdural hematomas represent 10 to 20% of all craniocerebral trauma cases and occur in up to 30% of fatal brain injuries. An acute subdural hematoma can appear isodense against gray matter if the hemoglobin concentration is below 10 to 11 g/dl. If the subdural hematoma is not isodense, it is easily recognized on CT as an extraaxial, crescentic, and homogeneously hyperdense collection of blood that conforms to the cerebral surface. It often has a mass effect that can be gauged by the degree of sulcal effacement and inward buckling of the gray matter–white matter interface. Often there is a midline shift of the falx noted.<sup>25</sup> Bilateral isodense subdural hematomas may cause diagnostic difficulty, but they can be detected if one pays attention to identifying the displacement of gray matter with effacement of cortical sulci and compressed ventricles. [Figure 5.1](#) demonstrates well a posttraumatic subdural hematoma found the day of injury. Notice the conformation of blood products to the left superior brain surface. Contrast enhancement of the CT scan may be needed to assist with diagnosis of bilateral isodense subdural hematomas.<sup>32</sup>

The neuropsychiatric examiner may be faced with evaluating a person with a chronic subdural hematoma. The subdural hematoma is thought to arise from a slow effusion of venous blood into the subdural space. Unlike acute subdural hematomas, parenchymal brain injury often is not found in association with chronic subdural hematomas.<sup>33</sup> The CT appearance of a chronic subdural hematoma depends on the interval between the last major episode of bleeding and the current examination. In most cases, the blood products have broken down to a point where the fluid appears to be low density relative to the brain. However, the high protein content of the fluid makes the density higher than that of cerebrospinal fluid. A chronic subdural hematoma can be of low density, high density, isodensity, or mixed density. In isodense subdural hematomas, the breakdown of blood

products has reached a stage where there is essentially no difference between the density of the hematoma and that of the adjacent brain.<sup>34</sup>

In the abused child, acceleration–deceleration forms of injury may cause a whiplash shaken injury or shaken impact injury. These children usually do not have evidence of skull fractures or bruises, but they may have fractures of the long bones and swollen basal ganglia. The injury may consist of a syndrome of subdural hematomas associated with subretinal hemorrhages and long bone fractures.<sup>35,36</sup> On CT scan, parietal-occipital acute interhemispheric subdural hematomas often are found.<sup>37</sup> These subdurals are hyperdense when acute. In patients who are brought to medical attention a week or more after injury, the subdural hematomas may be isodense or hypodense relative to brain tissue, and therefore more difficult to recognize on CT. Sometimes, diffuse brain swelling is seen on CT accompanying shaking injury, and at the present time, its etiology remains not completely understood.<sup>14</sup>

### **Subarachnoid Hemorrhage**

Subarachnoid hemorrhage accompanies most cases of head trauma. It can be caused by direct injury to the pial vessels, blood from a hemorrhagic cortical contusion, or extension of an intraventricular hemorrhage into the subarachnoid space. On a nonenhanced CT scan, acute subarachnoid hemorrhage appears as a linear, high-density fluid collection within the superficial sulci and cerebrospinal fluid cisterns. CT defines acute subarachnoid hemorrhage quite effectively.<sup>13</sup> CT is the procedure of choice for identifying the radiographic findings of subarachnoid hemorrhage because blood that occupies the full thickness of a CT slice reveals the increased density as a distinct area of brightness. When a subarachnoid hemorrhage is present along the falx, it typically disappears during the ensuing week.<sup>33</sup> In children, the incidence of subarachnoid hemorrhage identified on CT increases with the increasing severity of a head injury.<sup>1</sup>

When the patient is examined long after the initial trauma, blood in the subarachnoid space may have decreased in density to isodense so that the subarachnoid spaces appear obliterated. However, they are not; thus, subarachnoid hemorrhage is difficult to appreciate when the CT study is done more than several days after trauma. As noted in [Chapter 1](#), subarachnoid hemorrhage may cause fibroblastic proliferation within the subarachnoid space and the arachnoid villi. This may lead to the production of a communicating hydrocephalus. As a result, normal-pressure hydrocephalus may develop with a resulting dementia syndrome, which the neuropsychiatric examiner should consider during evaluation.

### **Intraparenchymal Hemorrhage**

Large intracerebral hematomas generally occur in the same distribution of contusions. That is, they are mostly found in the frontal and temporal brain areas. They may be related to a hemorrhagic contusion into which bleeding has occurred with clot formation. The clot may dissect through the white matter, or it may arise from the rupture of a penetrating vessel deep within the white matter.<sup>38</sup> The intracerebral bleeding may occur from *coup* or *contrecoup* mechanisms. Other areas wherein intraparenchymal hemorrhage may occur are the anterior and middle cranial fossae, the sphenoid wings, and petrous ridges. On CT, even contusions without significant hemorrhage appear as high-density areas. However, if there is significant blood involved, they will appear focal, fairly well margined, and hyperdense.<sup>13</sup> They may be found to have a surrounding rim of hypodensity caused by extravasated serum from a retracting clot. During the week after the formation of the hematoma, edema develops around the structure and extends through the white matter pathways, causing an increased mass effect. Most intracerebral bleeding is demonstrated on the initial day of injury; however, a small percentage of bleeding develops in a delayed fashion and appears 1 to 7 days after the injury. These delayed hematomas are more likely to be seen in

the frontotemporal and temporal regions.<sup>33</sup> **Figure 8.5** reveals a frontotemporal intraparenchymal hemorrhage in a child injured at age 4½ during a motor vehicle accident. This CT was obtained a few hours after injury.

In the weeks following formation of the hematoma, the mass will decrease in size and density because of chemical breakdown of the globin molecule. Eventually, the density of the hematoma on CT will approximate that of the adjacent brain. Usually, the decrease in mass effect does not follow. Thus, while the hematoma may not be apparent as a density difference, the mass effect persists on CT. New vessel formation occurs in the tissue surrounding the hematoma, and if a contrast agent is injected, there will be an enhancement of a rim of tissue surrounding the hematoma.<sup>24</sup> When a clot is no longer visible on CT, it remains highly visible on MRI. Exactly how long the clot will remain visible on MRI is uncertain, but Zimmerman has followed patients for more than 4 years, during which time residual high-signal-intensity methemoglobin on T1-weighted images was present.<sup>1</sup> On CT, a hematoma has a high density as a result of the relative density of the globin molecule in attenuating the x-ray beam.<sup>47</sup> Clot retraction occurs over the hours following hematoma formation and serum is extruded. The hematoma becomes higher in density after clot retraction. As the globin molecule breaks down, the density of the clot progressively diminishes. Clot density decreases from the periphery inward. A 2.5-cm clot becomes isodense in 25 days.<sup>47</sup> However, the clot is not gone; it simply is no longer visible on CT. Slowly, macrophages digest the blood products, and a cavity within brain tissue will typically be found following an old hematoma.

### **Intraventricular Hemorrhage**

As many as 25% of patients with severe head injuries have intraventricular hemorrhages. Focal and diffuse areas of high attenuation are identified within the ventricles following CT imaging in these cases. Blood tends to settle in the dependent portions of the ventricles (i.e., the occipital horns) where a cerebrospinal fluid–blood level forms. If no rebleeding occurs, intraventricular hemorrhage is rarely seen after about 1 week.<sup>25</sup> Intraventricular hemorrhage frequently exists with other findings of head trauma. The hemorrhage can be a consequence of tearing of subependymal veins or rupture of the ependymal layer and an extension of a subarachnoid hemorrhage or a parenchymal hemorrhage.<sup>32</sup> CT reveals this finding effectively.

### **Diffuse Axonal Hemorrhage**

Diffuse axonal injury was covered clinically in the “Diffuse Brain Damage” section of **Chapter 1**. As previously learned, shear–strain forces that develop during rotational acceleration or deceleration of the head are the forces most likely to produce diffuse axonal injury. This has been confirmed in primate studies.<sup>39</sup> Most injuries will be noted in the lobar white matter, particularly at the corticomedullary junction of the frontal and temporal lobes. Diffuse axonal injury may also occur near or in the corpus callosum, or in the dorsolateral aspect of the brain stem in cases of severe trauma. When corpus callosum or brain stem lesions are present, rarely will they occur without associated lesions in the lobar white matter. About 75% of callosal injuries occur in the posterior body and splenium of the corpus callosum because the posterior falx prevents lateral displacement of the hemispheres during rotational acceleration of the head.<sup>40</sup>

As Zimmerman<sup>1</sup> points out, when one cerebral hemisphere is placed in motion relative to the other, shearing stresses result along the tracts of the white matter axons that interconnect the two hemispheres. The neuropsychiatric examiner is most likely to see this among persons who have been involved in high-speed motor vehicle accidents or falls from height. The patient is generally rendered comatose and then has a prolonged hospital course. CT examination of these patients may be unremarkable, revealing only cerebral swelling or small focal hemorrhages. In summary, the lesions in diffuse axonal injury occur in four sites: (1) corpus callosum, (2) corticomedullary

junctions, (3) upper brain stem, and (4) the basal ganglia.<sup>41</sup> The presence of a small amount of intraventricular blood in the occipital horn of one or both ventricles should arouse suspicion that there has been a tear of the corpus callosum with transependymal extension of the bleeding. However, it often is not possible to see small hemorrhages in the corpus callosum on CT.<sup>42</sup> After edema resolves and hemorrhage is physiologically removed, the CT scan may appear normal even though the patient has significant cognitive and behavioral abnormalities. In other cases, the follow-up CT scan may show only generalized cerebral atrophy.<sup>43</sup>

## **Brain Swelling**

Diffuse cerebral swelling is commonly associated with closed-head injury and is well visualized by CT. Massive cerebral edema may lead to higher mortality outcome among all possible secondary traumatic lesions. This can cause secondary brain injury, as discussed in the “Diffuse Brain Damage” and “Secondary Injury after Head Trauma” sections of [Chapter 1](#). CT findings of edema are obliteration of the cerebral sulci and basal cisterns and the effacement of the gray matter–white matter interface.<sup>13</sup> On normal CT soft tissue window settings, the cerebellum, the cerebral vasculature, and the dural surfaces (falx and tentorium) appear hyperdense against the background of diffusely swollen, edematous hypodense brain.<sup>25</sup> A herniation across the tentorium is commonly present, and the mortality rate is high if swelling is not controlled quickly.

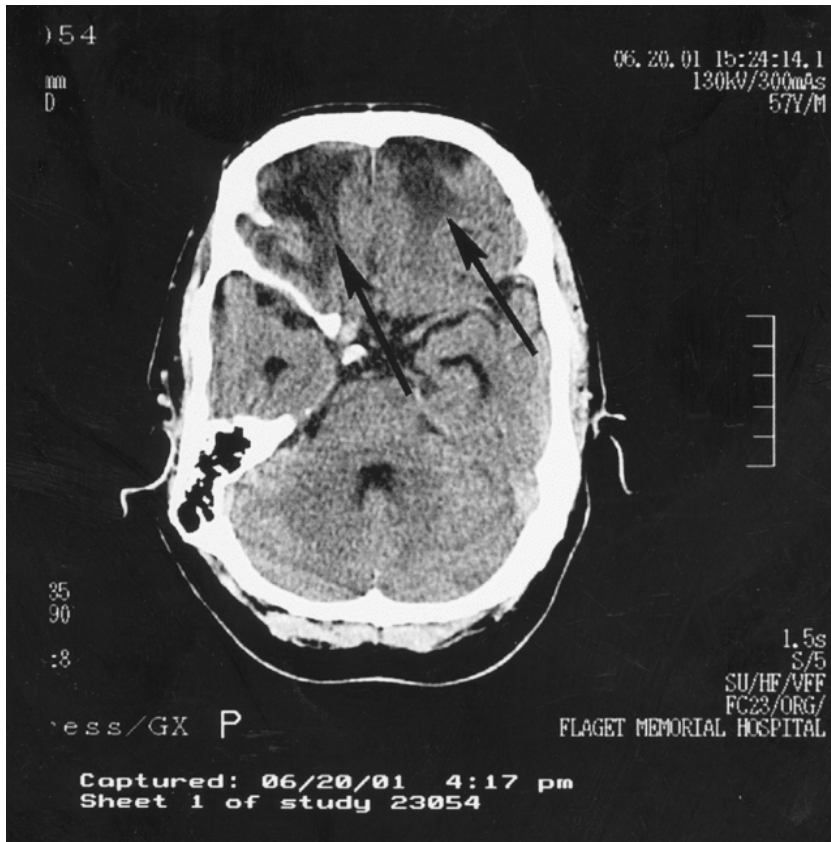
## **Brain Shift and Herniation**

Four main types of brain displacement can occur: (1) subfalcine, (2) descending and ascending transtentorial, (3) descending and ascending transalar, and (4) cerebellar tonsillar herniation.<sup>22</sup> Subfalcine herniation describes a midline shift that displaces the cingulate gyrus beneath the falx. A midline shift of 5 mm or more is considered significant from a surgical standpoint. A shift of this magnitude is associated with a 50% mortality rate.<sup>44</sup> In transtentorial herniation, the descending type is the result of medial and inferior displacement of the uncus and parahippocampal gyrus of the temporal lobe through the tentorial notch. On CT scan, it will be seen as an encroachment on the lateral aspect of the ipsilateral suprasellar cistern. In severe cases, the brain stem will be displaced and the contralateral cerebral peduncle will be compressed against the adjacent tentorial incisura. Complete uncal herniation results in obliteration of the suprasellar and perimesencephalic cisterns.<sup>45</sup>

Ascending transtentorial herniation is much less common than the descending variety but may occur in two clinical situations: (1) direct effect of a posterior fossa mass, or (2) following rapid decompression of a supratentorial space-occupying lesion.<sup>25</sup> In the first case, the vermis is pushed upward to obliterate the quadrigeminal plate or the superior cerebellar cisterns. CT will reveal flattening of the posterior aspect of the quadrigeminal plate cistern. Eventually, compression of the cerebral aqueduct causes hydrocephalus of the third and lateral ventricles. Transalar herniation refers to brain shifts across the sphenoid wing (ala). These shifts may be caused by swelling or bleeding in the anterior cranial fossa (descending type) or in the middle cranial fossa (ascending type). If transalar herniation is severe enough, it can cause infarctions in the distribution of both the anterior and middle cerebral artery branches.<sup>46</sup> Tonsillar herniation results from an enlarging mass in the posterior fossa or following supratentorial cerebral swelling. The CT scan will demonstrate crowding of the cisterna magna by the downward displacement of the cerebellar tonsils. This results in an obliteration of the cerebrospinal fluid cisterns around the medulla. The ultimate result of tonsillar herniation is cardiopulmonary arrest due to brain stem compression.<sup>25</sup>

## **Posttraumatic Neurodegeneration**

Certain neuropathological changes take place following a traumatic hemorrhagic contusion. The evolution of these changes can be correlated with imaging studies.<sup>48</sup> Four distinct phases occur:



**FIGURE 5.2** CT scan revealing a volume averaging that appears as an area of decreased density at the site of contusion.

(1) acute damage, (2) liquefaction of the contusion with the development of edema, (3) repair during which macrophages remove blood elements and damaged tissue causing proliferation of blood vessels, and (4) sloughing of necrotic tissue and forming of cystic cavities.<sup>1</sup> During the liquefaction phase, the softening and swelling that result from edema formation occur between the third and seventh days after injury. At this time, the components of hemorrhage are converted from deoxyhemoglobin to methemoglobin. Subsequently, the CT scan will reveal a volume averaging that may appear as an area of decreased density at the site of contusion<sup>48</sup> (see [Figure 5.2](#)). This CT finding is dependent upon the relative proportions of globin and water within the brain tissue. This is a critical time during the acute care of the brain-injured patient, as swelling and edema may increase the mass effect and produce cerebral herniation.

During the third phase, new blood vessels proliferate around the area of healing. However, a blood–brain barrier disturbance is present. At this point with CT imaging, if a contrast agent is given, enhancement analogous to that seen with a cerebral infarction occurs at the margin of the contusion.<sup>48</sup> During the fourth stage, evolution occurs slowly over a 6- to 12-month period. Contused brain tissue may be sloughed into the cerebrospinal fluid pathways such that an irregular surface of the contused portions of the hemisphere results. CT scan at this time will show an area of decreased density within the brain parenchyma, often with enlargement of the overlying cerebrospinal fluid spaces. The size of the cortical gyri may diminish, and the adjacent underlying ventricle may increase in size.<sup>48</sup> Most neuropsychiatric brain trauma examinations will take place during or after this fourth stage.

## MAGNETIC RESONANCE IMAGING

### Use in the Acute Care Setting

As noted previously, MRI now is rarely used in the acute care setting; however, this role is rapidly shifting with the advent of fast MRI. In the detection of subacute and delayed sequelae of brain trauma, MRI is more sensitive than CT. If CT cannot explain the current clinical setting, such as a focal neurological deficit or prolonged period of unconsciousness, then MRI should be used.<sup>13</sup> One of the great advantages of modern CT scanners has been their ability to assess a head injury patient in less than 5 min, allowing prompt diagnosis of expanding intracranial hematomas and thereby facilitating early surgical intervention.<sup>49</sup> This is likely to be true for the immediate future, although as this book is written, MRI is beginning to capture some of the roles formerly held by CT in the acute imaging of brain injury. With the advent and refinement of newer, fast imaging techniques and improvements in scanner hardware, the examination time for MRI has been reduced to 2 to 3 min for high-quality, T1-weighted scans using standard short TR/TE spin-echo (SE) techniques. Some have suggested that T1-weighted scans alone may be adequate for detecting virtually all significant intracranial hematomas.<sup>50</sup> Recently, fast spin-echo (FSE) pulse sequences have been developed that allow proton density (PD)-weighted and T2-weighted scans to be obtained in less than 2 min.<sup>51</sup> Fluid-attenuated inversion recovery (FLAIR) T2-weighted sequences have also been developed that are rapid and highlight both parenchymal lesions that touch the subarachnoid space and extraaxial hemorrhages as they suppress normal cerebrospinal fluid signal. T2-weighted gradient-echo scans are used in the evaluation of both acute and chronic trauma, and they may be acquired in less than 2 min as well.<sup>52</sup> Current, state-of-the-art MRI scanners can complete a thorough study of brain injury patients in less than 15 min.<sup>53</sup>

Gentry<sup>53</sup> believes that all moderate to severe head injury patients should be evaluated with MRI at some point during the first 2 weeks after injury. The full extent of traumatic brain injury will not be determined fully if only CT is used to evaluate this group of patients. MRI is clearly more valuable than CT for assessing the full magnitude of injury. It also provides more accurate information regarding the expected degree of final neurologic recovery.<sup>53</sup> The detection by MRI of traumatic brain lesions is summarized in [Table 5.2](#).

### Skull Fracture

MRI is not useful for detecting skull fractures. In general, CT is superior even to planar skull x-ray for assessing depressed skull fractures. High-resolution CT with thin slices will easily evaluate facial and orbital fractures and basilar skull fractures.

### Contusions

MRI is extremely sensitive for the detection of hemorrhagic and nonhemorrhagic cortical contusions. These are the second most frequently encountered group of primary intraaxial lesions. They comprised about 44% of intraaxial lesions in a series of studies.<sup>54</sup> As previously noted, these lesions involve the superficial gray matter of the brain and are most frequently found in the inferior, lateral, and anterior aspects of the frontal and temporal lobes. They are often hemorrhagic and superficially located. Both T1-weighted and T2-weighted images clearly demonstrate hemorrhagic contusions. Multiple research studies have demonstrated the superiority of T2-weighted spin-echo images over either CT or T1-weighted spin-echo images in the detection of nonhemorrhagic contusions. The MRI signal changes are most likely related to abnormally increased water content due to edema in the lesion.<sup>55</sup>

Because brain gray matter is much more vascular than the white matter, cortical contusions are much more likely to be hemorrhagic than diffuse axonal injury lesions (52 vs. 19%).<sup>53</sup> The hemorrhagic foci may vary in size from small, petechial hemorrhages to larger, nonhemorrhagic

**TABLE 5.2**  
**MRI and Traumatic Brain Injury**

Lesion	Image Findings
Contusions	Both T1-weighted and T2-weighted spin-echo images will demonstrate hemorrhagic contusions; T2-weighted spin-echo images superior for demonstrating nonhemorrhagic contusions <sup>1,4,55</sup>
Brain stem injury	Detected by sagittal or coronal T2-weighted or FLAIR axial images <sup>4,59,60</sup>
Epidural hematoma	High-signal-intensity extruded serum seen as a biconvex form; T1-weighted and T2-weighted images will detect acute lesion, while subacute stage seen by T1-weighted imaging; displaced dura usually seen on T2-weighted imaging <sup>1,4,61</sup>
Subdural hematoma	Subacute crescentic lesion detected by T1-weighted and T2-weighted imaging; isodense subdural on CT detected by T1- and T2-weighted imaging; FLAIR imaging may detect subtle coincidental subarachnoid hemorrhage; chronic subdural hematoma is seen as high-signal-intensity methemoglobin, low-signal-intensity protein fluid on T1 weights; proton density weighting demonstrates fluid to be higher in signal than CSF <sup>1,4,64,65</sup>
Subarachnoid hemorrhage	T2-weighted FLAIR quite sensitive <sup>1,4,67</sup>
Intraparenchymal hemorrhage	Deoxyhemoglobin signal hypointense to isointense on T1-weighted images; markedly hypointense on T2-weighted images; methemoglobin, high signal intensity on T1 weights, black on T2-weighted images; hemosiderin, black on T2-weighted images <sup>1,4,68-70</sup>
Intraventricular hemorrhage	Hyperintense relative to CSF on T1-weighted images; especially intense on T2-weighted FLAIR images <sup>1,4,73</sup>
Diffuse axonal injury	Small areas of hyperintense signal on T2-weighted images early after injury; hypointense T2-weighted signal seen as lesion ages due to hemosiderin; gradient-echo sequences superior for detecting old DAI hemorrhages <sup>1,4,55,74,75</sup>
Chronic neurodegeneration	Encephalomalacia detected as hypointense signal on T1-weighted images with high signal intensity on T2-weighted images; ventricular dilatation and cortical atrophy are common <sup>1,4,80-85</sup>

*Note:* DAI = diffuse axonal injury.

zones of injury. Multiple large, confluent regions of hemorrhage may occupy most of an entire lobe following severe trauma. Contusions, when present, tend to be multiple and bilateral.<sup>56</sup> Temporal lobe lesions are most likely to occur just above the petrous bone or slightly behind the greater sphenoid wing. Frontal lobe lesions tend to lie just above the cribriform plate, the orbits, the planum sphenoidale, or the lesser sphenoid wing. The parietal and occipital lobes are the least likely to demonstrate cortical contusions. About 10% of brain trauma-causing contusions may show lesions in the cerebellum. These are typically found in the superior vermis, tonsils, and inferior hemispheres.<sup>54</sup> Gentry's series of trauma patients revealed that cortical contusions were much less likely to be associated with severe initial impairment of consciousness than is diffuse axonal injury. If a severe impairment of consciousness was present with a contusion, typically there were very large, multiple, bilateral lesions or it was associated with diffuse axonal injury.<sup>57</sup> Thus, a minimal initial impairment of consciousness may be associated with significant cortical contusions.

### Brain Stem Injury

Trauma-induced brain stem injuries include contusion and shearing injury. These are most common within the dorsolateral aspect of the upper midbrain and usually occur because the brain stem strikes the edge of the tentorium. Secondary injury to the brain stem can occur by hypoxic or ischemic injury and is associated with Duret's hemorrhages. These hemorrhages are caused by prolonged transtentorial herniation and are usually located in the midline within the midbrain and



pontine tegmentum.<sup>25</sup> They often accompany transtentorial herniation, resulting in damage to the medial pontine branches of the basilar artery.<sup>13</sup>

The hypothalamus and pituitary are the most frequently injured portions of the diencephalon.<sup>58</sup> Injury to these structures often leads to the syndrome of inappropriate antidiuretic hormone, causing a diabetes insipidus syndrome. Anterior pituitary dysfunction may also be found, causing alteration of other hormonal systems as well. Trauma affecting the lower brain stem is usually mixed with trauma to the cerebral hemispheres. Isolated significant lower brain stem injuries are rare.<sup>58</sup> The cerebellum is not part of the brain stem proper, but on rare occasions, it may be injured as a result of trauma to the posterior fossa.

Brain stem lesions were thought to be fairly insignificant until the advent of MRI.<sup>59</sup> It may be required, if brain stem injury is suspected, to obtain additional sagittal or coronal T2-weighted or FLAIR axial images to detect such lesions. Some studies indicate that prognosis can be deduced by brain stem MRI. Two patterns of brain stem injury have been noted. The good prognosis group showed ventral brain stem lesions or dorsal superficial brain stem lesions. On the other hand, the poor prognosis group showed deep dorsal brain stem lesions. These findings may be detected only in the acute stage, and long after injury, MRI may not predict prognosis as well.<sup>60</sup>

### **Extradural (Epidural) Hematoma**

In a stable patient, MRI will reveal a biconvex mass separated from the overlying dura by a thin stripe of high-signal-intensity extruded serum lying between the clot and the dura. This will be seen on both T1-weighted and T2-weighted imaging. In the subacute stage, MRI will show the epidural hematoma as a biconvex high-signal-intensity mass on T1-weighted images. The dura is often visible as a thin, hypointense stripe displaced inward by the clot.<sup>1</sup> On good-quality MRI, the dura often can be seen to be displaced away from the inner table of the skull. It may be visualized as a thin line of low signal intensity between the brain and the biconvex-shaped hematoma. If one visualizes the dura on MRI, this allows one to be absolutely certain of the diagnosis of an epidural hematoma. With CT, small epidural hematomas cannot be differentiated always from a subdural hematomas because, in these cases, the epidural hematoma may not have a classic biconvex shape on CT.<sup>4,49</sup>

As noted previously, venous bleeding can produce an epidural hematoma as well as arterial bleeding. Venous epidurals are much more variable in shape than those of arterial origin.<sup>61</sup> However, all venous epidural hematomas are invariably separated from adjacent brain by displaced dura that can usually be seen on T2-weighted imaging. Another characteristic feature of venous epidural hematomas is that they lie always in direct proximity to a dural sinus that is crossed by a fracture line.<sup>62</sup> A venous epidural hematoma, unlike a subdural hematoma, will often lie both above and below the tentorium. Since the pressure is lowered by venous bleeding rather than arterial bleeding, these clots expand more slowly and may be delayed in onset relative to an arterial bleed.<sup>63</sup>

### **Subdural Hematoma**

The outcome from subdural hematoma after trauma continues to be poor (35 to 90% mortality), primarily because of secondary forms of injury and associated underlying brain injury.<sup>4,62</sup> MRI of patients with subdural hematoma generally demonstrates a typical crescentic collection of blood between the brain and the falx, tentorium, or inner table of the skull. Subdural hematomas will be visualized on all MRI pulse sequences as crescentic areas that have a signal intensity that is always higher than that of the adjacent cortical bone.<sup>50</sup> The MRI signal appearance of the subdural hematoma will vary with the age of the lesion. MRI has been shown to be considerably more sensitive than CT for detection of subdural hematoma. Gentry and colleagues noted through scientific study that CT detected only 53% of subdural hematomas when compared with MRI T1-weighted and T2-weighted scans, which detected 70 and 95% of lesions, respectively.<sup>50</sup> MRI has an advantage over

CT in that direct, multiplanar imaging is more easily accomplished. This, together with the ability to show a subacute hemorrhage as high-signal-intensity methemoglobin, has distinct advantages in demonstrating subacute subdural hematomas. This occurs in part because the cortical bone does not produce a signal, so that the methemoglobin is seen unimpeded by a bone-causing artifact.<sup>64</sup> However, if MRI is used in the very acute phase, when the blood is still in the deoxyhemoglobin stage, recognition of a subdural hematoma is much more difficult. At that stage, displacement of the brain and extrusion of serum from the subdural hematoma become important criteria in identifying the subdural hematoma. Extruded serum comes to lie laterally between the clot and the inner table of the skull and medially next to the compressed arachnoid. The serum is high in signal intensity on T2-weighted imaging, while the deoxyhemoglobin is low in signal intensity.<sup>24</sup>

MRI has revolutionized the ability to identify chronic subdural hematomas because with coronal imaging, the relationship between the brain and the inner table of the skull can be exquisitely demonstrated.<sup>1</sup> When the brain is displaced from the inner table, the signal intensity of the mass producing the displacement becomes critical. Chronic subdural hematomas may be seen as high-signal-intensity methemoglobin, lower-signal-intensity proteinaceous fluid on T1 weights, or higher-than-CSF-signal-intensity proteinaceous fluid on proton density-weighted imaging.<sup>65</sup> Gadolinium contrast can be used to bring out subdural membranes as an area of increased contrast enhancement on T1-weighted images. Sometimes contrast will leak into the subdural hematoma and increase its overall signal intensity on T1-weighted images. Subdural hygromas (CSF-filled) do not show membrane formation, and they do not enhance with gadolinium. They behave in the manner of a cerebrospinal fluid-filled subdural space on CT, and on T1-weighted images, proton density-weighted images, and T2-weighted images.<sup>1</sup>

### **Subarachnoid Hemorrhage**

Subarachnoid hemorrhage is poorly demonstrated or not shown at all on MRI.<sup>66</sup> Oxyhemoglobin is not paramagnetic and does not produce a change in signal intensity that can be detected.<sup>1</sup> In Gentry's series of cases,<sup>4</sup> in trauma victims who had CT-documented subarachnoid hemorrhage, the hemorrhage was seen in only 15% of cases when followed up by MRI. Subarachnoid hemorrhage is seen on either T1-weighted or T2-weighted scans only when there are associated large focal clots. However, the recent addition of T2-weighted imaging using inversion recovery (FLAIR) has been shown to be quite sensitive to all ages of subarachnoid hemorrhage in both clinical and *in vitro* studies.<sup>67</sup>

### **Intraparenchymal Hemorrhage**

Traumatic intracerebral hematomas are focal collections of blood that most commonly arise from rotationally induced shear-strain injury to intraparenchymal arteries or veins.<sup>4</sup> These have been reported to occur in 2 to 16% of head trauma victims.<sup>49</sup> Differentiation from a hemorrhagic contusion or diffuse axonal injury is often difficult for the radiologist to complete. The distinction rests primarily with the fact that intraparenchymal hemorrhage primarily expands between relatively normal neurons. On the other hand, hemorrhage occurring within bruises or contusions is interspersed in areas of simultaneously injured and edematous brain.<sup>4</sup> Eighty to 90% of intraparenchymal hematomas are located in the frontotemporal white matter or within the basal ganglia. Lesions of this type are usually associated with other neuronal lesions or fractures of the skull. Interestingly, these patients may not lose consciousness, and 30 to 50% remain lucid throughout the duration of their injuries.<sup>62</sup> Delayed detection of intraparenchymal hemorrhage should be considered in patients who subsequently deteriorate in their level of consciousness following injury. This occurs in about 2 to 8% of all patients with severe head injury.<sup>63</sup>

When the clot is no longer visible on CT, it is highly visible on MRI, as noted previously. In fact, intraparenchymal hemorrhage may be visible indefinitely on MRI, due to the persistence

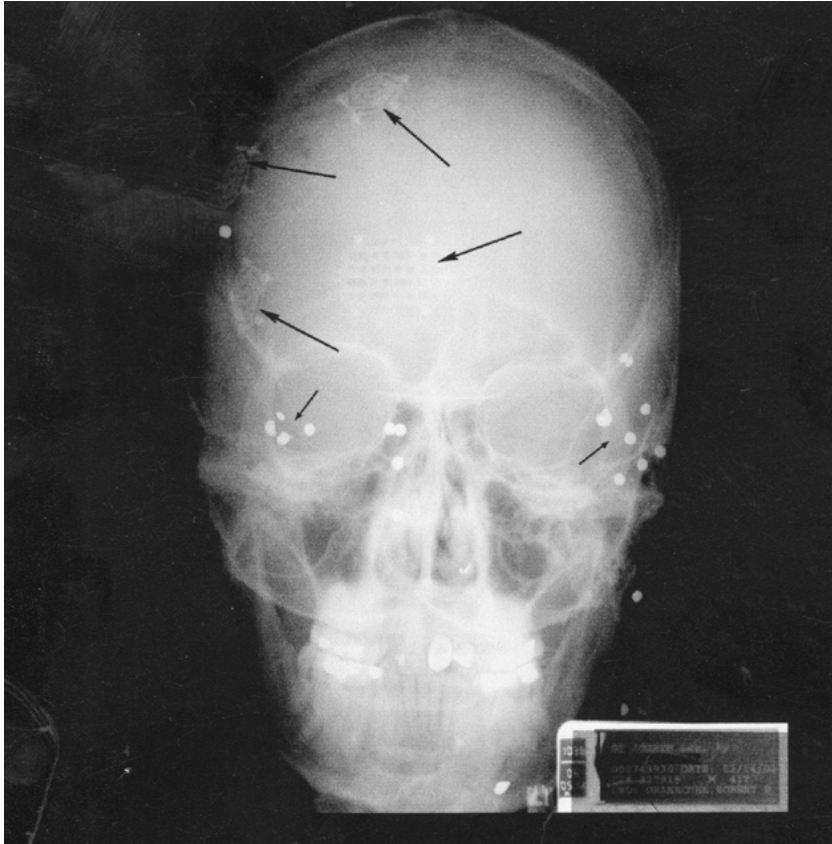
of hemosiderin deposits within macrophages around the lesion. It is not clear whether all hemorrhages produce hemosiderin. Wardlaw and Statham<sup>68</sup> studied 116 survivors of moderate to severe head injury and examined them 1 to 5 years after their injuries. Imaging was reviewed blindly and correlated with prior acute CT to determine how many hemorrhages from the acute stage were identifiable by virtue of hemosiderin deposition on late MRI. Of 106 hemorrhages detected acutely in 78 patients at the time of their injuries, 90% were visible as hemosiderin on late MRI. Of the hemorrhages without hemosiderin, 7 of 10 were in patients where other hemorrhages with hemosiderin were still visible elsewhere in the brain. This study indicates that about 10% of definite intraparenchymal hemorrhages will show no trace of hemosiderin on routine spin-echo MRI. Radiologists have been alerted to supplement routine spin-echo MRI with gradient-echo sequences if there is a reason to suspect, or specifically exclude, prior hemorrhage. If a physician is performing a neuropsychiatric examination of a person known to have had an intraparenchymal hemorrhage acutely at some significant time prior to the neuropsychiatric examination, he is well advised to order gradient-echo sequences in addition to standard sequences during the cognitive examination.

Blood goes through a sequence of changes during detection by MRI if imaging is performed on a serial basis. Blood in arteries or veins is in the oxyhemoglobin state. When it becomes a clot, it is changed to deoxyhemoglobin and later to methemoglobin.<sup>69</sup> On MRI, the deoxyhemoglobin is hypointense to isointense on T1-weighted images. On T2-weighted images, it is markedly hypointense (a susceptibility effect). Susceptibility refers to the inherent magnetic fields within the different tissues that constitute the brain. Intact red blood cells containing deoxyhemoglobin have a susceptibility different from that of the surrounding extracellular fluid. If a proton is exposed to the varying local magnetic fields, one due to intracellular deoxyhemoglobin and one due to surrounding extracellular fluid, it will have its spin thrown out of phase so that it does not give back a signal. This appears as an area of blackness on MRI. About 3 days after the formation of the hematoma, deoxyhemoglobin is oxidized to methemoglobin. It will now appear as a high signal intensity on T1-weighted images. This occurs first at the periphery of the hematoma. On T2-weighted images, the hematoma appears black. As red blood cells die and rupture, a solution of methemoglobin is formed that is bright on both T1-weighted and T2-weighted images.<sup>70</sup>

Intracellular methemoglobin is found first around 3 days after the formation of the hematoma. The formation of intracellular methemoglobin progresses from the periphery of the hematoma toward the center.<sup>72</sup> Extracellular methemoglobin is found about the end of the first week postinjury. Deoxyhemoglobin within the center of the hematoma may persist for weeks. Macrophages are mobilized and move in to digest the hematoma. As a result of the ingestion of blood products, hemosiderin is found within the lysosomes of the macrophage.<sup>71</sup> Again, this creates a susceptibility effect that makes the area of hemosiderin black on T2-weighted images. It then is found within the brain tissue at sites of traumatic bleeding, perhaps for the rest of the patient's life. Methemoglobin has been found for months to years following a brain injury, but it is eventually resorbed. However, the neuropsychiatric examiner should specifically ask the radiologist to look for hemosiderin when evaluating patients by MRI some length of time following their brain injuries. [Figure 5.3](#) shows a planar x-ray subsequent to a shotgun blast to the head during a wild turkey hunting accidental shooting. Note the titanium instruments placed during surgery. [Figure 5.4](#) reveals post-traumatic surgical changes and encephalomalacia detected by MRI at the time of a neuropsychiatric examination of this individual.

### **Intraventricular Hemorrhage**

The MRI appearance of intraventricular hemorrhage is variable. The blood is almost always hyperintense relative to cerebrospinal fluid on T1-weighted images. It is especially hyperintense on FLAIR scans, and this allows easy detection.<sup>4</sup> Gentry and others have studied intraventricular hemorrhage associated with diffuse axonal injury.<sup>73</sup> The etiology of intraventricular hemorrhage in



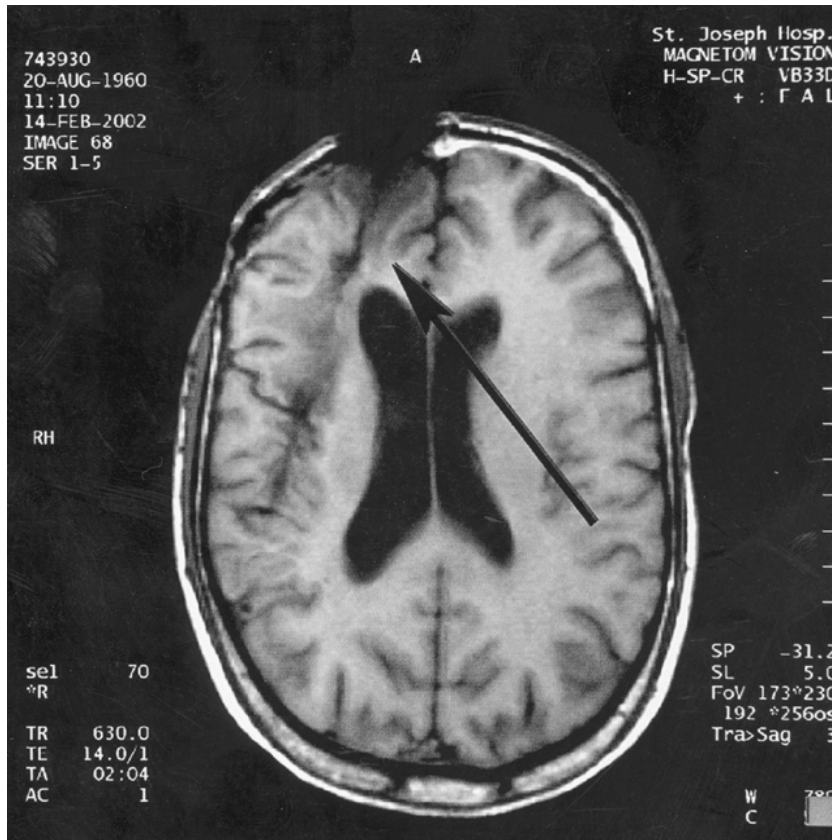
**FIGURE 5.3** Planar x-ray subsequent to a shotgun blast to the head. Note the titanium instruments placed during surgery.

most cases is due to rotationally induced tearing of subependymal veins on the ventral surface of the corpus callosum and along the fornix and septum pellucidum. These veins are often disrupted by the same force that causes diffuse axonal injury. Gentry et al.<sup>73</sup> found that intraventricular hemorrhage occurred in 60% of patients with diffuse axonal injury of the corpus callosum, but also in about 12% of patients without callosal injury ( $p < .002$ ). In those patients who had no callosal injuries, the hemorrhage was invariably due to dissection of a large intracerebral hematoma into the ventricular system.

### **Diffuse Axonal Injury**

MRI is superior to CT scanning in the detection of diffuse axonal injury. Lesions are generally located at the gray matter–white matter interface and are characterized by multiple small focal areas of damage<sup>74</sup> (see [Chapter 1](#)). Most lesions are nonhemorrhagic, but up to 20% may contain a small amount of hemorrhage. They occur in four primary locations: (1) lobar white matter, (2) corpus callosum, (3) dorsolateral aspect of the upper brain stem, and (4) internal capsule.<sup>55</sup>

Acute shearing injuries, such as the nonhemorrhagic lesions, occur as small oval or round areas of hyperintensity on T2-weighted images. A hemorrhagic lesion may have a central hypointensity within it on the T2-weighted images. Diffuse axonal injury also can be detected on FLAIR and proton density-weighted images. As the injury ages, in the chronic phase of diffuse axonal injury, hemorrhagic shear injuries are quite hypointense on T2-weighted images due to the presence of hemosiderin. At this point, gradient-echo imaging will increase the sensitivity for detecting hem-



**FIGURE 5.4** MRI revealing posttraumatic surgical changes and encephalomalacia.

orrhagic shearing injury. Gradient-echo sequencing is superior to other sequences in showing old hemorrhagic lesions.<sup>55,75</sup>

Diffuse axonal injury may rarely be seen without evidence of direct head trauma and with a delayed onset of coma.<sup>76</sup> This can occur in high-velocity accidents without immediate evidence of head injury. Other studies have confirmed findings of diffuse axonal injury on MRI following minor brain injury (Glasgow Coma score = 15).<sup>77</sup> However, diffuse axonal injury in the aforementioned cases cannot be assumed without visual evidence on MRI.

### **Posttraumatic Neurodegeneration**

How useful is MRI in predicting outcome severity following traumatic brain injury? Recent studies have found that the use of various MRI techniques at early and delayed time points can provide useful information with regard to the severity and clinical outcome of patients following traumatic brain injuries.<sup>78</sup> MRI performed early after head injury may provide several indicators for unfavorable outcome. In the severe head injury subgroup, lesions within the corpus callosum, the basal ganglia, and the midbrain are predictive of poor outcome.<sup>79</sup> In the older patient, it is often difficult to know if hippocampal volumes have changed due to trauma or the normal effects of aging. Bigler's group<sup>80</sup> studied 96 healthy volunteers and 94 patients with traumatic brain injury using coronal intermediate and T2-weighted MRI. No significant age group differences were found in the normative group from ages 16 to 65. Comparisons between patients with traumatic brain injuries and control subjects showed significant yet modest bilateral atrophic changes in hippocampal tissue and compensatory temporal horn enlargement in the patients with brain injury. The

hippocampal and temporal horn volumes were inversely correlated in the group with traumatic brain injury. This suggested a differential relationship of these structures in patients with brain injury, as compared with aged control subjects. In the subacute phase, these studies suggest that the volume of the temporal horn may be indicative of intellectual outcome, whereas the hippocampus volume appears to be indicative of verbal memory function. Encephalomalacia can be detected as a hypointense signal on T1-weighted images and as a high signal intensity on T2-weighted images.<sup>1</sup> Figure 8.3 demonstrates by MRI late-appearing hippocampal atrophy following motor vehicle head trauma as an adult. Figure 8.6 reveals degeneration of brain tissue and right cerebral atrophy in a brain-injured child. This MRI was obtained 1½ years following the CT image in Figure 8.5.

The thalamus and upper brain stem are, at times, injured following traumatic brain injury. Significant correlations have been observed between sensory-perceptual functioning, as measured by the *Reitan-Kløve Sensory Perceptual Examination*, and thalamic volume in brain-injured patients. A decrease in thalamic volume was associated with an increase in sensory-perceptual errors.<sup>81</sup> Many patients also will show ventricular dilatation and cortical atrophy. In those groups with the highest ventricular change, significantly lower memory scores will be found. However, these patients do not show significant differences on tests of intellectual functioning.<sup>82</sup> With regard to atrophy in association with the drug-abusing brain-injured patient, interesting results have been noted. Few studies have examined the consequences of alcohol and drug abuse on traumatic brain injury even though they commonly coexist. Since traumatic brain injury most frequently occurs in older adolescents and younger men, Barker's group<sup>83</sup> examined male participants between 16 and 30 years of age. Young substance abusers were compared with controls, and the third group of patients included substance abusers who had been traumatically brain-injured. When controlling for head injury severity, the effects of substance abuse in combination with traumatic brain injury resulted in greater atrophic changes than seen in either controls or substance abusers without evidence of brain injury. These findings suggest that deleterious interactions of substance abuse combined with traumatic brain injury result in greater neuropathological changes.

Brain-injured children also show significant posttraumatic defects, which can be detected by MRI. The depth-of-lesion model in children and adolescents has been used to predict severity in outcome. The deepest lesion present on the MRI is used for calculating the depth-of-lesion classification. The depth of lesion significantly correlates with Glasgow Coma Scale severity, the number of lesions, and the time of discharge from the rehabilitation unit vs. findings at 1-year follow-up. The depth of lesion is most predictive of the time the child will be discharged from the rehabilitation unit. On the other hand, the Glasgow Coma Scale is the most predictive indicator of the level of disability at 1-year postinjury. It is suggested that a depth-of-lesion classification of traumatic brain injury severity may have clinical utility in predicting functional outcome in children and adolescents who have sustained moderate to severe traumatic brain injury.<sup>84</sup> Unlike adults who often reveal a significant correlation between ventricular dilatation and neuropsychological outcome, children may not show the same pattern. Diminishment in size of the corpus callosum in children correlates strongly with several measures involving processing speed and visuospatial function. Ventricular enlargement in children appears to be less related to neuropsychological outcome. Quantitative measurement of the corpus callosum on MRI seems to more accurately reflect neuropsychological outcome in children rather than ventricular dilatation.<sup>85</sup>

In assessing the late effects of inflicted child abuse, signs of preexisting brain injury are often found at the time of the neuropsychiatric examination. These include cerebral atrophy, subdural hygroma, and *ex vacuo* ventriculomegaly. These findings are present in about 45% of children who have sustained inflicted traumatic brain injury and are found in no children with noninflicted traumatic brain injury. Retinal hemorrhage was only identified in inflicted traumatic brain injury children. Glasgow Outcome Scale scores indicate a significantly less favorable outcome for inflicted than for noninflicted traumatic brain injury.<sup>86</sup> Intraparenchymal hemorrhage, shear injury, and skull fractures are more frequent after noninflicted traumatic brain injury.<sup>87</sup> Children with severe nonin-

flicted traumatic brain injury may show frontal lobe changes even in the absence of focal brain lesions detected by MRI. Children who are received in the emergency department with a Glasgow Coma Scale score at or below 8 may show by MRI that the total prefrontal cerebrospinal fluid has increased and the gray matter volume has decreased relative to a mildly injured comparison group. The gray matter volume seems most reduced in the orbitofrontal and dorsolateral regions in children who have sustained severe brain injury relative to those youngsters who have sustained a mild head trauma. Nearly two-thirds of children who sustain severe closed-head injury are moderately disabled after an average postinjury interval of 3 years or more, whereas the majority of child patients with mild closed-head injuries attain a good recovery.<sup>88</sup>

## FUNCTIONAL IMAGING OF BRAIN TRAUMA

### SINGLE-PHOTON EMISSION COMPUTED TOMOGRAPHY

Single-photon emission computed tomography (SPECT) produces both quantitative and qualitative measures of cerebral blood flow. The most common radioligand used to produce brain imaging of traumatic injury is <sup>99m</sup>Tc hexamethylpropylene amine oxime (HMPAO). It is injected intravenously and accumulated by endothelial cell membranes within several minutes. It concentrates in these cells proportional to regional cerebral blood flow, and its activity may remain constant for up to 24 h. Because of this, SPECT is useful in that it can be injected during very controlled conditions, away from the noise and anxiety of the scanning room. A snapshot of the relative cerebral perfusion can then be collected at a time later, up to several hours later. A second radioligand sometimes used for SPECT is <sup>99m</sup>Tc ethyl cysteinate dimer (ECD). This radioligand produces less extracerebral uptake, and it has some dramatically different patterns of uptake within the brain tissue compared with HMPAO. Thus, there may be differences in uptake based on the ligand used, and therefore, variance in activity patterns may result not only from changes in brain activity but also from the radioligand itself. Most brain trauma SPECT studies in the U.S. now use HMPAO.<sup>120</sup>

Though PET provides the highest-resolution tomographic images of brain function, modern SPECT images have similar resolution, making any differences relatively inconsequential in most clinical applications applied to traumatic brain injury. While the breadth of radiopharmaceuticals available for brain SPECT is not as great as that for PET, the variety of SPECT tracers is expanding rapidly.<sup>121</sup> Images usually are obtained using multihead gamma camera-based systems. These are widely available because they can perform both head and body SPECT. State-of-the-art SPECT systems can be expected to provide high-resolution imaging of statically distributed brain radiopharmaceuticals with patient imaging times of approximately 10 to 20 min. Currently available three-head systems offer spatial resolution of about 6 mm in the cortex and about 7 mm at the center of the brain using HMPAO. The resolutions are approximately the same for ECD. One- or two-head gamma camera systems have resolution ranges of 7 to 10 mm.<sup>121</sup>

Computed tomography and magnetic resonance imaging have proved to be extremely useful in the evaluation of acute and chronic head trauma resulting in brain injury, as has been noted previously. However, structural imaging techniques, particularly in minor head trauma, do not always correlate with the cognitive and psychological deficits that patients manifest. In the mid-1990s, a report by the Therapeutics and Technology Assessment Subcommittee of the Academy of Neurology still considered SPECT in head trauma to be investigational.<sup>89</sup> However, by 2000, SPECT imaging in patients with closed cranial trauma was becoming recognized as a clinically useful evaluation procedure.<sup>90</sup> SPECT imaging should not replace CT or MRI for the identification of major structural lesions or the presence of hemorrhage, hematomas, or edema following brain trauma. However, functional imaging can contribute to evaluating alterations in perfusion and metabolism (by positron emission tomography) in the cerebral cortex, basal ganglia, and thalamus that may result from traumatic brain injury.<sup>90-93</sup>

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**TABLE 5.3**  
**SPECT and Traumatic Brain Injury**

Uses	Findings
Acute brain injury	Zone of reduced blood flow is larger than hemorrhagic lesions imaged on CT; contrecoup injuries more easily demonstrated than they are by structural imaging; frontal blood flow often reduced; overall blood flow often reduced; functional changes often seen distant from focal injuries; reduced thalamic blood flow often noted <sup>96,98,100,109</sup>
Neuropsychological outcome	Diffuse blood flow reduction reveals a high relationship to abnormal neuropsychological function; focal SPECT lesions may poorly correlate with neuropsychological outcome; there is a general relationship between frontal and thalamic blood flow and neuropsychological test performance; personality change has a stronger relationship to blood flow than cognitive changes <sup>5,114,115</sup>
Mild head injury	SPECT useful in detecting brain injury in mild trauma (GCS score > 12), particularly contrecoup injury; SPECT can provide an objective correlate of subjective complaints <sup>90,94,116</sup>

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SPECT images are obtained by injecting patients with radiopharmaceutical tracers that indirectly measure cerebral blood flow. These tracers are detected by nuclear cameras within a rotating multihead detector system generally found within a nuclear medicine department of most modern hospitals. Generally speaking, the same equipment used for cardiac SPECT imaging is applied to head SPECT imaging. Different radiotracers are used for head SPECT than cardiac SPECT, and specific computer software for analyzing head SPECT images is required. Thus, if the software is present and radiopharmaceuticals are available, almost any hospital performing cardiac SPECT can also perform head SPECT. However, a skilled nuclear medicine or neuroradiology physician is a must for proper interpretation of SPECT images.

While recent reports in the SPECT literature suggest that these techniques may be useful in establishing the presence of brain injury in minor head trauma, it is important to note that current research does not show a direct or strong correlation between diffuse cerebral perfusion deficits on SPECT and specific neurobehavioral impairments.<sup>90,94</sup> Alexander has argued that functional imaging with SPECT or PET should not be performed in the immediate postacute state, and that it should be used for patients with persistent behavioral, cognitive, or psychiatric symptoms after a reasonable recovery period has elapsed (approximately 6 months) with or without treatment.<sup>95</sup> However, neurosurgeons are applying SPECT or PET during acute care of brain trauma, as noted next. When used appropriately and with conservative interpretation, SPECT can play an important part in the clinical evaluation of patients with traumatic brain injury.<sup>90</sup> Table 5.3 lists important SPECT brain trauma findings.

### **SPECT and the Pathophysiology of Acute Brain Injury**

Early studies using SPECT in patients who had sustained acute head injuries demonstrated zones of reduced cerebral blood flow corresponding to the sites of structural lesions such as hematomas. Studies have reported that the zone of reduced cerebral blood flow on SPECT is larger than the hemorrhagic lesions imaged on CT.<sup>96</sup> Abdel-Dayem and others studied 14 patients while they were in brain injury-induced comas. CT imaging was carried out within 24 h of the head injury and SPECT within 72 h. This study found that the pericontusional low-density areas seen on CT scans were much smaller than the corresponding SPECT perfusion deficits.<sup>97</sup> Roper et al.<sup>98</sup> reported data indicating that lesions seen only on SPECT are often contrecoup injuries. His group noted that 7 of 17 patients had contrecoup injuries when they were evaluated with respect to the site of the initial impact to the head.



Distant nonfocal changes associated with trauma may reflect diffuse injury or hemispheric damage. Choksey's group<sup>96</sup> noted that when focal lesions were found on CT imaging after acute injury, there appeared to be low cerebral blood flow in the rest of the structurally normal ipsilateral hemisphere. Other authors also have found asymmetrical perfusion in patients who had sustained skull fractures and who showed no parenchymal lesions on CT.<sup>99</sup> In normal brain, frontal perfusion is usually approximately the same as flow in the occipital regions. In the studies reported by Abdel-Dayem et al. and Roper et al., a relative decrease in frontal flow has been reported following head trauma.<sup>97,98</sup> Following head trauma, absolute cerebral blood flow techniques reveal that head injury often results in an overall reduction in blood flow. A diffuse reduction in cerebral perfusion occurs often in the acute state, as noted by SPECT imaging.<sup>100</sup>

While SPECT has been used to study the physiology of acute brain injury, its use in diagnostic purposes for acute brain injury is limited. However, neurosurgeons are recently recognizing that findings on SPECT can be the most sensitive and unique descriptors of dynamic alterations in brain function due to trauma. When used in conjunction with other imaging modalities, such as MRI, CT, or EEG, brain blood flow imaging by SPECT enhances the ability of the neurosurgeon to determine the posttrauma perfusion status of the brain.<sup>101</sup> On the other hand, issues such as acute subarachnoid hemorrhage and thin subdural hematomas exert little mass effect and do not produce a perfusion defect measurable within the spatial resolution of current SPECT scanners. Also, SPECT may not image larger intraparenchymal lesions due to the dissociation between cerebral blood flow and the reduced metabolic demand in these lesions. There is a relative hyperemia that makes them invisible on SPECT.<sup>5</sup>

Neuroimaging with SPECT using the cerebral blood flow tracer <sup>99</sup>Tc-HMPAO has been used to study acute functional alterations after head injury and the residual abnormalities at 6 months follow-up. Thirty-two patients were studied, and comparison was made between the anatomical abnormalities defined acutely with CT and later on follow-up with MRI. SPECT showed slightly more abnormalities than CT in the acute phase, and 22 of the acute SPECT abnormalities were in regions interpreted as normal on CT scan. Comparison of the intensity of late and early SPECT deficits showed that only 4 early deficits deteriorated, whereas 28 improved. Only 5 of 27 lesions seen on both acute SPECT and CT resolved, compared with 16 of 22 lesions seen on SPECT but not on CT. Regions of abnormally high tracer uptake were detected in the acute stage in five of the patients evaluated by SPECT. However, there were no high focal uptake regions evident on follow-up by SPECT.<sup>102</sup> In another study, 21 consecutive patients admitted to a trauma hospital underwent MRI examination and examination using SPECT. Neurocognitive assessment was made within 5 days of injury. Neurocognitive follow-up assessment was conducted 2 and 6 months after injury, and MRI was repeated after 6 months. Lesion size and brain atrophy were measured on the MRI studies. Fifty-seven percent of patients had abnormal MRI findings, and 61% had abnormal SPECT findings associated with brain atrophy. The association between hypoperfusion seen on acute SPECT and at follow-up after 6 months suggested the possibility of ischemic brain damage. The authors were not able to correlate well between neuroimaging findings and neurocognitive outcome.<sup>103</sup> Other studies have reported serial SPECT scans in the early phase after trauma. These have tended uniformly to demonstrate that the SPECT perfusion deficits may not necessarily increase with time and, indeed, generally reduce in size.<sup>104,105</sup> SPECT studies have demonstrated that blood-brain barrier breakdown around contusions is more frequent after the first 48 h following injury.<sup>106</sup>

In acute studies, SPECT images discrete areas of hyperperfusion adjacent to perfusion deficits, and distant nonfocal changes in patients with less severe head injuries. It is argued that a focal structural lesion occurring as part of a moderate or minor head injury may still be associated with widespread distant functional changes in the brain. These are clearly distinct lesions from the centrifugal sequence of structural changes that occur in severe acceleration-deceleration injuries, in which rotational forces produce damage to deep midline regions and the brain stem.<sup>107</sup> Prelim-

inary studies in Glasgow have shown that the more minor perfusion deficits seen on early scanning, and lesions that appear on SPECT only, are more likely to resolve.<sup>108</sup>

Significant attempts at standardization of SPECT studies in the acute phase are currently under way throughout the world. Some centers are using statistical parametric mapping (SPM) to detect hypoperfusion on <sup>99</sup>Tc-HMPAO SPECT scans. Recent studies have compared acute SPECT findings with those of MRI and found more extensive abnormalities noted by SPECT. In patients who sustain diffuse injuries, follow-up demonstrated an even more pronounced ability to detect lesions with SPECT than MRI. As expected by the anatomical studies of head-injured patients, most acute head injuries involve the frontal and temporal lobes and the anterior cingulate. Blood flow abnormalities persisted at follow-up as long as 366 days after injury, but at a lesser extent than the acute studies. In a number of patients, additional involvement of the thalamus was noted. SPM is thought to have a role in SPECT image interpretation because it allows better visualization than other methods of quantitative analysis of the spatial distribution of abnormalities in diffuse and focal head injury. Blood flow abnormality in the anterofrontal regions was found to be common after head injury.<sup>109</sup>

Other centers are performing anatomic standardization and comparing SPECT imaging with normal templates. This allows automated, operator-independent volume-of-interest (VOI) or voxel-based analysis of whole-brain data. In recent studies, voxel-based analysis was more accurate than SPM analysis. SPM analysis was also significantly less sensitive at thresholds corresponding to low false positive results. Under clinical conditions, classification of brain SPECT studies can be aided greatly by anatomic standardization techniques in reference to normal data.<sup>110</sup>

## SPECT and Neuropsychological Outcome

A few years ago, it was thought that correlation of metabolic information with neuropsychological data might yield new insight into the functional organization of the brain.<sup>111</sup> Newton et al.<sup>112</sup> and Bavetta et al.<sup>113</sup> have demonstrated that relatively simple measures derived from early and late SPECT imaging can show good agreement with outcome. Newton's group used the Glasgow Outcome Scale and showed a fairly high correlation between the number of SPECT lesions and the Glasgow Outcome Scale ( $r = .82$ ). The Glasgow group included detailed neuropsychological testing in an early SPECT study. They were one of the first groups to demonstrate that focal brain lesions found by SPECT often reveal little neuropsychological impairment, whereas diffuse injury shows a high relationship between blood flow abnormalities on SPECT and neuropsychological deficits.<sup>114</sup> [Figure 8.1](#) represents a brain SPECT obtained 2½ years after a severe motor vehicle accident. The patient was unable to cooperate for rehabilitation, and the SPECT was ordered during a behavioral evaluation.

Goldenberg's group<sup>115</sup> and Oder and others<sup>100</sup> have studied the relationship between cognitive and psychosocial problems after trauma using HMPAO SPECT. They administered a neuropsychological battery emphasizing memory and executive functioning. They did not find the expected relationships between memory functions and temporal lobe blood flow, and between executive functions and frontal and thalamic blood flow. There was, however, a general relationship between neuropsychological test performance and blood flow in the frontal and thalamic areas, with a correlation of about 0.5. Oder and others studied 36 very severely brain-injured people. Their median duration of posttraumatic amnesia was about 2 months. Oder's colleagues found the highest correlation ( $r = .6$ ) between frontal lobe blood flow and disinhibited behavior. The lower the flow, the greater the disinhibition. Social isolation was associated with low blood flow in left hemisphere regions, while aggressive behavior was associated with low perfusion in the right hemisphere. These correlations were weak at about  $r = .4$ . These studies have been analyzed together,<sup>5</sup> and they suggest that personality change has a stronger relationship with frontal cerebral blood flow than does cognitive deficit. Low frontal perfusion appears to be specifically related to personality change and psychosocial problems. These results also suggest that blood flow in frontal and thalamic regions may be indicative of the degree of diffuse damage.

## SPECT and Mild Head Injury

Masdeu's group reported that SPECT may be useful in establishing the presence of brain injury in minor head trauma (GCS score > 12).<sup>94</sup> Tikofsky and VanHeertum believe that SPECT techniques are particularly useful in identifying regions of contrecoup injury.<sup>90</sup> These alterations in perfusion suggest impaired neural function, which may account for a patient's clinical presentation when no structural lesions are found with CT or MRI. This finding is of particular importance when evaluating patients with "minor" head trauma, as they may experience only a brief period of unconsciousness and leave the emergency room with no observable neurological impairment. Then they later return with complaints of visual, cognitive, or behavioral changes. Alexander<sup>95</sup> opposes Masdeu et al.<sup>94</sup> and Tikofsky and VanHeertum<sup>90</sup> and argues that functional imaging should not be performed in the immediate postacute state, and that it should be used for patients who demonstrate persistent behavioral, cognitive, or psychiatric symptoms after a reasonable recovery period of approximately 6 months, with or without treatment. Jacobs et al.<sup>116</sup> studied a group of 67 patients, which included 25 who had sustained mild head injuries. All patients who had normal SPECT findings early after injury were asymptomatic at 3 months. However, six of nine patients who had abnormal early SPECT changes after injury had clinical signs and symptoms 3 months later. Jacobs et al.<sup>116</sup> concluded that normal regional cerebral blood flow in the early phase is a predictor of favorable outcome, and that SPECT can provide an objective correlate of the complaints made by patients with minor head injury.

The evidence is that persisting neuropsychological deficits following minor head injury do correlate with abnormal regional cerebral blood flow as detected by SPECT. What is not known is whether the abnormalities are secondary to subtle structural damage to which current structural imaging methods are insensitive, or are a form of functional brain damage, or whether they represent changes in mental state arising from other causes, such as depression. Most experts recommend that interpretation of SPECT images in minor head injury, when findings are unsupported by structural imaging, should be exercised cautiously.<sup>5,117</sup>

## POSITRON EMISSION TOMOGRAPHY

Positron emission tomography (PET) has been the workhorse of functional imaging for many years.<sup>118</sup> The basic principles of PET are based on techniques developed by Kety and others<sup>119</sup> using xenon for the measurement of cerebral blood flow. PET is named from its use of positron-emitting isotopes to image brain functioning. Positron-emitting isotopes are very short lived, and most PET studies in traumatic brain injury use oxygen-15 or fluorine-18 to tag radiotracers. The most common metabolic agent used to study traumatic brain injury is a glucose analog 18-fluorodeoxyglucose (18-FDG). Oxygen-15 is used to manufacture water, which is then injected to measure cerebral blood flow with PET, whereas 18-FDG is used to image metabolism. After radioactive agents are intravenously injected into the subject, the head is positioned within a radiation detector. The radioactive isotope decays within the brain, releasing a positron. The positron travels a short distance and collides with an electron, resulting in the emission of two photons that travel at 180° to each other at the speed of light. Photons are detected at the opposite sides of the head simultaneously, and the location of the emitted positron can thus be calculated.<sup>120</sup> PET findings in brain trauma are represented in [Table 5.4](#).

## PET and the Pathophysiology of Acute Brain Injury

Much is currently being learned regarding PET imaging in brain trauma from rat studies. Recent research has developed microPET, a high-resolution PET scanner that is capable of performing *in vivo* molecular imaging at a resolution sufficient to image major structures in the rat brain. FDG-microPET is quantitative, reproducible, and sensitive to metabolic changes, including a new approach to the longitudinal study of small animal models in brain trauma research.<sup>122</sup> PET scans have recently been used to assess adult rats subjected to a moderate lateral fluid percussion brain injury followed

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**TABLE 5.4**  
**PET and Traumatic Brain Injury**

Uses	Findings
Acute brain injury	Hyperglycolysis occurs regionally and globally following trauma; brief hyperventilation does not cause energy failure or acute ischemia <sup>124,126</sup>
Neuropsychological outcome	O-15 PET-measured blood flow correlates better with prognosis than CT or MRI; frontal-cingulate systems are preferentially injured during closed-head trauma; regional metabolic rates can be an objective marker of neuropsychological sequelae <sup>8,127,128</sup>
Mild head injury	PET useful for arguing general rather than specific neuropsychological dysfunction <sup>90,135,136</sup>

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by survival periods of 2 and 12 h. Studies have noted changes in receptor binding of muscarinic acetylcholine, N-methyl-D-aspartate (NMDA) subtype glutamate, and gamma aminobutyric acid (GABA) type A receptors. After 12 h, a significantly decreased binding potential at receptors sensitive to these neurotransmitters was noted. The altered receptor systems were associated with the development of cellular dysfunction, which was widespread and not limited only to the site of head percussion.<sup>123</sup> The receptor changes were detected by autoradiography and short-lived PET tracers.

PET studies have revealed that cerebral hyperglycolysis is a pathophysiologic response to injury-inducing ionic and neurochemical cascades (see [Chapter 1](#)). Bergsneider and others were the first to demonstrate posttraumatic hyperglycolysis in humans following traumatic brain injury using FDG-PET. Hyperglycolysis in their study was defined as an increase in glucose utilization that measured 2 standard deviations above expected levels. Their findings indicated that by FDG-PET imaging, hyperglycolysis occurred both regionally and globally following severe head injury in humans. The results of their studies directly complement those previously reported in the animal experimental brain injury studies indicating that one can now image a fundamental component of cellular pathophysiology that is characteristic of head injury.<sup>124</sup> While there is emerging evidence of hyperglycolysis following traumatic brain injury, other studies by Bergsneider's group have demonstrated that the level of consciousness as measured by the Glasgow Coma Scale correlates poorly with the global cortical cerebral glucose utilization as determined by FDG-PET. In their studies, cerebral glucose utilization decreased regionally in 88% of brain-injured patients studied. Interestingly, the reduction of cerebral metabolic glucose rates was not highly correlated to the level of consciousness.<sup>125</sup> Bergsneider et al. have continued their studies to analyze the time course of changes in the cerebral metabolic rate of glucose following traumatic brain injury in humans. Their most recent studies reveal that the intermediate metabolic reduction phase begins to resolve approximately 1 month following injury, regardless of injury severity. The dynamic profile of cerebral glucose metabolism that changes following traumatic brain injury is seemingly stereotypic, but it is across a broad range of severity and injury types. Their recent studies cautioned that quantitative FDG-PET cannot be used as a surrogate technique for estimating the degree of global functional recovery following traumatic brain injury. They were not able to correlate the extent of change and neurologic disability assessed by the *Disability Rating Scale* with changes in the rate of change of glucose metabolism.<sup>9</sup>

Neurosurgeons in the past frequently hyperventilated patients following severe traumatic brain injury. There has been some developing concern that this technique could lead to cerebral ischemia. PET studies using oxygen-15 revealed that after severe traumatic brain injury, brief hyperventilation produced large reductions in cerebral blood flow, but there was no evidence of cellular energy failure, even in those regions in which the cerebral blood flow fell below the threshold for energy failure, defined as acute ischemia (below 18 to 20 ml/100 g/min). Neurosurgeons now believe that oxygen metabolism is preserved due to the low baseline metabolic rate in the injured brain and compensatory increases in the oxygen extraction fraction as measured by PET. The reductions in cerebral blood flow following hyperventilation are now thought unlikely to cause further brain injury.<sup>126</sup>

## PET and Neuropsychological Outcome

Recent PET studies have indicated that detection of changes in regional cerebral blood flow measured by oxygen-15 PET correlate better with neurological status and prognosis than abnormalities detected by CT or MRI alone.<sup>127</sup> There is substantial indication that the frontal-cingulate systems are preferentially impaired during closed-head injury. Recent French PET studies suggest a predominant role of prefrontal and cingulate dysfunction in cognitive and behavioral disorders of patients with severe traumatic brain injury. Many of the frontal-cingulate regions appear structurally intact in MRI, wherein PET detects defective activation of the prefrontal-cingulate network.<sup>128,129</sup> Figure 8.7 represents a PET image made during a neuropsychiatric examination. This PET was obtained concurrently with the MRI in Figure 8.6.

If children or adolescents are imaged by PET during the rehabilitation phase following traumatic brain injury, PET may not offer significant data for prediction of outcome. It appears that PET provides no advantage to this prediction compared to contemporaneous CT or MRI.<sup>130</sup> In adult patients who demonstrate posttraumatic anosmia, PET findings strongly suggest that the anosmia is closely related with hypometabolism in the orbitofrontal cortex and the medial prefrontal cortex, as would be expected. The results of PET studies underscore the importance of posttraumatic anosmia as a clinical sign of orbitofrontal damage.<sup>131</sup>

Recent Polish studies have noted that the areas of decreased local cerebral blood flow and reduced local cerebral metabolic glucose rates exceeded those of brain injury demonstrated by CT predominantly in patients with brain posttraumatic cysts. In another group of patients with posttraumatic cerebral atrophy documented by CT, PET demonstrated cortical and subcortical lesions in most cases, providing objective evidence for neurological symptoms.<sup>132</sup> The Kessler Medical Rehabilitation Research and Education Corporation recently reported PET evidence of alterations in specific substrates involved in verbal recall. They imaged individuals who sustained a severe traumatic brain injury (GCS average score = 6.8, years postinjury = 3.18). These PET studies demonstrated changes in the frontoparietal regional cerebral blood flow using oxygen-15. When compared with non-brain-injured controls, the frontal lobe regional cerebral blood flow changes following traumatic brain injury were reduced during free recall of words but enhanced during recognition.<sup>133</sup> A second study at the Kessler Corporation, using oxygen-15 PET and functional magnetic resonance imaging, indicated a prominent role for the frontal lobes in learning and memory functioning, and supported the concept of distributed neural networks for memory-related functions, cognitive loading, and the potential for examining brain reorganization following injury.<sup>134</sup> Mase and others measured regional cerebral blood flow, regional oxygen extraction fraction, and regional metabolic rates of oxygen using positron emission tomography in patients at an average of 9 months after traumatic brain injury. The PET study showed mild decreases of regional cerebral blood flow and regional metabolic rate of oxygen consumption in all patients. However, half the patients showed a frontal type of injury with relative decreases of blood flow and regional metabolic rate of oxygen utilization bilaterally in the frontal cortex, and the other half showed a posterior cerebellar-type injury with relative decreases of blood flow and regional metabolic rate of oxygen utilization in the bilateral occipital cortex and cerebellum. The regional oxygen extraction fraction was normal in all patients. However, the metabolic rate of oxygen seems to be more sensitive for detecting lesions than is regional cerebral blood flow. These Japanese studies have concluded that the evaluation of cerebral blood flow and oxygen metabolism using PET can become an objective assessment of neuropsychological sequelae after diffuse traumatic brain injury.<sup>8</sup>

## PET and Mild Head Injury

Tikofsky and VanHeertum's group noted that both SPECT and PET may be useful techniques in establishing the presence of brain injury in mild head trauma.<sup>90</sup> As noted previously, current research does not show a direct or strong correlation between diffuse perfusion deficits and specific neurobehavioral impairments. Thus, while PET imaging in minor head injury can show the presence

of injury and correlate that with nonspecific cognitive change measured by neuropsychological assessment, currently it is very difficult to show a 1:1 relationship between a specific PET hypometabolic lesion and a specific neuropsychological dysfunction. Therefore, PET is useful for arguing general neuropsychological impairment but, at the present time, probably not specific neuropsychological impairment. In general, 18-FDG deficits of glucose metabolism are shown to be most prominent in midtemporal, anterior cingulate, precuneus, anterior temporal, frontal white matter, and corpus callosum brain regions following traumatic brain injury. These findings do correlate overall with clinical complaints and general neuropsychological impairment. This finding is present even in mild traumatic brain injury following PET imaging.<sup>135</sup> Abnormal PET findings have been reported in a child 4 years after a whiplash injury. Standard EEG was normal, but a PET scan showed evidence of marked hypometabolism in both temporal lobes, and the neuropsychological test findings were consistent with verbal and visual memory deficits within the context of high average intelligence.<sup>136</sup>

## FUNCTIONAL MAGNETIC RESONANCE IMAGING

fMRI couples the spatial resolution of structural MRI with an ability to image areas related to neural activity. It performs this noninvasively, without the use of radiopharmaceutical agents, and without the use of contrast materials. Oxygenated hemoglobin is less paramagnetic and has a greater intensity than deoxygenated hemoglobin on images created with T2-weighted pulse sequences. fMRI uses the blood oxygen level-dependent effect to image changes in neural activity. Although the exact mechanism is not known at this time, it appears that the supply of oxygen is much greater around neurons than what they actually utilize. This results in an increased concentration of oxygenated hemoglobin relative to deoxygenated hemoglobin in areas of neural activity.<sup>120</sup>

fMRI requires no radiation, and the patient can be imaged multiple times. Thus, patients can be imaged during different clinical states and during or after pharmacologic intervention. fMRI is performed in standard, clinically available 1.5-T magnetic resonance scanners, which are widely used today. Theoretically, fMRI can be performed at any site having a modern MRI scanner. In MRI studies using BOLD imaging, detection of very small signal intensity changes can be noted. These are as low as 0 to 3% at 1.5 Tesla and up to 6% at 3.0 Tesla.<sup>10</sup> The increased signal response is a result of localized hemodynamic changes induced by regionally increased neuronal activity during the performance of a defined cognitive task.<sup>137</sup> Table 5.5 summarizes fMRI.

The applications of fMRI to traumatic brain injury have been scanty to date. They do show great promise for the future.<sup>138</sup> Working memory has been measured using fMRI following mild traumatic brain injury at the Dartmouth Medical Center. McAllister's group evaluated 12 mild traumatic brain injury patients within 1 month of their injuries and compared them with 11 healthy control subjects. The control subjects showed bifrontal and biparietal activation in response to a low processing load during a working memory task. There was little increase in activation when the task load was increased. On the other hand, mild traumatic brain injury patients showed some activation during the low processing load task, but a significantly increased activation during the

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**TABLE 5.5**  
**fMRI and Traumatic Brain Injury**

- fMRI uses BOLD effects to image changes in neural activity.<sup>120</sup>
  - Oxygenated hemoglobin is less paramagnetic and more intense on T2-weighted pulse sequences than is deoxygenated hemoglobin.<sup>120</sup>
  - No radiation is used, and the patient can be imaged multiple times.<sup>120</sup>
  - Very small signal intensity changes can be detected (as low as 0–3% at 1.5 T).<sup>10</sup>
  - Local hemodynamic changes can be detected during performance of a defined cognitive task.<sup>137</sup>
-

high load condition; particularly noteworthy was the increase in the right parietal and right dorso-lateral frontal regions.<sup>139</sup> fMRI has also been used to determine motor recovery following a penetrating brain injury into the right capsular region. The patient sustained a left hemiparesis, which resolved fully over several weeks. When fMRI was performed 18 months later, there was no pyramidal weakness present, but there was a mild hemidystonia and a sensory disturbance. fMRI revealed contralateral primary and supplementary motor cortex activation during the tapping of each hand. Smaller ipsilateral primary motor areas were activated by the recovered hand only. This fMRI study did not suggest any substantial reorganization of the motor cortex. The initial deficit was thought to be caused mostly by reversible local factors, including edema and mass effect.<sup>140</sup>

## MAGNETIC RESONANCE SPECTROSCOPY

Spectroscopy is performed in the same scanning equipment as that used for structural MRI. However, the scanning parameters are altered and the signal that returns represents chemical entities from brain areas. A particular atom in a magnetic field has a characteristic response based upon the number and nature of its subatomic particles. Spectra can be obtained from these molecules and plotted; they then represent characteristics for nuclei within certain chemical structures. In magnetic resonance spectroscopy (MRS), typically spectra can be obtained from a number of different nuclei, including hydrogen-1, carbon-13, sodium-23, lithium-7, and phosphorus-31. MRS is being widely used in psychiatric investigation, but this aspect will not be discussed in this text. However, most of the studies in psychiatry are focusing upon hydrogen-1 and phosphorus-31 nuclei.<sup>120</sup>

MRS is noninvasive and easily repeatable. It can provide information about cellular membrane function and various metabolic processes. It has been used successfully to image abnormalities of brain trauma, tumor growth, and ischemia. However, it has limited spatial resolution, although modern scanning techniques are improving this. If molecules are present in very low concentrations, for instance, at various neurotransmitter receptor sites, PET is a superior imaging modality at this time because it uses radiolabeled ligands.<sup>120</sup>

MRS has three fundamental concepts: (1) nuclear magnetism, (2) chemical shifts, and (3) resonance in which the frequency of an excitatory radio frequency pulse is matched to the frequency at which nuclei are wobbling about the axis of the externally applied magnetic field. Various brain substances can be used as markers of tissue damage. Choline and other lipids are markers of myelin breakdown. Creatine intensity may be used as a constant or internal standard to which the resonance intensities of other metabolites are normalized. However, arguably the most important signal for the assessment of traumatic brain pathology is the intensity of N-acetylaspartate. This is a surrogate marker of neuronal integrity. Lactate can be used as a marker of anaerobic metabolism.<sup>141</sup>

MRS has been used and validated in the measurement of the syndrome of inappropriate antidiuretic hormone (ADH) after head trauma. It also has found use in evaluating comatose patients following traumatic brain injury producing a hyperosmolar state. However, with regard to neuropsychiatric evaluation, its most important use has been in the delineation of diffuse axonal injury. In this case, the N-acetylaspartate pattern is prominent and consistent with diffuse axonal injury. In fact, MRS has been used to predict outcome following diffuse axonal injury. Moreover, MRS has shown functional utility in the ability to predict neurological outcome in patients who are comatose following traumatic brain injury. Generally, the level of decrease of N-acetylaspartate will predict the level of neuronal loss, and thus the likelihood of poor outcome following substantial traumatic brain injury.<sup>142</sup>

By using hydrogen-1, proton MRS is becoming useful in identifying patients with neuronal injuries after traumatic brain injuries. MRS can quantify damage after brain injury using the magnetization transfer ratio (MTR). This correlates with N-acetylaspartate levels and is a sensitive indicator of the neuronal damage that results in worst outcome brain injury.<sup>143</sup> Another study has used MRS to correspond to neuropsychological function following traumatic brain injury. Patients with traumatic brain injuries display reduced N-acetylaspartate in white matter and elevated choline

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**TABLE 5.6**  
**MRS and Traumatic Brain Injury**

Marker	Functional Disturbance
N-acetylaspartate	Neuronal integrity (e.g., diffuse axonal injury) <sup>143</sup> ; poor outcome <sup>142</sup>
Choline	Gray matter inflammation <sup>143</sup> ; myelin breakdown <sup>144</sup>
Lactate	Anaerobic metabolism <sup>141</sup>
Creatine	A constant or internal <sup>141</sup> standard

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in gray matter, which is consistent with neuronal injury and postinjurious inflammation, respectively. Behavioral dysfunction measured neuropsychologically correlates positively with the injury predictions associated with N-acetylaspartate and choline.<sup>144</sup> Proton MRS has been noted to correlate with outcome in MRS studies performed in the U.K. as well.<sup>145</sup> Accordingly, MRS may be useful to monitor cellular response to therapeutic interventions in traumatic brain injury.<sup>146</sup> [Table 5.6](#) outlines MRS markers.

### ELECTROENCEPHALOGRAPHY

EEG was the mainstay of neurological laboratory testing prior to the onset of CT. Williams and Denny-Brown<sup>147</sup> found that in cats subjected to experimental head injuries, there was an almost immediate reduction in amplitude of the EEG. This sometimes amounted to complete attenuation of the rhythm. After some 10 to 80 sec, delta activity appeared and remained evident for 10 to 160 sec, after which the record returned to its premorbid state. Walker et al.<sup>148</sup> confirmed these findings and noted additionally that the period of suppression was preceded by a generalized high-voltage discharge. The EEG changes seen after head injuries in humans are extremely varied, and this is due to three main facts: (1) The general diagnostic label of head injury or traumatic brain injury encompasses a number of different types of lesions, the character, extent, and distribution of which vary widely from patient to patient; (2) A head injury gives rise to an illness that is a dynamic process with an evolution and devolution that varies greatly in form and timing in different patients; (3) Certain features of traumatic brain injury, notably alterations of consciousness, may in their own right produce EEG abnormalities.<sup>149</sup> The EEG frequencies of the human are generally described as alpha waves (8 to 11 Hz), beta waves (>13 Hz), delta waves (1 to 3 Hz), theta waves (4 to 7 Hz), and sleep spindles (12 to 14 Hz), seen in drowsiness or stage 2 sleep. Immediately after any head injury sufficiently severe to cause a loss of consciousness, some degree of generalized reduction of the amplitude of the waveform occurs. However, in the majority of cases, this phase is over by the time the EEG recording is carried out clinically following the head injury. Therefore, attenuation is seen rarely, except in the most severe brain trauma-producing coma. Attenuation appearing after an interval of several days or after surgical intervention is not uncommon and coincides with returning consciousness and a period of restlessness and confused behavior. This variety has a good prognosis. Complete and persistent attenuation carries a bad prognosis and usually is accompanied by deep coma and, in many cases, death<sup>149</sup> (see [Table 5.7](#)).

Following the initial attenuation by trauma, the EEG demonstrates disorganized slow activity, and frequently no alpha rhythm is detectable. In the less severe injuries, the basic frequency may be 7 to 8 Hz and return to normal over a period of hours or days. In the more severe injuries, the basic background frequency slows to 4 to 6 Hz. The rate at which this occurs is prognostic. The outlook is poor when it occurs within 48 h and better if it takes place more slowly.<sup>150</sup>

From a neuropsychiatric perspective, in many cases, even those following severe head injury, the EEG may return to normal. Therefore, in most neuropsychiatric examinations, the EEG rarely contributes to the examination unless posttraumatic seizures are an issue. However, an abnormal EEG may occur when neurological or psychiatric abnormalities persist. If substantial behavioral



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**TABLE 5.7**  
**EEG and Traumatic Brain Injury**

- EEG is generally normal at the time of most neuropsychiatric examinations.<sup>148</sup>
  - In the acute stage, EEG reveals a reduction of waveform amplitude following a loss of consciousness.<sup>12</sup>
  - Attenuation may appear for several days following injury.<sup>149</sup>
  - Persistent attenuation carries a poor prognosis.<sup>149,150</sup>
  - Subclinical seizures in the ICU are very common.<sup>153–155</sup>
- 

problems are present, the existence of a normal EEG indicates that the disabilities have a poor prognosis, for it implies the presence of areas of irreversible destruction of brain tissue that are incapable of giving rise to electrical activity and, therefore, of modifying the normal EEG produced by intact regions of the brain. Such a situation in which a normal EEG indicates a poor prognosis is sometimes referred to as Williams' paradox.<sup>151</sup> Thus, in legal cases, when lawyers wave in front of the jury the normal EEG of a person who sustained a severe brain injury, that normal EEG may in fact indicate severe prognosis if other poor prognostic markers are present.

The EEGs of children are notoriously labile, and the effects of head injuries often are more dramatic than the clinical state would warrant. The changes are similar in character to those described earlier for adults, but they tend to be more severe and more widespread. The amplitude of the waveform is higher than it is in adults. Although occasionally very marked foci may disappear with remarkable speed, resolution of the abnormalities after severe injury usually takes considerably longer than it does in an adult. In brain-injured infants, hypsarrhythmia may occur. This pattern is characterized by generalized continuous slow activity with an amplitude higher than 300  $\mu$ V and the appearance of multiregional spikes or sharp waves over both hemispheres. This is considered to be definitely epileptogenic.<sup>152</sup>

As noted previously in this text (see [Chapter 2](#)), monitoring of EEG within the neurosurgical unit, immediately following brain injury, has dramatically revealed that subclinical seizures are far more frequent immediately after brain injury than previously recognized. In fact, many of these are status epilepticus.<sup>12,153–155</sup> Seizures occur in more than one in five patients during the first week after moderate to severe brain injury and may play a role in the secondary injuries sustained by other pathological conditions following traumatic brain injury.<sup>156</sup> Thus, the neuropsychiatric examiner should review prior EEG studies that may have occurred during the acute phase of the traumatic brain injury. In most instances, the neuropsychiatric examination will not utilize EEG monitoring unless concurrent posttraumatic seizure disorders are present in the patient at the time of examination.

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# 6 Standardized Neurocognitive Assessment of Traumatic Brain Injury

## INTRODUCTION

Generally, the complete neuropsychiatric assessment of traumatic brain injury should contain a screening neuropsychiatric mental status examination, a screening neurological examination, appropriate brain imaging, and also a standardized neurocognitive and neurobehavioral assessment. The nature of the injury and the needs of the patient for treatment planning will dictate how extensive and intensive the examination should be. The first structured mental status examination was introduced in 1918 by Adolf Meyer.<sup>1</sup> While this procedure became the *sine qua non* for the training of American psychiatrists for more than 50 years, it was not standardized. In other words, it was not empirically tested and it contained no precise administration rules or scoring rules. It required extensive narrative descriptions of the patient's behavior and retained substantial subjectivity in recording the results of evaluation. That level of qualitative examination is insufficient, even performed by the most expert psychiatrist, for measuring cognitive changes following traumatic brain injury. Moreover, Lord Kelvin aptly stated the importance of measurement:

When you can measure what you are speaking about, and express it in numbers, you know something about it; but when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meager and unsatisfactory kind: it may be the beginning of knowledge, but you have scarcely in your thoughts, advanced to the stage of science.<sup>2</sup>

In medical settings of ordinary daily practice, the use of brief structured mental status examinations as screening tools for the detection of cognitive impairment is justified; however, while they may be helpful for examination, they are not sufficient in scope or precision to quantify cognitive changes following traumatic brain injury. [Table 6.1](#) outlines the clinical value of these examinations, but it also points out their substantial weaknesses. This section will provide the examiner performing a neuropsychiatric assessment with a guide for obtaining useful cognitive measurements following traumatic brain injury.

It is expected that within a quality neuropsychiatric examination of brain injury, the practitioner will consult with, and generally use, the services of a psychologist or neuropsychologist skilled in the assessment of traumatic brain injury. Information in this chapter is not an exhaustive evaluation of neuropsychological methods, nor is it intended to be. It has a twofold purpose within the overall mission of this text: (1) to acquaint the neuropsychiatric examiner with an overview of the available neuropsychological methodology for performing an adequate assessment of neurocognitive dysfunction following brain injury, and (2) to provide neuroanatomical and neuroimaging bases for the various neuropsychological domains of human cognitive function that are currently sufficiently studied to allow description.

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**TABLE 6.1**  
**Strengths and Weaknesses of Brief, Structured Mental**  
**Status Examinations**

**Strengths**

- They are brief and nondemanding for the patient.
- They reveal little practice effect.
- They require little formal training for their use.
- Physicians find them familiar because they derive from traditional exams.
- Uniformity is present in administration and scoring.
- Quantified results allow comparisons over time.

**Weaknesses**

- The questions are easy to answer, thus producing high false-negative rates.
  - Low intelligence, race, and old age lead to high false-positive rates.
  - They differentiate organic and functional disturbances poorly.
  - They differentiate acute from chronic organicity poorly.
- 

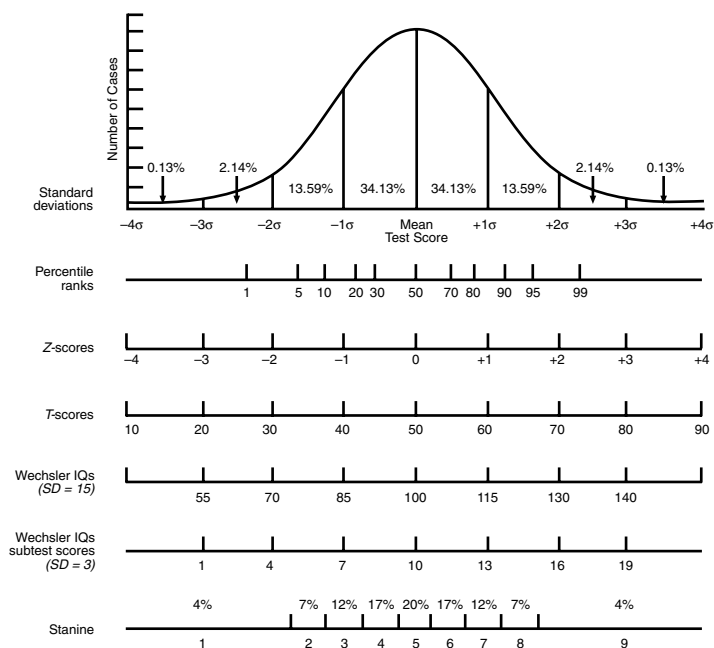
Recently, psychological testing in psychological assessment was exhaustively reviewed by respected research and clinical psychologists.<sup>70</sup> They reviewed data from more than 125 meta-analyses on test validity, while 800 samplings examined multimethod assessment, and came to four general conclusions regarding psychological testing within psychological assessment: (1) Psychological test validity is strong and compelling; (2) Psychological test validity is comparable to medical test validity; (3) Distinct assessment methods provide unique sources of information; (4) Clinicians who rely exclusively on interviews are prone to incomplete understandings. With regard to neuropsychological testing, Prigatano views neuropsychological tests as essentially questions or tasks presented to a person with the intent of revealing something about the nature of higher cerebral functions. Typically, the questions or tasks are administered in a standardized manner so that reliable and valid conclusions can be made regarding the patient's functioning.<sup>71</sup>

## **BASIC STATISTICS OF PSYCHOLOGICAL TESTING**

Many physicians have a poor understanding of the fundamental mathematical principles involved in the analysis and numerical representation of psychological measurements. A simple but fundamental understanding of these principles will remove much of the "aura" of psychological testing and help the examiner understand the simple logic behind psychological measurement as has been developed by our psychological colleagues. [Table 6.2](#) outlines common definitions used within the language of psychological testing. Upon review of [Figure 6.1](#), it can be seen that certain probabilities exist within the normal distribution. For instance, approximately 68% (68.26%) of a normally distributed population lies between  $\pm 1$  standard deviation (SD) of the mean of that distribution. If one were reviewing *Wechsler* IQ scores, it can be seen that 68% of the normally distributed population would have an IQ that lies between the standard scores of 85 and 115 ( $\pm 1$  SD; SD = 15). Also by reviewing [Figure 6.1](#), it can be noticed that a deviation IQ of 130 on the *Wechsler-III* corresponds to a T-score of 70 or a percentile of approximately 97. The reader is cautioned that the data in [Figure 6.1](#) cannot be used to equate scores on one test to scores on another. For instance, a T-score of 70 on scale 2 of the *Minnesota Multiphasic Personality Inventory* (MMPI) means one thing, whereas a deviation full-scale IQ of 130 on the *Wechsler Adult Intelligence Scale-III* (WAIS-III) means another. Both scores on these tests are 2 SDs above their respective group means, but they do not represent "equal" standings because the scores were obtained from different samples within the individual normative data for the tests.<sup>3</sup> However, the examiner clearly can use the data in [Figure 6.1](#) to compare the same test to itself. For instance, if within the context of a brain injury evaluation

**TABLE 6.2**  
**Glossary of Psychological Testing Terms**

Term	Meaning
Deviation IQs	Standard IQ scores having a mean of 100 and a standard deviation of 15 (e.g., WAIS-III IQ scores)
Ecological validity	Predictive relationship between neuropsychological test performance and real world function (e.g., Does an IQ test predict driving ability?)
Mean	The arithmetic average of a group of numerical data or scores
Median	The exact midpoint of a group of numerical data or scores
Percentile	A point on a distribution at or below which there is a given percentage of individuals
Practice effect	Increases in test performance resulting from having practiced on preceding tests (e.g., If a woman takes the WAIS-III in March, will her verbal IQ increase slightly if she retakes the test the following May?)
Reliability	A special type of correlation that measures consistency of observations or scores (e.g., Will a person produce the same verbal IQ on the WAIS-III if it is administered again 9 months later?)
SAT scores	Standard scores having a mean of 500 and a standard deviation of 100
Standard deviation	A measure of the extent to which scores cluster around the mean
Standardization	Uniformity of procedure in administering and scoring the test
Standard scores	Scores expressed in standard deviation units
Stanine score	Divides the normal curve into 9 equal units, with a mean of 5 and SD of 2; each interval is numbered 1 to 9 (e.g., a verbal IQ of 100 would lie within a stanine score of 5)
T-scores	Standard scores having a mean of 50 and a standard deviation of 10 (e.g., MMPI-2 scores)
Validity	The extent to which measurements are useful in making decisions relevant for a given purpose (e.g., Does the WAIS-III validly measure verbal IQ?)
Z-score	The number of standard deviation units that a particular score is above or below the mean of the distribution



**FIGURE 6.1** Relationship of the normal curve to various types of standard scores.

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**TABLE 6.3**  
**Various Methods Used for Neuropsychological Assessment**

- Batteries for general use (e.g., Halstead–Reitan Test Battery, Luria–Nebraska Neuropsychological Battery, and NEPSY)<sup>9,10,205</sup>
  - Analytical approach (a flexible San Diego Neuropsychological Test Battery and eclectic use of tests of intelligence, visual perception, semantics, literacy, language, event memory, reasoning, and behavior)<sup>6,22</sup>
  - The Boston process approach<sup>7</sup>
  - The Iowa–Benton school of neuropsychological assessment<sup>8</sup>
  - The Lezak approach<sup>3</sup>
- 

a man produces a verbal IQ of 117 on the WAIS-III for his rehabilitation psychologist in August, but a verbal IQ of 94 when he is examined by a neuropsychologist the following November, clearly an explanation for this difference must be sought. One would not expect this difference by chance alone. In fact, if the reader refers to [Table 6.2](#), it can be seen that a practice effect would be expected, and one would expect the neuropsychologist to have found a slightly higher verbal IQ in most instances, unless there has been brain function deterioration from disease, intercurrent psychiatric illness, medications, poor performance, faking, or other factors. The same can be said for an individual producing a T-score of 62 on the Depression Scale of the MMPI-2 (scale 2) and later producing a T-score of 115 on the same MMPI-2 scale when examined by a psychiatrist 6 weeks later. Clearly, this difference must be explained, and obviously it is not expected unless there has been an interval change in the person’s mood between the two testings, or as we shall see in [Chapters 7 and 9](#), the person may be symptom magnifying or faking at the second examination. The use of standardized test data is a powerful tool for making intertest comparisons over a time interval. For a more precise analysis of the statistics of psychological testing, refer to comprehensive texts.<sup>3-5</sup>

## ADULT NEUROCOGNITIVE ASSESSMENT

No unitary method is available for neuropsychological assessment of brain-injured patients. [Table 6.3](#) describes philosophical and methodological approaches to neuropsychological testing. The neuropsychiatric examiner may find these various approaches confusing. It is intriguing that while psychologists use individual standardized tests, their approach to a neuropsychological testing situation often is not standardized. Unlike in the practice of neurosurgery, neurology, or psychiatry, where the clinical examination is essentially the same whether performed in California, New York, or Kentucky, the neuropsychological examination may vary tremendously depending upon the training, orientation, and philosophical approach of the individual neuropsychological examiner. Thus, when the physician uses a neuropsychologist to assist in a cognitive examination of an adult or a child, *caveat emptor*. It is incumbent upon the medical examiner of a brain-injured person who uses neuropsychological test data to be highly aware of the training, background, and skills of the psychologist or neuropsychologist upon whom the physician intends to rely. This is by no means an attempt to cast aspersions on our psychology colleagues; it is just the nature of the beast. A competent full neuropsychiatric assessment of a traumatically brain-injured patient cannot be completed without also using neuropsychological test data in that assessment. Certain tests will be highlighted in this chapter to facilitate examples of neuropsychological cognitive testing methods. These will be further analyzed medically in [Chapter 8](#). Referred to excellent reviews of the various neuropsychological approaches to examination for further information.<sup>3,6-12</sup>

## MEASURING COGNITIVE DISTORTION

Two basic methods are used to distort conscious effort during a brain injury evaluation. The first is by cognitive distortion, wherein the individual slows down during timed portions of neuropsych-

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**TABLE 6.4**  
**Tests Useful for Measuring Neuropsychological Effort**

**Tests Based on Binomial Probability**

- Miller Forensic Assessment of Symptoms Test (M-FAST)
- Letter Memory Test (LMT)
- Portland Digit Recognition Test (PDRT)
- Test of Memory Malingering (TOMM)
- Victoria Symptom Validity Test (VSVT)

**Tests Not Based on Binomial Probability**

- Dot Counting: Ungrouped Dots
  - Dot Counting: Grouped and Ungrouped Dots
  - Rey 15-Item Test
- 

chological testing or produces false responses to memory tests or other measures of cognitive function. The second major way to distort outcome in a neuropsychological assessment is by psychological means. That is, the person falsely reports or exaggerates symptoms of depression, anxiety, or other psychiatric symptoms to magnify or fake the intensity of psychological reporting. The issues of psychological distortion will be covered more fully in [Chapters 7 and 9](#); this chapter focuses upon cognitive distortion only.

Neuropsychological testing is particularly vulnerable to poor effort on the part of the examinee, as an individual's test performance depends on his cooperation or motivation to produce optimal effort. One can never do better than his maximal ability, but an individual can certainly do worse than his maximal ability. Thus, it is incumbent upon the psychological examiner to determine the level of cognitive effort at the time the individual is tested.<sup>12</sup> [Table 6.4](#) lists suggested tests that may be used to test effort at the time of a neuropsychological assessment. The fundamental property involved in all tests of cognitive malingering or cognitive effort is that the tests must be easy for even a brain-injured person to pass. If this were not so, one could not distinguish whether a person with mild Alzheimer's disease, brain injury, or mild mental retardation was providing adequate effort during psychological examination. The tests in [Table 6.4](#) are grouped according to whether or not they are based on binomial probability theory. The power of more recent tests developed to measure cognitive effort is that they are in fact based on a forced-choice procedure, which pushes an individual into one of two statistically measured groups. These tests are based on a simple proposition: If one asks a person to make choices between two alternatives, and if a large number of choices are offered, the responses will statistically sort into equally represented populations. For instance, within basic probability theory, it is well known that if one flips a quarter into the air 100 times and allows it to land randomly on the floor, approximately 50 heads and 50 tails will appear. This two-alternative task represents the purest form of the binomial distribution. Thus, a test of cognitive effort is designed so that the person is "forced" to choose between two alternatives, the correct one and the incorrect one. If she properly chooses the correct one most times, her responses will exceed chance (50% probability) by a considerable degree. If, on the other hand, the individual deliberately chooses wrong answers, her responses will dramatically fall below chance levels. Those truly confused or damaged individuals who cannot make a choice between "correct" and "wrong" will produce a random response due to guessing, and they will approximate 50% correct answers and 50% wrong answers. Three tests useful for measuring neuropsychological effort are specifically examined next.

Tests not based on the binomial probability theory have a long history of use in neuropsychology. The dot-counting tests were first proposed in 1941, and the *Rey 15-Item Test* was later proposed in 1964.<sup>13,14</sup> Of these, the most widely used today by psychologists is the Rey 15-Item Test. This is performed with a card that contains 15 visual items. However, there are really only nine items,

as the card consists of A, B, C and a, b, c; and 1, 2, 3 and I, II, III. The last three items are a square, triangle, and circle. Thus, in reality, the person looks at the card and has to remember only nine items, as two sets are repeated. There is some argument as to what the cutoff score for an abnormal response is. Some define the cutoff score as low as 7, while others define it as low as 9. Several investigators have reported that this test lacks sensitivity in identifying malingerers or those providing poor effort. Its efficacy to detect feigned memory impairment appears to be limited.<sup>15</sup> No assessment of effort or malingering should be based solely on this test.<sup>16</sup> The dot-counting measures have been found to have a 40% false negative rate, and thus, their use is no longer recommended.<sup>17</sup>

### **Portland Digit Recognition Test**

The *Portland Digit Recognition Test* (PDRT) is a forced-choice test that is an outgrowth of earlier Hiscock and Hiscock procedures that required subjects to identify after a brief delay which of two five-digit numbers shown on a card was the same as a number seen on a prior card.<sup>18</sup> The PDRT consists of a total of 72 items of digit recognition using an auditory stimulus presentation. Five-digit numbers are orally presented at the rate of one digit per second by the examiner. Following presentation, the subject counts backward aloud until interrupted with a 3-by-5-in. card containing one distracter number (the false number) and the correct five-digit number. The brilliance of this test is its simplicity. The distracter number is off by only one digit in either the first or last digit. Thus, the person being examined can quickly scan the cards and determine the correct from the noncorrect response. The first 18 trials include 5 sec of counting backward from 20 before the second card is shown. The second block of 18 trials involves counting backward from 50 for 15 sec, and the third and fourth blocks of 18 trials both involve counting backward from 100 for 30 sec. Although 72 trials are conducted, there are only 18 different correct target items, and thus, 18 items are administered four times. The target items are no different for counting backward from 100 for 30 sec than the targets for counting backward from 20. Patients are more likely to “fake bad” when the activity interval increases.<sup>19</sup> For obvious reasons, statistically accurate cutoff scores on tests measuring malingering or effort will not be given in this text.

### **Test of Memory Malingering**

The *Test of Memory Malingering* (TOMM) is used for discriminating between memory-impaired persons and those who are either malingering or providing poor effort for other reasons. The TOMM is a 50-item recognition test that includes two learning trials and a retention trial. During the two learning trials, the patient is shown 50 line drawings (target pictures) of common objects for 3 sec each, given at 1-sec intervals. The patient is then shown 50 panels to recognize, one at a time. Each panel contains one of the previously presented target pictures and a new picture (a distracter). The patient is required to select the correct picture (i.e., the picture shown during the learning trial). The same procedure is used on the optional retention trial, except target pictures are not readministered.

To assess effort or malingering, the learning trials alone are usually sufficient. Use of the retention trial (which is optional) adds only a few minutes to the test time and helps corroborate the results. It takes about 15 min to administer the two learning trials. The power of this test lies in the impression to the patient that it is much more difficult than it really is. By administering a large number of visual stimuli, the test leads malingerers to believe that it will be difficult for people with genuine memory impairments, and thus, they intentionally perform poorly. The other major power of TOMM is that, while it is sensitive to malingering, it is insensitive to a person with true neurological impairment. Almost all individuals with neurological impairments have a remarkably high capacity for storing and retrieving simple pictures of common everyday objects. The validation data of the TOMM include head-injured subjects.<sup>20</sup>

## Victoria Symptom Validity Test

The *Victoria Symptom Validity Test* (VSVT) includes a total of 48 items, presented in three blocks of 16 items each. During each block of 16 items, there is a study trial and a recognition trial. This test is administered visually by computer. During the study trial, a single five-digit number is presented on the screen for 5 sec. Following the presentation of this number, there is a retention time interval during which the patient views a blank computer screen. This interval is then followed by the recognition trial in which the correct study number is shown and a five-digit distracter number is displayed as well. The patient is asked to choose the number he saw in the study trial. In the second block of 16 items, the retention interval is increased to 10 sec, and in the third block, the retention interval is increased to 15 sec.

Much of the power of this test to detect poor effort or malingering lies in the standard instructions. Patients are told that they are “taking a test of memory that requires concentration,” and that “people with memory problems often find this test to be difficult.” Instructions indicating that the patient may find the items becoming more difficult are given to minimize deception. Research has found that a majority of patients with real memory problems did not make significantly more errors when the retention interval was increased.<sup>21</sup>

Within each trial or block, items are given that appear to be either “easy” or “difficult.” For easy items, the study numbers and the distracters share no common digits (unlike the PDRT). Thus, recognition of any one of the digits in the five digits will allow the patient to make a correct choice. For the difficult items, the distracter is identical to the study number with the exception that second and third, or third and fourth digits have been transposed. To choose the correct answer on the difficult items, the patient must recall the order of the middle digits. Recognition of the first or last digit will not aid in choosing the right answer. All three blocks contain an equal number of easy and difficult items. Like the PDRT and the TOMM, a person providing poor effort will perform significantly below chance, whereas a person providing good effort will perform significantly above chance levels.

## ESTABLISHING A PREINJURY COGNITIVE BASELINE

Rarely, when examining patients who have sustained traumatic brain injury, does the practitioner have premorbid or preinjury test data in order to draw comparisons between preinjury cognitive performance and postinjury cognitive performance. Deficits can be assessed directly when there are normative comparison standards for the ability in question. In indirect measurement, the examiner compares the present performance with an estimate of the patient’s original ability level.<sup>3</sup> These estimates may be from a variety of sources, and for the most part, they are based on tests of verbal or reading skill or by using demographic data. [Table 6.5](#) lists tests that have been found useful for estimating the premorbid ability of a traumatically brain-injured patient.

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**TABLE 6.5**  
**Tests for Estimating Preinjury Mental Abilities**

Demographic tests:	Barona Index The Oklahoma estimate Wilson’s formula
Reading-based tests:	National Adult Reading Test North American Adult Reading Test Wechsler Test of Adult Reading Wide Range Achievement Test

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Wilson and colleagues devised a formula using the demographic variables of age, sex, race, education, and occupation.<sup>23</sup> However, this formula has been found weak and will predict only two thirds of premorbid Wechsler IQ scores within a 10-point error range. Barona and colleagues elaborated on Wilson et al.'s work and included variables of geographic region, urban–rural residence, and the handedness of the person into the estimation formula first proposed by Barona et al.<sup>24</sup> They devised three formulae for predicting each of the Wechsler IQ scores directly from these data. They caution that, where the premorbid full-scale IQ was above 120 or below 69, serious under- or overestimation errors may occur. Some studies have claimed that at best, the Wilson and Barona estimates misclassify more than half of patients, which of course is no better than a chance level prediction. Krull et al. have used demographic variables similar to those of Wilson and Barona, but they have combined these with either the vocabulary or picture completion test scores from the Wechsler IQ scales to estimate premorbid IQ.<sup>25</sup>

The overall estimate of a person's premorbid ability can rely on measures using tests such as those in Table 6.5. However, the estimates should take into account as much information as possible about the patient. For instance, school performance data, school psychology data, armed forces entrance scores, SAT scores, ACT scores, and other such similar measures made premorbidly will supply data for a direct measure of deficit in an adult, as these are based upon standards that can be compared with test data developed by neuropsychological assessment after the injury.

Vocabulary and related verbal skill scores may provide the best estimates of the general premorbid cognitive ability level if preinjury measures are not available. However, the vocabulary subscale on the Wechsler IQ tests requires the patient to produce oral definitions. Therefore, this test is more vulnerable to brain damage than verbal tests that can be answered in a word or two, or that call on practical experience or recognition, such as in the reading tests noted below. Moreover, if the patient's brain injury is preferential to the dominant cerebral hemisphere, vocabulary ability may be impaired as well. In an attempt to improve upon vocabulary-based methods, the use of reading scores derived by the *Wide Range Achievement Test (WRAT)*, *National Adult Reading Test (NART)*, and *Wechsler Test of Adult Reading (WTAR)* have been used. The original NART was first standardized on British subjects, but there is now a North American version available (NAART). The very recent Wechsler Test of Adult Reading is probably superior to both the WRAT and NART because, similar to the Oklahoma estimate, the WTAR is based upon reading measures and demographics and also has been standardized on traumatic brain injury patients. The classification of both preinjury and postinjury ability levels can be done in many fashions. Lezak<sup>3</sup> argues that the classification of ability levels should be based on Z-scores or percentiles as one way to avoid the many difficulties inherent in test score reporting. Table 6.6 lists classification ability levels based upon Z-scores and percentile ranges (see Figure 6.1 for further analysis).

**TABLE 6.6**  
**Classification of Ability Levels**

Classification	Z-Score	Percent under Normal Curve	Lower Limit of Percentile
Very superior	+2.0 and greater	2.2	98
Superior	+1.3 to 2.0	6.7	91
High average	+0.6 to 1.3	16.1	75
Average	±0.6	50.0	25
Low average	-0.6 to -1.3	16.1	9
Borderline	-1.3 to -2.0	6.7	2
Retarded	-2.0 and less	2.2	—



For many years, it was felt that the vocabulary and picture completion test scores from the Wechsler IQ scales would hold relatively unchanged following injury for most brain-damaged persons. Both of these tests involve verbal skills. Some have claimed that the average of these two scores, or the highest score of the two, should provide the standard against which other Wechsler test scores are compared to determine within-test changes. Other experts in the field have claimed that the information in the picture completion subtest of the Wechsler scales was resilient to the effects of brain injury, and thus could be used as a standard for assessing premorbid ability. However, further studies in psychology have found that these assumptions do not hold.<sup>3</sup>

### **National Adult Reading Test**

The National Adult Reading Test (NART) has been restandardized against the *Wechsler Adult Intelligence Scale-Revised* (WAIS-R).<sup>26</sup> This restandardization allows the reading score taken from the NART to be used to predict the WAIS-R full-scale, verbal, and performance IQs, which are predicted from the number of errors made on the NART. This allows the estimation of a predicted full-scale IQ within the interval of 69 to 131. If a person has a language disturbance following a brain injury, the NART may underestimate premorbid ability. Therefore, patients who are aphasic, dyslexic, or who have articulatory or visual acuity defects probably should not be screened using this instrument.<sup>27</sup> Moreover, the standardization sample did not include subjects of more than 70 years age.

### **Reading Subtest of the Wide Range Achievement Test-III**

This test has been standardized on thousands of persons across the U.S. in nearly half of the 48 continental states and also Alaska. The data were compiled using a stratified sampling of nearly 5000 individuals. This test can be used to measure reading recognition levels in persons aged 5 to 75 years.<sup>28</sup> The test begins with letter reading and recognition. The word pronunciation format of this test is identical to that of the NART. The WRAT-III, on the other hand, was developed to evaluate educational achievement rather than assess premorbid ability. However, it can be used for assessing premorbid ability in predicting verbal IQ on the Wechsler scales. This instrument has not been used significantly in neuropsychological research protocols; nor has it been used as greatly in neuropsychological test protocols as the NART.<sup>27</sup>

### **Wechsler Test of Adult Reading**

The Wechsler Test of Adult Reading (WTAR) was developed specifically to provide clinicians with an assessment tool for estimating premorbid intellectual functioning of adults ages 16 to 89. It has been developed and conormed with the WAIS-III and the *Wechsler Memory Scale-III* (WMS-III). This codevelopment of the WTAR with the WAIS-III and the WMS-III provided data for direct comparison between predicted and actual intelligence and memory function of a large sample of functionally normal adults.<sup>29</sup> With regard to traumatic brain injury, this test has been specifically evaluated in persons who have sustained traumatic brain injury, both adults and adolescents. It was found that WTAR performance by the brain-injured group did not differ significantly from that of the control group. Thus, the WTAR appears capable of predicting premorbid intellectual test scores and memory scores based on the Wechsler IQ and memory scales.

The WTAR is probably the most powerful test available at this time for estimation of premorbid intellectual and memory abilities in traumatically brain-injured persons. It has increased power in this ability because the predictions are based not only upon reading scores, but the WTAR also specifically includes a combination of WTAR reading scores and a demographics prediction of WAIS-III and WMS-III scores. Thus, the WTAR builds upon the goal of the Oklahoma premorbid test and has expanded that format.

## Practice Effects from Cognitive Retesting

Practice effects come about by repeated psychological examination. These differences have been studied in normal and in brain-injured patients. The general rule for practice effects is that tests having a large speed component, requiring an unfamiliar or infrequently practiced mode of response, or having a single solution, particularly if it can be easily conceptualized once it is attained, are much more likely to show significant practice effects than tests that do not have these features.<sup>30</sup> Tests that involve learning tend to show large practice effects as well as do tests such as the *Grooved Pegboard*, which contains unfamiliar motor responses.<sup>31</sup> In traumatic brain injury, the *Block Design Test* of the WAIS-III is difficult to conceptualize, and patients are unlikely to improve with practice alone. In tests of this nature, improvements attributable to practice tend to be minimal, but this varies with the location of the brain injury and the age of the patient.<sup>32</sup>

As noted, tests that measure learning and memory, such as the *California Verbal Learning Test*, are likely to show large practice effects. Practice effects are most pronounced with repetition of the same test. However, test taking alone can also substantially improve subsequent performance on unrelated tests. This is a phenomenon referred to as test sophistication.<sup>4</sup> Unfortunately, when one reviews the current neuropsychological literature, little guidance is offered about the interpretation of practice effects within brain injury assessment. Moreover, within the existing neuropsychological literature, there is little consensus regarding how practice effects may vary as a function of the first score, type of task, length of the retest interval, age of the subject, or population.

The length of the test–retest interval is an important variable one must consider when interpreting reliability data of neuropsychological tests. As the retest interval increases, the correlation between test and retest scores should decrease. Studies on the *Wechsler Adult Intelligence Scales* (WAIS) have proved well that the longer the test–retest interval, the smaller the gains by retesting.<sup>33</sup> However, how these trends develop with other neuropsychological retest measures has not been well studied, and the adequate length of time between neuropsychological tests to remove practice effects is not well known. The clinical folklore of neuropsychological testing suggests that 6 months is an adequate length of time to diminish or remove practice effects. This assumption is rarely based on data among the many neuropsychological test instruments available to the clinician. The text by McCaffrey et al.<sup>34</sup> is probably the largest compendium of data available enabling practitioners to assess practice effects among contemporary neuropsychological test instruments.

## MEASURING ATTENTION

### The Neuroanatomical and Neuroimaging Bases of Attention

An attentional domain exists for each of the five senses. The modulation of attentional tone exists for all of these senses and occurs in a bottom-up fashion. The bottom-to-top arousal mechanisms are transmitted via the ascending reticular activating system (ARAS).<sup>35</sup> The ARAS influences the cerebral cortex directly and also through thalamic relays. The projections from the brain stem to the thalamus contain mostly cholinergic neurons, and these originate in the pons and nuclei of the brain stem reticular formation.<sup>36</sup>

Whereas the ARAS functions in a bottom-up fashion, the prefrontal cortices and the parietal and limbic systems mediate the top-down modulation of attentional responses. This is done in ways that are sensitive to the context of the stimulus, the motivation of the person, the acquired significance of the stimulus, and the conscious volition of the patient.<sup>35</sup> Metabolic activation of the prefrontal cortex and posterior parietal cortex is a common finding in almost all attentional tasks, regardless of the sensory modality or the stimulus character. The neuroimaging importance of the human prefrontal cortex to working memory was confirmed almost 30 years ago.<sup>37</sup> This functional imaging experiment found that reverse digit span tasks requiring working memory resulted in blood flow activations that were maximal over the frontal lobes. Working memory has been functionally divided into two groups of processes: (1) the online maintenance of information, and (2) the active

manipulation of information in cognition. The active manipulation aspect is within the function of a central executive agency. In humans, tasks that emphasize this executive aspect of working memory will elicit the preferential activation of the dorsolateral cortex in the prefrontal brain. Tasks that are based upon the online maintenance of information activate the prefrontal cortex and also the posterior parietal cortex.<sup>38</sup>

In humans, it has been determined that mood and motivation strongly influence how attentional resources are allocated to extrapersonal space. The mood and motivation modulation of attention is mediated through top-down projections that emanate primarily from limbic structures. Activity in the amygdalae modulates the response of the visual processing areas in the occipital cortex to faces displaying certain types of emotional expression.<sup>39</sup> The anterior cingulate gyrus within the limbic structures also exerts a generalized influence on the modulation of attention. During selective and divided attention, the anterior cingulate is activated, regardless of within which sense the stimulus is applied. Cingulate activation is associated with an improvement of performance within tasks of vigilance and spatial attention. Regional cerebral blood flow measurements have confirmed this.<sup>40</sup> Furthermore, the limbic, parietal, and prefrontal cortex top-down modulation of attention places an attentional valence upon sensory events. Motivational and mood factors can modify this valence. Damage to the top-down portions of the attentional matrix during brain injury can provide for the emergence of multiple attentional deficits and may explain why focal lesions in the prefrontal cortex, posterior parietal cortex, and medial temporal cortex can lead to an acute confusional state.<sup>41</sup>

The bottom-up control of attentional tone from the midbrain structures seems to have no laterality. However, the top-down control of the attentional matrix by prefrontal and parietal cortices displays a pattern of right hemisphere specialization. Sustained attention and divided attention in any sensory modality elicit a greater activation of the right posterior parietal and prefrontal cortices than their analogs in the left hemisphere.<sup>42</sup> Moreover, clinical evidence based upon thousands of patients reveals that neglect syndromes are more frequent, severe, and lasting after a right hemisphere injury than after an equivalent injury of the left hemisphere.<sup>43</sup> This has been further confirmed by the intracarotid injection of sodium amytal, which produced a visual neglect and tactile extinction syndrome only after the right hemisphere was inhibited, but not with left hemisphere inhibition.<sup>44</sup> Mesulam<sup>35</sup> has noted that the left hemisphere attends predominantly to the right side of space and coordinates the distribution of attention mainly within the right hemispace. It shifts its attention mostly in a rightward direction. On the other hand, the right hemisphere attends to both of the hemispaces and distributes attention within both areas. It shifts attention both to the left and the right, and it devotes more neuronal resources to spatial attention and attentional tasks than the left hemisphere. Mesulam cautions that it is no longer accurate to designate neglect syndromes as parietal syndromes. The more accurate designation is to characterize them as an attentional network syndrome, because the responsible lesion can be anywhere within the network.<sup>35</sup> The network is comprised of the ARAS producing upward attentional tone, whereas the downward control is a triad of the posterior parietal cortex, frontal eye fields in the anterior brain, and the cingulate gyrus. This triad coordinates and integrates through the thalamus, striatum, and superior colliculus.

The posterior parietal lobe plays a primary role in attention. Neurological observations suggest that the brain does not have a single spatial map. Instead, the posterior parietal cortex contains several mappings, and the representation of space in this anatomical area appears to be encoded in terms of strategies aimed at shifting the focus of attention to a behaviorally relevant target. Neurons in the posterior and medial parietal cortex, based on studies in monkeys, play an important role in the exploratory aspects of spatial attention, such as with reaching, grasping, searching with the hands, and manual maneuvers.<sup>45</sup> Because a target can move relative to the person, the neuro-mechanisms that direct attention to external targets must be sensitive to motion of the person and of the target. In the macaque monkey, these motion-sensitive neurons have been detected in the superior temporal sulcus. These neurons help to direct attention toward targets that are in motion or to navigate our bodies among solid objects in the environment, such as when walking through a crowded hotel lobby.

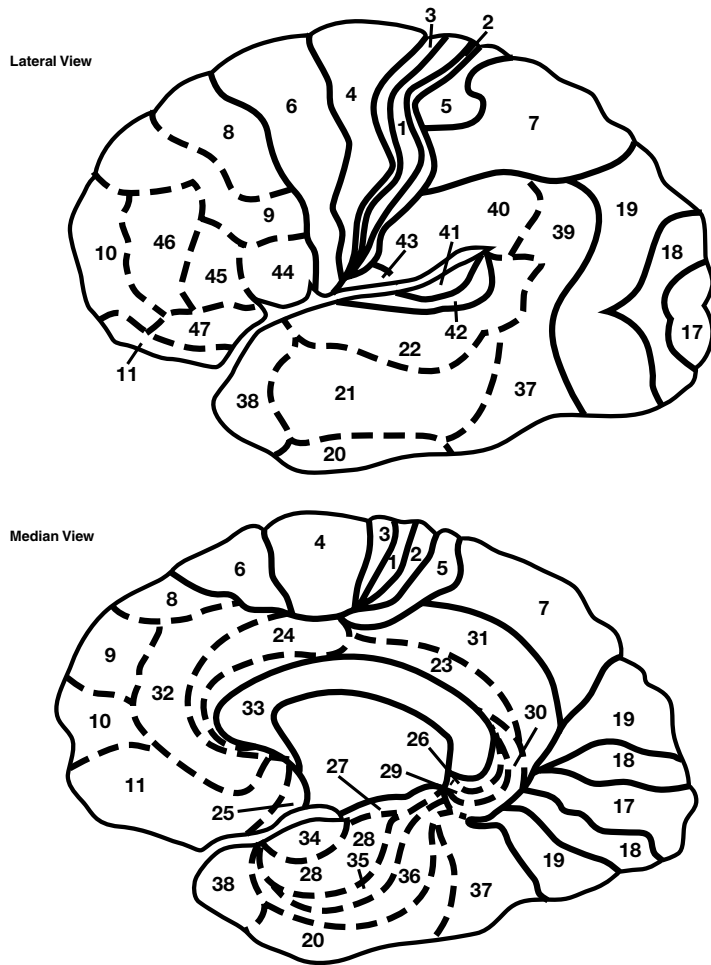
In terms of the control of attention, a distinction is made at the cognitive level between stimulus-driven or bottom-up effects on attention selection and the top-down influences that are goal driven.<sup>46</sup> The physical features of visual stimulation, such as the arrangement of objects in the extrapersonal space, will affect what information is selected by the eye. This is a bottom-up factor that is stimulus driven and qualitatively different from the top-down effect, which involves actively choosing a specific stimulus to select,<sup>47</sup> such as when locating a Canadian goose flying across one's visual field. As a result of neurophysiological and neuroimaging studies, spatial attention currently refers to the act of covertly attending to a location within the visual field that lies outside the fovea of the retina. Kastner and others used functional magnetic resonance imaging (fMRI) to examine the effects of spatial attention, and they reported that spatial attention increases stimulus-driven activity in visual areas V2 and V4, but not in the striate area V1 of the occipital lobe.<sup>48</sup> Corbetta et al. used positron emission tomography (PET) studies to determine the cortical areas of stimulation during scanning for a target. They found activations in the superior parietal lobule (Brodmann's area 7) and the superior frontal cortex (within Brodmann's area 6) during the shifting of attention condition. However, these activations were absent when the attention was fixed on the target. Thus, activity in both the parietal and frontal regions was selective for movements but not for fixation.<sup>49</sup> Both PET and fMRI studies have been used to examine whether the same cortical systems are involved in orienting attention to visual space or orienting attention to discrete time intervals, such as when an event is expected to occur at a predictable moment in time (e.g., a horse crossing a finish line during a race). Both forms of attentional orienting produced frontal activations of the dorsolateral prefrontal cortex (Brodmann's area 46). However, spatial attention selectively activated the right intraparietal sulcus, while temporal orienting selectively activated the left intraparietal sulcus. When both spatial and temporal attentions were concurrent, bilateral activations were seen in both the intraparietal sulci.<sup>50</sup> Table 6.7 outlines anatomical brain areas subserving attention. Figure 6.2 provides Brodmann's numbers to assist the reader in locating anatomic areas discussed in this chapter.

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**TABLE 6.7**  
**The Neuroanatomy of Attention**

Function	Purported Location
Bottom-to-top arousal (stimulus driven)	Projections of ARAS from the brain stem reticular formation and pons to the thalamus <sup>35,36</sup>
Top-down modulation of arousal by online maintenance of information (goal driven)	Prefrontal cortex (frontal eye fields), posterior cingulate gyrus, and parietal cortex <sup>35,38</sup>
Executive aspects of working memory	Dorsolateral prefrontal cortex <sup>37</sup>
Mood and motivational modulation of attention	Top-down projections from limbic system <sup>39</sup>
Modulation of responses to facial expressions	Amygdala <sup>39</sup>
Improvement of performance within vigilance and spatial attention	Anterior cingulate gyri <sup>40</sup>
Attentional valence upon sensory events	Limbic, posterior parietal, and prefrontal cortex <sup>41</sup>
Sustained attention and divided attention	Preferentially right prefrontal and posterior parietal cortex <sup>42,44</sup>
Exploratory attention (reaching, grasping, searching with hands)	Posterior and medial parietal cortex <sup>45</sup>
Visual stimulation during scanning for a target	Brodmann's areas 7 and 6 <sup>49</sup>
Selective spatial orienting of attention	Right intraparietal sulcus <sup>50</sup>
Selective temporal orienting of attention	Left intraparietal sulcus <sup>50</sup>

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**FIGURE 6.2** Brodmann's cortical localization.

### The Neuropsychological Measurement of Attention

vanZomerén and Brouwer have stated that attention cannot be tested. They hold that one can assess only a certain aspect of human behavior with special interest for its attentional component.<sup>51</sup> Lezak argues that while attention, concentration, and mental tracking can be theoretically differentiated, in practice these are very difficult to separate.<sup>3</sup> Attentional defects may appear as distractibility or impaired ability for focused behavior. Intact attention is a necessary precondition both for concentration and for mental tracking activities. Problems of concentration may be an outcome of a simple attentional disturbance or, on the other hand, the inability to maintain an attentional focus. Moreover, slowed processing speed often underlies attentional deficits, and simple reaction time is often slowed following traumatic brain injury. The slowing increases disproportionately as the complexity of the task increases. It has been pointed out that traumatic brain injury patients may be distinguished from normal controls due to their relatively huge variability during testing and their inconsistencies in performance.<sup>52</sup> As has been previously stated in this text, attentional measures following brain injury are usually only performed within the visual or auditory domains and sometimes in the tactile domain, but almost never in the olfactory or gustatory domains. [Table 6.8](#) lists common neuropsychological instruments for measuring attentional deficits.

**TABLE 6.8**  
**Neuropsychological Tests of Attention**

Test	Measurement
Brief Test of Attention	Auditory divided attention
Color Trails Test	Visual tracking attention (excellent where English skills are lacking)
Continuous Performance Test	Visual target vigilance
Digit Span Test (of WAIS-III)	Auditory working memory
Paced Auditory Serial Addition Test	Divided auditory attention (sensitive to subtle alterations of sustained and divided auditory attention)
Ruff 2 and 7 Selective Attention Test	Visual selective attention (can be administered at the bedside)
Stroop Color and Word Test	Visual attention and concentration (sensitive to poor patient effort)
Symbol Digit Modalities Test	Complex visual scanning and tracking
Trail-Making Test	Visual conceptual and visuomotor tracking

To further complicate the assessment of attention, many recent studies suggest that tasks thought to occupy the attentional domain in fact overlap into executive areas or executive control. For instance, the *Conner's Continuous Performance Test (CPT)* is often seen as a measure of attention and is widely used for the clinical assessment of attention deficit disorder in children. However, recent studies suggest that it may measure executive control rather than sustained attention and, therefore, may represent functions of more than one brain system. The executive control issue has been further enlarged by consideration that traumatic brain injury patients have a working memory impairment in most instances, and it appears to be due to dysfunction of the central executive system as measured by standard neuropsychological testing.<sup>53,54</sup>

Assessment of attention following mild traumatic brain injury may be the most demanding aspect of detecting change within a neuropsychiatric examination. Patients frequently complain of distractibility and difficulty attending to more than one stimulus at a time. Several neuropsychological studies have found evidence for a specific attentional deficit, whereas other neuropsychological measures may show little or no impairment. The *Ruff 2 and 7 Test* has been used to determine if processing speed declines in mild traumatic brain injury patients relative to controls. Cicerone found that patients with mild traumatic brain injury exhibit relatively subtle cognitive deficits that are apparent primarily under conditions that require effortful or controlled cognitive processing and exceed the patient's cognitive resources. In other words, a cognitive load must be placed upon the patient before attentional deficits can be seen easily, but these methods usually confirm the patient's voiced complaints.<sup>55</sup> Other studies have explored whether complex issues of attention are involved in traumatic brain injury patients beyond simple measurement of reaction times. Posner's *Covert Orienting of Attention Task (COAT)* demonstrated in one study that although the reaction times of patients with traumatic brain injury were significantly slower than those of control patients, there was no difference between the two groups in terms of their ability to disengage from a stimulus or move and engage their attention elsewhere.<sup>56</sup>

With the older patient, it is well known that slower processing speed is a consequence of aging. However, it is unclear whether older individuals with traumatic brain injury show greater relative impairment than younger individuals with traumatic brain injury. Johnstone et al. determined that the greater neuropsychological impairment noted in older individuals following brain injury is most likely related to normal aging more so than the actual injury when controlled for age.<sup>57</sup> Since the aging brain is an issue of ecological validity as applied to neuropsychological assessment, another question often encountered during the neuropsychiatric examination is whether brain injury affects a person's driving ability. Scandinavian studies have provided a wealth of information for the world in this regard. One recent study concluded that if a patient had reduced visuoconstructive ability, reaction time, and visual attention, driving was generally impaired. However, the study concluded

that, while neuropsychological assessment of these targeted functions is an ecologically valid prediction of driving skill after brain damage, on-road evaluations are still needed as supplements in cases where test findings might be ambiguous.<sup>58</sup>

With regard to whether attentional deficits improve following traumatic brain injury, arousal and motivation seem to improve over time, whereas focused attention, impulsivity, and hyperactivity, if present following brain injury, may remain stable. As far as motivation is concerned, it has been noted that, while self-motivation may be impaired with regard to attention following traumatic brain injury, external motivators may improve the attentional performance of brain-injured persons.<sup>59,60</sup>

#### *Brief Test of Attention*

The *Brief Test of Attention* (BTA) is a relatively simple and easily administered test of auditory divided attention. It is designed to be sensitive to subtle auditory attentional impairments and to reduce confounding task demands such as psychomotor speed and conceptual reasoning.<sup>61</sup> The BTA consists of two parallel forms: Form N (numbers) and Form L (letters), which are presented by an audiocassette. Each form requires about 10 min to administer. The normative sample was a reference group of 740 persons, which included 667 adults between 17 and 82 years of age and 74 children between 6 and 14 years of age.

The BTA has been used to assess patients who have sustained traumatic brain injuries and has been found to be sensitive to the auditory attentional problems of these patients for assessments even as long as 8 years after injury. It appears to possess some ecological validity in that it may predict the driving outcome of elderly patients.<sup>62</sup> Its chief limitation is that it may not be appropriate for individuals from different cultural backgrounds or those whose primary language is not English. Also, obviously this test instrument would not be appropriate for a patient with significant auditory impairment or aphasia.

#### *Digit Span Subtest*

This is a subtest of the WAIS-R or the new third edition. It consists of an oral presentation of random number sequences at a rate of approximately one per second. The patient must repeat the digits in the exact sequence in which they are presented. After each correct performance, the examiner adds a digit until the patient fails. Most patients are able to recall six digits forward and four digits backward. A difference of three or more digits between the patient's forward and backward scores is observed more commonly in brain-damaged patients than in intact individuals.<sup>63</sup>

Poor performance on this test can be due to many factors besides traumatic brain injury, such as anxiety, depression, being preoccupied, and poor effort. The *Digit Span* subtest seems more sensitive to left hemisphere brain damage than to right-sided brain damage. It is fairly resistant to the aging process. It is primarily a test of auditory working memory. Moreover, it does not correlate highly with the other 10 subtests on the Wechsler Adult Intelligence Scales. It appears to measure a very specific skill or ability. The digits-backward score appears to be more sensitive to brain damage and the effects of aging than is the forward score.<sup>12</sup>

#### *Ruff 2 and 7 Selective Attention Test*

The *Ruff 2 and 7 Selective Attention Test* (2 and 7 Test) was developed to measure two overlapping aspects of visual attention: sustained attention and selective attention. Within this testing format, sustained attention is defined as the ability to maintain a consistent level of performance over an extended period, while selective attention refers to the ability to select relevant stimuli (targets) while ignoring salient but irrelevant stimuli (distracters).<sup>64</sup>

The normative group consisted of 360 normal volunteers. These persons were stratified by age, gender, and education. One hundred of these subjects were later retested to establish the test reliability.<sup>65</sup> Ruff reported that this test can be administered easily and is sensitive to patients with brain damage involving the frontal lobes as well as temporal, parietal, and occipital lobes. It is reported to be one of the key predictors in whether patients who have sustained traumatic brain

injuries are capable of returning to work or to school, and the majority of patients with major depression are not impaired on this test, particularly if they do not exhibit clinical evidence of psychomotor retardation.<sup>66</sup>

The major strength of this test is its easy administration and the fact that it can be given at the patient's bedside. Not only is it sensitive for patients who have sustained traumatic brain injuries, but also it has been shown to be sensitive to early attentional changes in those afflicted with cerebral AIDS. It may not be appropriate for individuals who demonstrate poor vision or those who are severely anxious at the time of testing. Patients with significant motor impairment or psychomotor retardation may perform poorly on this test.<sup>12</sup>

### *Stroop Color and Word Test*

The *Stroop Color and Word Test* was developed from the observation by early experimental psychologists that the naming of color hues is always slower than the reading of color names in literate adults.<sup>67</sup> Stroop suggested that the difference in color naming and word reading was due to colors being associated with a variety of behavioral responses, while words were associated with only one behavioral response — reading.<sup>68</sup> The test consists of three pages. The first page (word page) contains color names printed in black ink. The second page (a color page) contains groupings of four X's printed in colors. The third page (word-color page) contains words from the first page printed in colors from the second page (the interference task). There are a number of different versions of this test and multiple scoring systems for the test as well. It can be administered in less than 10 min, and it is scored easily. Brain-injured patients typically respond more slowly on each of the three sections of this test, although they do not consistently demonstrate difficulties on the word-color page.<sup>69</sup>

The main strength of this test is its ease of administration to patients. It usually takes only 5 to 10 min to administer the test. It appears to be sensitive to subtle attentional and cognitive difficulties in patients who have sustained traumatic brain injuries. However, it is also sensitive to dementia. Its weakness lies in possible false positives due to anxiety, depression, or poor motivation on the part of the patient. Individuals who deliberately fake on this test may be inaccurately diagnosed as brain impaired. When using the Stroop Test, it is recommended that effort testing be included in the overall assessment process.<sup>12</sup>

### *Trailmaking Test*

The *Trailmaking Test* is an integral part of the *Halstead-Reitan Battery*. This is a timed paper-and-pencil test that consists of parts A and B. On each part, the patient is given a sample page that is used for practice to aid in understanding instructions. The examiner then gives the patient part A, which is a white sheet of paper with 24 numbered circles distributed in a random pattern, and the patient is required to connect the circles with lines in numerical order as quickly as possible. Part B consists of 25 circles. Some are numbered from 1 to 13, and the remainder are lettered from A to L. The patient is required to connect the circles beginning with number 1, then going to A, and from A to 2, 2 to B, B to 3, and so on, in an alternating sequence.<sup>72</sup>

This test is widely used as a measure of attention, visual scanning, and visuomotor tracking. Thus, it is not a pure test of attention. Part B is more difficult than Part A, as it requires the patient to shift sets (switch from a number to a letter, and vice versa), rather than connecting only numbers. One of the chief strengths of this test is that it is widely used since it is a component of the *Halstead-Reitan Battery*. It appears to be sensitive to various forms of brain damage. Moreover, a skilled examiner can observe the patient's behavior while he takes the test and easily detect qualitative errors. It is backed by a solid body of research data and normative data. The weakness of the test lies in negative effects from patients with low educational backgrounds or low intellectual functioning. Thus, it may misclassify normal adults as brain-damaged if these persons have low levels of education or intelligence. Moreover, the test may not be appropriate for persons whose native language is not English. Since it is a timed test, it may provide false positives in persons



who are anxious or depressed. It is not useful as a stand-alone test to differentiate brain-injured patients from psychiatric patients, and it discriminates poorly between these populations.<sup>12</sup>

## MEASURING MEMORY

### The Neuroanatomical and Neuroimaging Bases of Memory

As was noted in [Chapter 2](#), memory disorders are frequent abnormal neuropsychiatric conditions following traumatic brain injury. Moreover, it was pointed out in [Chapters 2 and 4](#) that explicit (declarative) memory is limbic dependent until it is consolidated, but not after consolidation. Explicit memory consists of episodic (autobiographical) and semantic (factual) memories. On the other hand, implicit (nondeclarative) memory is not limbic dependent. This form of memory is concerned with skills and habits and also classical conditioning. Priming memory, for example, may occur while instead of being asked to memorize words, the patient is asked to count how many A's the words contain. When presented again at a later time, the previously presented A word stimuli are more likely to be selected or to guide subsequent performance. This is called priming and is another form of implicit, non-limbic-dependent memory.<sup>73</sup> Furthermore, when explicit (episodic or semantic) memories are encoded, they are then stored in long-term memory. Semantic memory is used for "knowing the present," while episodic memory is for "remembering the past."

Implicit memory, such as priming and procedural skill learning, is processed very differently from episodic and semantic memory. Procedural memory is thought to be processed predominantly within regions of the cerebellum and the basal ganglia. The dorsolateral frontal cortex may participate as well.<sup>74,75</sup> Visual priming may be processed primarily within the peristriate unimodal sensory cortex. Recent functional brain imaging methods and evoked potentials suggest that visual priming also includes heteromodal association areas found in the temporal and parietal cortex.<sup>76</sup>

We have seen earlier in this text that working memory ([Chapter 2](#)) is a function of attention more so than memory. Functional imaging techniques have recently confirmed the dominant role of the dorsolateral prefrontal regions for working memory in the human brain. A functional imaging experiment suggests that the dorsolateral frontal region, as well as the ventrolateral portions of the prefrontal cortex, contributes to both spatial and nonspatial working memories.<sup>77,78</sup> Explicit episodic memory has a different neuroanatomical substrate than implicit memory. The explicit memory system is dependent upon neural networks containing limbic as well as nonlimbic components. It has been argued that encoding and consolidation can be functionally separated. However, at this historical point in medical science, the neuroanatomical basis of implicit memory functions remains unclear. The storage of memory information also is not fully elucidated. A significant body of scientific evidence points to changes in synaptic morphology, protein synthesis, and gene expression as functionally necessary for long-term memory. However, the functional changes specifically occurring within memory storage still are not known.<sup>73</sup> On the other hand, the retrieval of stored information is better understood than storage. Functional imaging studies point to a consistent activation of the left prefrontal cortex during encoding, but activation of the right prefrontal cortex during retrieval.<sup>79</sup> Following head trauma, typically the patient has a time-graded retrograde amnesia. This same amnesia is seen in patients with medial temporal or medial diencephalic brain damage from causes other than traumatic brain injury.<sup>80</sup> Memory research to date concludes that inferolateral prefrontal and temporopolar regions play an important role in the retrieval of old memories. The right hemisphere seems more critical for retrieving episodic (autobiographical) information, whereas the left hemisphere plays a more critical role in the retrieval of stored general knowledge (semantic or factual memories).<sup>81</sup>

Memory is not a unitary phenomenon. Moreover, it is now well established that a significant distinction lies between short-term and long-term memory. Limbic lesions may result in intact working memory, but impaired long-term memory. This is because working memory is an attentional function more than a memory function. Patients with memory impairment following traumatic

**TABLE 6.9**  
**The Neuroanatomy of Memory**

Function	Purported Location
Procedural (implicit or skill) memory	Cerebellum, basal ganglia, and probably dorsolateral prefrontal cortex <sup>74,75</sup>
Visual priming of memory	Peristriate, temporal, and parietal cortices <sup>76</sup>
Retrieval of stored information	Preferentially right prefrontal cortex <sup>79</sup>
Encoding of stored information	Preferentially left prefrontal cortex <sup>79</sup>
Retrieving autobiographical memories (episodic)	Right hemisphere more critical <sup>81</sup>
Retrieving factual memories (semantic)	Left hemisphere more critical <sup>81</sup>
Naming objects and reading words	Bilateral fusiform gyri, left activation greater during reading <sup>84-86</sup>
Identifying and naming animals	Lateral fusiform gyrus, medial occipital cortex, and superior temporal sulcus <sup>85</sup>
Visual processing pictures of tools	Medial fusiform gyrus, left middle temporal gyrus, and left premotor cortex <sup>85</sup>

brain injury usually show neither total obliteration of previously learned information nor a total inability to acquire new information. Varying degrees of dysfunction within old and new memories generally remain. For instance, the ability to drive an automobile, to learn to avoid sticking one's hand in a fire, or to know basic information about social function generally is preserved even after damage to the limbic system during brain trauma. [Table 6.9](#) describes known brain anatomy of memory function.

fMRI and PET studies have markedly enhanced our understanding of episodic memory. The most remarkable finding from imaging research of episodic memory is the low functional activity in the medial temporal lobe. Imaging studies seem to indicate that medial temporal lobe activation is consistently associated with retrieval success of episodic memories rather than with the cognitive attempt to retrieve those memories. The attempts to retrieve episodic memories appear localized mostly in the frontal cortex. The prefrontal cortex appears to play a very prominent role in the modulation of episodic memory encoding. Left prefrontal activation is consistently associated with the encoding of episodic verbal memories. Left prefrontal activation has also been associated with enhanced memory for nonverbal stimuli, specifically faces. The right prefrontal cortex activates during the encoding of nonverbal materials in a wide variety of situations. A consistent right, prefrontal activation occurs for many types of memory data, including verbal, nonverbal, recall, and recognition. Thus, the right prefrontal cortex appears to play a very prominent role in nonverbal episodic memory while working in parallel with the left prefrontal cortex during encoding of verbal episodic memory.<sup>82</sup>

With regard to the functional imaging of semantic (factual) memory, the clinical literature has suggested that semantic processing may be dependent on left temporal lobe function. Activation of the left inferior frontal cortex detected by neuroimaging is consistent with the clinical literature. There is a large body of functional brain imaging studies documenting this anatomical area during word selection and retrieval.<sup>83</sup> The other prominent site of activity detected by functional imaging is in the posterior temporal lobe centered over the fusiform gyrus, located on the ventral surface of the temporal lobes. Many studies have reported this anatomical area to be activated bilaterally during object naming.<sup>84</sup> Neuroimaging studies indicate that during both naming objects and reading words, this ventral region of the posterior temporal lobes centered within the fusiform gyrus is activated. This effect is greater on the left hemispheric side than the right side, especially during word reading. These data suggest that activation of this region is independent of the physical form of the stimulus presented to the subjects.<sup>85</sup> Thus, the ventral region of the temporal lobes, particularly the fusiform gyrus, is engaged during lexical or semantic processing. For tasks that require effortful

**TABLE 6.10**  
**Neuropsychological Tests of Memory**

Test	Measurement
Auditory–Verbal Learning Test	Immediate memory span (provides a learning curve)
Benton Visual Retention Test	Visual recall
Brief Visuospatial Memory Test-Revised	Visual learning, delayed recall, and recognition
Brown–Peterson Technique	Short-term verbal retention
Buschke Selective Reminding Test	Verbal short-term retention, storage, and retrieval
California Verbal Learning Test	Verbal memory and verbal learning strategies
Complex Figure Test	Immediate and delayed visual recall
Recognition Memory Test	Recall of words and faces
Rivermead Behavioral Memory Test	Tests everyday verbal and visual memory
Ruff–Light Trail Learning Test	Visuospatial learning
Wechsler Memory Scale-III	Complex battery for testing verbal and visual memories, working memory

retrieval of semantic information, the pattern of left hemisphere activation broadens. The areas included are the ventral and lateral regions of the posterior temporal lobe and the inferior parietal and prefrontal cortices.<sup>86</sup>

Very recent functional neuroimaging studies suggest that different classes of objects, such as animals and tools, are differentially represented in the cerebral cortex. In a task dependent on identifying and naming pictures of animals, neuroimaging activity is greatest in the more lateral aspects of the fusiform gyrus, medial occipital cortex, and superior temporal sulcus. On the other hand, visual processing pictures of tools are associated with activation of the more medial aspect of the fusiform gyrus, the left middle temporal gyrus, and the left premotor cortex.<sup>85</sup>

### The Neuropsychological Measurement of Memory

The measurement of memory is complex. There is a functional memory system for each of the five senses. In general, neuropsychological examinations measure memory in the visual and auditory domains almost exclusively. Lezak<sup>3</sup> believes that at a minimum, a memory examination should cover (1) span of immediate retention, (2) learning in terms of extent of recent memory, and (3) retrieval of recently learned and long-stored information.

The examiner should remember, as previously noted, that diminished attention may affect memory acquisition. However, it seems to affect implicit memory more than explicit memory.<sup>87</sup> Studies of traumatic brain injury patients suggest that initial acquisition of memory data is more compromised than its retrieval.<sup>88</sup> By studying pure verbal learning, there is evidence that the consolidation is impaired to a greater extent than the encoding or retrieval of memory data.<sup>89</sup> [Table 6.10](#) lists neuropsychological tests often used for measurement of memory.

#### *Ruff–Light Trail Learning Test*

The *Ruff–Light Trail Learning Test* assesses visuospatial learning and memory in adults. The test was specifically developed to avoid requiring the patient to possess drawing skills, keen eyesight, good motor control, and refined visuospatial integration. Thus, it is very useful in traumatically brain-injured persons.<sup>90</sup> This test makes a direct measure of immediate visual memory as well as visuospatial learning. It also has a delayed recall section, and it allows for the development of learning curves over the course of the testing. It has been standardized for use with individuals ages 16 to 70 years, and normative data are available for two age groups: 16 to 54 years and 55 to 70 years. It is not validated for individuals under the age of 16.

### *Wechsler Memory Scale-III*

Wechsler Memory Scale-III (WMS-III) is a revision of the *Wechsler Memory Scale-Revised* (WMS-R). The basic structure of the WMS-III is the same as that of the WMS-R, and it retains the tradition of assessing memory and attentional functioning within both auditory and visual stimuli.<sup>91</sup> Changes in the WMS-III relative to the WMS-R include the addition of new subtests, a revision of memory stimuli, an expansion of scoring options, an addition of subtest scaled scores, and an expansion of indices in both content and number.

The scores from WMS-III are organized into summary index scores. The primary index scores are:

1. Auditory immediate: the ability to remember information immediately after it is orally presented
2. Visual immediate: the ability to remember information immediately after it is visually presented
3. Immediate memory: the ability to remember both visual and auditory information immediately after it is presented
4. Auditory delayed: the ability to recall orally presented information after a delay of approximately 30 min
5. Visual delayed: the ability to recall visually presented information after a delay of approximately 30 min
6. Auditory recognition delayed: the ability to recognize auditory information after a delay of approximately 30 min
7. General memory: the delayed memory capacity based upon scores from Logical Memory II, Verbal Paired Associates II, Faces II, and Family Pictures II
8. Working memory: the capacity to remember and manipulate visually and orally presented information in short-term memory storage using performance data from the Spatial Span and Letter–Number Sequencing subtests

The WMS-III is one of the most widely used scales to assess memory. It now supplants the very widely used WMS-R. The tests are relatively easy for an experienced psychologist to administer and score. Normative data are available for persons ranging from 16 to 89 years. However, WMS-III takes much longer to administer than the older edition, WMS-R, especially if it is administered to brain-injured patients. Many neuropsychologists avoid administering the full WMS-III battery due to that limitation, and it may not be appropriate for a severely brain-injured person who is extremely impaired cognitively or physically. Currently, no normative data are available on this test instrument for persons in whom English is the second language.

Unlike the WMS-R, the WMS-III contains four supplementary auditory composites:

1. Single-trial learning: the capacity to immediately recall auditory data after a single exposure to material
2. Learning slope: the ability to acquire new auditory information after repeated exposures
3. Retention: the delayed recall capacity as a function of immediate recall performance after a delay of approximately 35 min
4. Retrieval: the retrieval for recall vs. recognition memory

## **MEASURING LANGUAGE**

### **The Neuroanatomical and Neuroimaging Bases of Language**

Language disorders are seen not only in audio-based languages such as English, French, or Spanish. In fact, persons who must use American Sign Language for communication can also demonstrate

aphasia while using only visuomotor signs. Those with oral language-based communication disorders often also demonstrate a deficit in the written aspects of language, including audio-based languages and languages based on ideograms such as Chinese or Japanese. Language disorders can affect multiple aspects of language processing. The Damasio's have described three major outcomes of language processing dysfunction, namely: (1) syntax, the grammatical structure of sentences; (2) lexicon, the words available in a language to denote particular meanings; and (3) the morphology of words, how individual speech sounds are combined from phonemes.<sup>92</sup>

In classical neurology, the aphasic syndromes are organized around Broca's aphasia, Wernicke's aphasia, and conduction aphasia. In general, the disorders of language following traumatic brain injury do not follow these patterns (please see [Chapter 2](#)). Whereas the classical aphasic disorders are distinguished by the afflicted person's inability to repeat sentences, the transcortical aphasias, another form of language dysfunction, are found in those persons who can provide normal sentence repetition, and these language disorders usually anatomically lie outside the perisylvian area. Neuroanatomically, those with Broca's aphasia are found to have damage in the dominant frontal lobe within Brodmann's areas 44 and 45 in the inferior left frontal gyrus. The surrounding Brodmann's frontal areas of 6, 8, 9, 10, 46, and 47 may also be affected, as well as the underlying white matter tracts in the subjacent basal ganglia.<sup>93</sup> Classical Broca's area is comprised of Brodmann's areas 44 and 45. Damage in this area alone, without involvement of the surrounding cortical areas and basal ganglia, will not produce classical Broca's aphasia. These persons are distinguished by a mild and transient aphasia. Structures usually damaged in patients who produce typical Broca's aphasia, such as seen in strokes, but rarely in traumatic brain injury, are those involved in the assembly of phonemes into words and the assembly of words into sentences. This requires ordering of linguistic components in time, and it has been suggested that this system is composed of three anatomical areas in the external left frontal cortex (Brodmann's areas 47, 46, and 9), the left parietal cortex (Brodmann's areas 40, 39, and 7), and in the sensory motor cortex above the Sylvian fissure (the lower sector of Brodmann's areas 3, 1, 2, and 4). The left basal ganglia and head of the caudate nucleus in the putamen also seem to be critical subcortical components of the entire Broca's aphasia syndrome.<sup>92</sup>

Wernicke's aphasia is usually due to neural damage to the posterior sector of the left auditory association cortex (Brodmann's area 22). There also may be secondary involvement of Brodmann's areas 37, 39, and 40, either any one or all three.<sup>94</sup> Damage to Wernicke's area disrupts auditory comprehension, but it is not the center in which auditory comprehension takes place. Wernicke's area is a processor of speech sounds and thus recruits auditory inputs to be mapped as words and to be used subsequently to evoke concepts. The process of auditory comprehension is much more complicated than mere reception and involves numerous areas of the cerebral cortex within various sensory modalities located in the parietal, temporal, and frontal brain regions.

Persons who sustain conduction aphasia can usually comprehend simple sentences and produce intelligible sentences without the severe dysfluency seen in Broca's aphasia. They generally cannot repeat sentences verbatim, and since they have difficulty assembling phonemes, they tend to produce phonemic paraphasias (sound errors). They also generally have an anomia when asked to name confrontationally. Thus, generally these persons show relatively preserved speech production and auditory comprehension with an inability to effectively repeat, assemble phonemes, and name.<sup>95</sup> Conduction aphasia usually occurs with damage in one of two brain regions: (1) left cerebral Brodmann's area 40 (supramarginal gyrus), or (2) the left primary auditory cortex (Brodmann's areas 41 and 42), which includes the insula and the underlying white matter. In either form, Brodmann's area 22 is usually spared. Often, damage occurs in the classical arcuate fasciculus, which traverses underneath the angular gyrus and supramarginal gyrus.<sup>96</sup> For the transcortical aphasias, the area of brain injury for the motor variant usually occurs with damage to the left frontal cortex superior and anterior to Broca's area. The sensory variant is usually found following lesions in the temporal or parietal cortex surrounding Wernicke's area.<sup>94</sup>

Recall from [Chapter 4](#) that a nonverbal language system operates parallel to the verbal system and is located in the nondominant hemisphere. Moreover, it is scientifically accepted that right

hemisphere injury may interfere with discourse, the skill with which one can organize a narrative story, make a joke, or write a letter.<sup>97</sup> Right hemisphere injury often affects prosody; this ability refers to the inflections, stresses, and melody of speech used during the production of words and sentences, providing meaning that goes beyond their basic dictionary descriptions.<sup>98</sup> The clinical syndromes that arise from language disturbances in the right hemisphere have been collectively called the aprosodias. These disorders selectively impair the production, comprehension, and repetition of affective prosody without disrupting the propositional elements of language.<sup>99</sup> MRI brain studies have generally shown that patients with impaired spontaneous affective prosody had lesions involving the posterior-inferior frontal lobe, which included the pars opercularis and triangularis, regions similar anatomically to Broca's areas in the left hemisphere. Those patients with more posterior lesions impairing comprehension of affective prosody had cortex lesions involving the posterior-superior temporal lobe, again a region similar and analogous to the anatomical Wernicke's areas in the left hemisphere. Thus, there appears to be a dual-highway language system with symbolic language produced and decoded primarily in the left hemisphere while the affective components of language are produced and decoded primarily in the right hemisphere.<sup>100</sup>

Imaging studies of language centers reveal a consistent activation of the superior temporal gyrus using PET and fMRI studies in subjects presented with speech sounds in contrast to no sounds at all.<sup>101</sup> The activated areas include Heschl's gyrus, the planum temporale, the dorsal superior temporal gyrus anterior to Heschl's gyrus, the lateral superior temporal gyrus, and the superior temporal sulcus. Sounds in general cause activation of these areas. In fact, speech and nonspeech sounds produce roughly equivalent activation of the dorsal superior temporal gyrus, including the planum temporale, in both the left and right hemispheres. However, speech sounds, rather than nonspeech sounds, preferentially activate the more ventral areas of the superior temporal gyrus within and surrounding the superior temporal sulcus.<sup>102</sup> The consistent findings of neuroimaging of language reveal that activation of the superior temporal gyrus and superior temporal sulcus does not differ from meaningful speech sounds vs. ones that have no meaning. These findings have been interpreted to indicate that the anatomic areas in the superior temporal lobe are unlikely to play a prominent role in the processing of semantic or lexical language information and are confined entirely to the analysis of speech sounds. There are areas more ventral and on the lateral surface of the superior temporal gyrus and within the superior temporal sulcus that respond to more complex auditory phenomena, such as the frequency and amplitude and spectral energy peaks that characterize speech sounds.<sup>101</sup> See [Table 6.11](#) to review the anatomy of language.

Neuroimaging of the perceptual processing of written symbols, such as letters, reveals that the calcarine cortex and the adjacent medial occipital extrastriate regions are activated by printed word stimuli in contrast to no stimulus. This activation is interpreted as representing early visual information processing, and it is thought that these areas do not differentially analyze words or pseudowords.<sup>103</sup>

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**TABLE 6.11**  
**The Neuroanatomy of Language**

Function	Purported Location
Brain activation by speech sounds	Heschl's gyrus, planum temporale, dorsal superior temporal gyrus, lateral superior temporal gyrus, and superior temporal sulcus <sup>101,102</sup>
Brain activation by written symbols	Calcarine cortex and medial occipital extrastriate region <sup>103,104</sup>
Letter processing	Posterior fusiform and inferior occipital gyrus, left greater than right <sup>104,105</sup>
Phoneme processing	Left frontal operculum (anterior insula and Brodmann's area 45) and inferior frontal gyrus (Brodmann's areas 6 and 44) <sup>106</sup>
Semantic (meaning) analysis	Brodmann's area 39 (angular gyrus) <sup>96</sup>
Self-generated word production	Frontal operculum, inferior frontal gyrus, middle frontal gyrus, inferior frontal sulcus, middorsal frontal sulcus (Brodmann's areas 6, 8, 44–47) <sup>101</sup>

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Puce and others have provided studies suggesting that the locus for letter processing is in the posterolateral fusiform or inferior occipital gyrus. Letter-specific activation appears to be strongly left lateralized.<sup>104</sup> There is a difference in the activation when using pronounceable letter strings vs. those that are not pronounceable. Both words and pseudowords that can be pronounced produce activation in a left ventromedial extrastriate region located in approximately the posterior lingual gyrus or the lingual-fusiform border. Consonant strings and false fonts do not activate this region.<sup>105</sup>

Phonological processing refers to operations involving speech sound perception or production of discrete sound elements (phonemes). There appear to be two distinct regions of the inferior frontal cortex that are involved in phoneme processing: the left frontal operculum (around the anterior insula and Brodmann's area 45) and a more posterior dorsal region found near the inferior frontal gyrus–premotor boundary in Brodmann's areas 44 and 6. These regions are activated on PET scans.<sup>106</sup> In summary, a ventral region of the left supramarginal gyrus is involved in some phoneme processing tasks with both heard words and nonwords. The same region is also activated for pitch discrimination of tones, for reading visually presented words relative to picture naming, and for reading pseudowords relative to true words.<sup>101</sup>

Semantic processes are concerned with storing, retrieving, and using factual knowledge about the world. These are a key component of language behaviors such as naming, comprehending, problem solving, planning and thinking, and the formulation of language expressions. Neuroimaging evidence indicates that the single most consistently activated region for semantic analysis is the angular gyrus (Brodmann's area 39). This brain area is phylogenetically a recent addition and specifically human in development, compared to primates and other mammals. It is a multimodal convergence area and is situated strategically between visual, auditory, and somatosensory centers.<sup>96</sup> With regard to word production, self-generated words produced by demand, such as during the performance of the *Controlled Oral Word Association Test* (COWA), activate the frontal operculum, the inferior frontal gyrus and Brodmann's areas 44 to 47, the posterior part of the middle frontal gyrus and inferior frontal sulcus, and the middorsal part of the precentral sulcus (Brodmann's areas 6, 8, and 44).<sup>101</sup>

## The Neuropsychological Measurement of Language

As noted in [Chapter 2](#), language disorders are not very common following traumatic brain injury and occur in only about 2% of traumatically brain-injured persons. It is further pointed out that anomia is the most common language disorder seen following traumatic brain injury with some occurrence of dysfluency also. Severe language disorders following traumatic brain injury are most likely seen in persons who have sustained a subdural hematoma over the dominant hemisphere language area or in those who have sustained a penetrating brain injury into either the anterior or posterior neuroanatomical language areas. Thus, there are times when it may be necessary to provide a full language assessment following a traumatic brain injury. It may be necessary for the neuropsychiatric examiner with limited experience in aphasias to consult with a speech pathologist. Moreover, if a neuropsychologist is used for evaluating language disorders following brain trauma, the neuropsychologist should have had significant experience and training in aphasia to be able to use a complex language instrument such as the *Boston Diagnostic Aphasia Examination*. Lezak further points out that the screening of language disorders requires at a minimum an assessment of spontaneous speech and repetition of words, phrases, and sentences, as well as an assessment of speech comprehension.<sup>3</sup>

Mild traumatic brain injury and postconcussive syndrome can result in subtle language changes. Narrative discourse production may be impaired. In other words, the patient may demonstrate difficulty relating a story to the examiner.<sup>107</sup> For those examiners determining language defects in persons whose first language is not English, special difficulties are presented. A few tests for language dysfunction of Hispanics are being developed, but most are not online at this time. In light of the increasing Spanish-speaking population in the U.S., there is available the *Multilingual Aphasia Examination-Spanish*. This appears to be a sensitive and accurate measure of language

**TABLE 6.12**  
**Neuropsychological Tests of Language**

Test	Measurement
Aphasia Screening Test	A language screening test of the Halstead–Reitan Battery; Lezak says to “junk it altogether” <sup>3</sup>
Boston Diagnostic Aphasia Examination (BDAE)	Available in English, Spanish, and French; requires diagnostic skills in aphasia to use properly; the gold standard of full language assessment
Boston Naming Test	Effectively elicits an anomia if present
Controlled Oral Word Association Test	Assesses word fluency; measures frontal lobe word output ability
Multilingual Aphasia Examination	Revised by Benton and is a full language battery; requires less time for administration than the BDAE; Spanish version is available
Token Test	Assesses ability to perform spoken commands; detects comprehension
Western Aphasia Battery	A full language assessment battery; the diagnostic classification poorly describes patients with mixed language disorders

disturbances in Hispanic populations.<sup>108</sup> Table 6.12 lists some common neuropsychological tests of language.

#### *Boston Diagnostic Aphasia Examination*

This test is based on the original development of an aphasia screening examination used within the Boston school of neuropsychological assessment<sup>109</sup> (see Table 6.3). This is the gold standard for evaluating language disorders. However, the examiner must remember that it was developed on aphasic patients following strokes and not on normative cases from traumatic brain injury. It does highly correlate with other tests of language, but it has no test–retest reliability data available should subsequent examinations be administered.<sup>110</sup>

The strength of this test lies in its excellent ability to diagnostically categorize the full scope of language disorders. Its main weakness lies in the time it takes to administer the test. A neurologically intact person generally requires 1½ to 2 h administration time to complete the test, whereas a traumatic brain injury patient may require as long as 4 h. Therefore, motivational factors certainly could come into play with this test. Spordone and Saul concurs with Lezak and admonishes that neuropsychologists need strong backgrounds in the study of language disturbances and aphasias to use the examination well.<sup>12</sup>

#### *Boston Naming Test*

This test is also an outgrowth of Kaplan et al.’s original work developing the Boston Diagnostic Aphasia Examination. It consists of 60 drawings of objects that become increasingly less familiar and difficult to name.<sup>111</sup> This test has good test–retest reliability and has been examined over as long a duration as 8 months from a prior testing.<sup>112</sup> The test manual provides normative data based on the patient’s educational achievement and age. The manual advises that poor scores on this test can be due to a variety of factors, including a limited cultural or language background, low intellectual functioning, low level of education, or a psychiatric disturbance.<sup>12</sup>

#### *Controlled Oral Word Association Test*

This is a test of fluency and consists of instructing the patient to name as many words as possible beginning with specific letters of the alphabet. The patient’s score is based on the total number of words produced during three trials while using the letters *F*, *A*, and *S*.<sup>113</sup> It appears to be sensitive to frontal lobe injury, and patients who have sustained more severe traumatic brain injuries score lower than patients with less severe brain injuries.<sup>114</sup> This test also is noted to be sensitive to injury in the left frontal-temporal area. However, the lowest score made on this test usually occurs in patients who have sustained bilateral frontal-temporal lobe injuries. Poor performance can occur



with patients who are suffering from anxiety, depression, sleep deprivation, cultural deprivation, and poor language skills.<sup>12</sup>

### *Token Test*

This test measures a person's ability to comprehend and perform commands that are presented orally. During testing, the patient is presented a set of tokens of varying forms and substances, such as cardboard, plastic, circle, square, etc. The examiner then instructs the patient with direct commands or complex commands such as "Touch the white square" or "Before touching the yellow circle, pick up the red square." This test has been found to be sensitive for examining patients with receptive language deficits following brain injury, and it has been used to evaluate children and adolescents who have sustained closed-head injuries.<sup>110,115</sup>

This test is simple and administered straightforwardly. Therefore, neurologically intact patients should have no difficulty obtaining excellent scores. Patients with receptive aphasia or language disorders due to posterior brain injury typically make a number of errors. However, performance can be confounded in those patients who have hearing loss, attentional deficits, psychiatric disorders, or pain.

## **MEASURING VISUOPERCEPTUAL ABILITIES**

### **The Neuroanatomical and Neuroimaging Bases of Visuoception**

As we noted in [Chapter 2](#), most individuals who suffer closed traumatic brain injury display normal visual-perceptual abilities. This statement does not hold for patients who have sustained brain contusions or hematomas or those who have right hemisphere bruising or bleeding. These individuals are the most likely to demonstrate a visuo-perceptual disorder. If a detailed understanding of visuo-perception is required, the reader should consult Damasio et al.<sup>116</sup>

The neuropsychiatric examiner may notice in the medical records produced immediately following brain trauma that individuals were unable to recognize relatives or their spouses. This dysfunction is known as prosopagnosia, which is a visual agnosia hallmarked by an inability to recognize the faces of previously known persons (retrograde visual amnesia) or to learn the faces of new persons (anterograde visual amnesia). For instance, the patient may not learn the face of his nurse or his physician while in the neurosurgical intensive care unit (ICU) because of visual agnosia. It is not unusual to find prosopagnosia occurring in patients who have sustained a visual field cut as a result of cerebral bleeding or intracerebral trauma. True prosopagnosia is almost entirely a disorder of visually triggered memory. For instance, the patient who fails to recognize his wife visually can recognize her usually by her voice.

Damasio et al. report that where prosopagnosia extends beyond the acute phase, the lesions are almost always bilateral.<sup>116</sup> The injury is most likely within either the inferior and mesial visual association cortex in the lingual and fusiform gyri or the subjacent white matter. These lesions tend to involve equivalent portions of the central visual pathways in the left and right hemispheres. Bilateral lesions located exclusively in the superior visual association cortex do not cause prosopagnosia. Human facial recognition appears to be represented in both hemispheres.

Within the disorders of complex visual processing lie disorders of topographic (spatial) orientation. For instance, if a person cannot locate a public building in a city or find his room in the hospital or at home, this would be a demonstration of topographic disorientation. Defects of this nature appear to represent impairments of visuospatial memory. By using functional MRI, Epstein and Kanwisher have found a specific area within the human parahippocampal cortex that responds to places more than faces. This area has been termed the *parahippocampal place area* (PPA) and is involved in perceptions of the local visual environment. This, of course, is an essential component of navigation.<sup>117</sup> Damasio et al. believe that the PPA represents places by encoding the geometry of the local environment.<sup>116</sup> The reader should reflect back to earlier discussions in this text regarding

episodic memory, as these would be operational within the visuospatial system while a person is, for instance, driving through a new city. Spatial analysis is required within complex visual processing. The most famous syndrome within a disorder of visual-spatial analysis is BÀlint’s syndrome.<sup>118</sup> This syndrome consists of visual disorientation, optic ataxia, and ocular apraxia. A patient with this disorder is visually disoriented and cannot reach to grab an object in the visual field, and the patient also demonstrates difficulty with visual scanning. Damasio et al. point out that the Cookie Theft picture from the Boston Diagnostic Aphasia Examination is an excellent means of deciding whether the patient can cope with the rapid analysis of a visual scene.<sup>116</sup>

The full BÀlint’s syndrome, when it occurs, is usually related to bilateral damage of the occipitoparietal area. It is unlikely that this syndrome is seen in a case of traumatic brain injury unless severe hypotension occurred following trauma, producing a watershed infarction in the border zone between the anterior and posterior cerebral artery territories. This has been seen following severe bleeds causing hypotension in persons who also sustained a traumatic brain injury concurrently with severe volume loss. The ability to judge the direction and orientation of lines is also an element of spatial analysis. This is usually examined within the *Judgment of Line Orientation Test*.<sup>119</sup> Right occipitoparietal lesions are thought most likely to impair performance on this particular test.

The processes of visual recognition include four main components: (1) early vision, (2) shape analysis, (3) matching to stored visual descriptions, and (4) accessing semantic and conceptual representations.<sup>120</sup> Recent PET studies have noted that the occipitotemporal areas are activated for a face-matching task, but a location-matching task activates occipitoparietal areas.<sup>121</sup> These findings were confirmed by Kohler et al., who found greater activation in ventral occipital regions for tasks requiring encoding of the identities of objects, but greater activation in dorsal regions when a location task had to be carried out on the same stimuli. These studies indicate that the ventral occipitotemporal cortex is the general region where any modular components of visual recognition are most likely to be found.<sup>122</sup> Kanwisher et al. detected a specific *fusiform face area* (FFA) in the mid-fusiform gyrus that seems to be a distinct face-selective region.<sup>123</sup> These researchers point out that the apparent specificity of the FFA for face perception dovetails with the evidence from prosopagnosia that face perception is subserved by specialized cortical mechanisms. However, it remains to be proven that the FFA is in fact the cortical region that is damaged causing prosopagnosia in the areas noted previously by Damasio et al. [Table 6.13](#) reviews the neuroanatomy of visuoperception.

While Epstein and Kanwisher were performing their face recognition experiments, they noted that in virtually every participant studied on the standard faces vs. objects comparison, a large region in the bilateral parahippocampal cortex showed the reverse effect: it was more active during object viewing than face viewing.<sup>117</sup> The responses to this region were tested using complex scenes such as landscapes, rooms, and outdoor campus scenes. The results from this experiment were startling. The same region of parahippocampal cortex that had repeatedly shown a greater activation for objects than for faces showed a much stronger activation for scenes than for either faces or objects.<sup>123</sup> Epstein and Kanwisher have named this region of the cortex the PPA, as noted earlier.

**TABLE 6.13**  
**The Neuroanatomy of Visuoperception**

Function	Purported Location
Prosopagnosia	Lingual and fusiform gyri; subjacent white matter (inferior and mesial visual association cortex) <sup>116</sup>
Topographic disorientation	Parahippocampal place area <sup>117</sup>
BÀlint’s syndrome	Bilateral occipitoparietal areas <sup>118</sup>
Judgment of orientation and direction of lines	Occipitoparietal areas <sup>119</sup>
Face selection	Fusiform face area (mid-fusiform gyrus) <sup>122,123</sup>
Emotional expression in faces	Amygdalae <sup>124</sup>

One question posed by this type of research is whether perception without awareness is possible? An elegant study partially answers this question. Mass groups of emotionally expressive faces were presented to subjects in an fMRI scanner. In this study, the amygdala produced a stronger activation for emotional expressive faces than for neutral faces, despite the fact that most subjects reported never having seen any expressive faces in the course of the entire experiment.<sup>124</sup>

## **The Neuropsychological Measurement of Visuospatial and Perceptual Ability**

Visuoperception is often impaired by brain injury. Typically, if one visual function is affected following brain injury, a cluster of functions will secondarily be affected as well.<sup>125</sup> Visual functions are broadly divided along the lines of verbal or symbolic and configural stimuli. Lezak warns that when using visually presented material during the neuropsychological examination of lateralized brain injury, the examiner cannot categorically assume that the right brain is doing most of the processing when the stimuli are pictures. There is some activity that occurs within the left hemisphere as well.<sup>3</sup> See [Table 6.13](#) for a survey of purported visuoperceptual neuroanatomy.

### *Bender–Gestalt Test*

Lezak places this the *Bender–Gestalt Test* within the domain of construction.<sup>3</sup> Others note that this test evaluates the patient’s visuoperceptual and visuoconstructional skills.<sup>126,127</sup> It is one of the most frequently used psychological tests in the U.S.; it has been used for over 60 years, and there are more than 1000 studies concerning its validity and reliability. However, it is only a screening test and it may be misused. Most experts feel that it should never be used as a stand-alone test or a test upon which to conclude that organic brain injury is present.<sup>12</sup>

The test consists of nine geometric designs that are presented individually to the person being examined. The patient is then asked to draw an accurate reproduction of the figure on a piece of blank paper. A number of different scoring systems exist based on the accuracy and organization of the reproduced drawing. However, there is a substantial amount of subjectiveness within this test, as its effective use depends upon the skill of the examiner.<sup>128</sup>

### *Benton Facial Recognition Test*

This test is designed to measure a person’s ability to compare photographs of faces. The patient is shown a photograph of a person’s face, and directly below the photograph are six other photographs containing someone’s face. The initial part of the test simply is to identify the person in the first six photographs. The second portion of the test reveals only three quarters of a person’s face, and the patient has to determine which face is present. In the third portion of the test, the patient must match the original photographs of faces to photographs that have been taken under low lighting conditions.

This test is quick to administer and requires about 15 min testing time.<sup>131</sup> Patients who have right parietal lesions perform more poorly than patients with right temporal lesions. Lezak suggests that this demonstrates a substantial visuospatial processing component to the test.<sup>3</sup> Thus, this test tends to be particularly sensitive to patients who have sustained posterior right hemisphere damage. It is not very sensitive to patients who have sustained left hemisphere or frontal lobe damage. Psychiatric conditions can lead to poor performance on this test. It is not a stand-alone examination, and Spordone and Saul<sup>12</sup> recommend that other neuropsychological measures be taken at the same time as this test is administered.

### *Benton Judgment of Line Orientation Test*

During this test administration, the patient is asked to match a pair of angled lines, which are shown on a card, to 1 of 11 numbered lines below it.<sup>132</sup> Essentially, the patient has to match the angle of the stimulus line to the correct angle of 1 of the 11 numbered control lines. While performing this test, cerebral blood flow in temporo-occipital areas increases bilaterally. However, the greatest increase is on the right side.<sup>129</sup> Most patients with left hemisphere damage alone perform this test

**TABLE 6.14**  
**Tests of Visuospatial and Constructional Skills**

Test	Measurement
Bender–Gestalt Test	Visual perceptual and visual constructional skills; right greater than left (R > L) parietal lobe
Benton Facial Recognition Test	Subtle perceptual and visual discrimination; R > L parietal lobe
Benton Judgment of Line Orientation Test	Ability to estimate angular relationships between line segments; rCBF increases in bilateral temporal-occipital areas; R > L <sup>129</sup>
Block Design Test (WAIS)	Visuospatial organization skills; glucose metabolism increases in posterior parietal lobe; R > L <sup>130</sup>
Clock-Drawing Test	Visual neglect, right parietal dysfunction
Hooper Visual Organization Test	Visual perceptual fragmentation from bilateral posterior brain dysfunction or right frontal dysfunction
Object Assembly Test (WAIS)	Constructional ability, visuospatial perception; posterior brain R > L
Visual Form Discrimination Test	Visual recognition, posterior brain injury, particularly left parietal lobe

*Note:* rCBF = regional cerebral blood flow.

within an average range, whereas those patients with right hemisphere damage are more likely to provide impaired scores, particularly if they have posterior lesions.

Poor performance on this test can be caused by impaired visual acuity, psychiatric disorder, significant pain, impairment of visual attention, and fatigue.<sup>12</sup> This test may not detect brain damage located in the left hemisphere, and it requires the administration of other neuropsychological tests to improve the overall neuropsychological screening.

### *Block Design Test*

This test consists of assembling 1-in. blocks with red and white colors to reproduce a specific printed design from a stimulus card. The task may require the use of four to nine blocks. It is one of the performance subtests of the Wechsler Adult Intelligence Scales. It is a timed test, and each design becomes more difficult than the prior design.<sup>133</sup> This test is generally recognized as the best measure of visuospatial organization within the Wechsler Scales.<sup>3</sup> It reflects a general ability in most individuals so that cognitively capable persons who are academically or culturally limited will frequently obtain their highest score among the 11 subtests. However, *Block Design* scores tend to be lower in the presence of any kind of brain dysfunction. It is particularly sensitive in detection when the injury is located in the frontal or parietal lobes. In normal subjects, *Block Design* performance is associated with an increased glucose metabolism in the posterior parietal regions when measured by PET scan. Generally, the more intense metabolic activation is in the right cerebral hemisphere.<sup>130</sup>

Edith Kaplan argues that the examiner should note whether lateralized errors on this test tend to occur more at the top or the bottom of the constructions, as the upper visual fields have a temporal lobe component, whereas the lower visual fields have parietal components. Thus, a pattern of errors clustering at the top or a bottom corner can give some indication of the anatomical site and extent of the lesion.<sup>3</sup> By taking a qualitative rather than a quantitative approach to *Block Design* analysis, other information may be detected. For instance, patients with left hemisphere, particularly parietal, lesions tend to show confusion and simplification while handling the design in a concrete fashion. However, their approach to the designs is likely to be orderly; they typically work from left to right, as do intact subjects, and their construction usually preserves the square shape of the design. However, their greatest difficulty may be in placing the last block, which most often will be on their right. On the other hand, patients with right-sided lesions may begin at the right of the design and work to their left. The visuospatial defect reveals itself in disorientation, design distortions,

and misperceptions. Left visuospatial inattention may compound this design-copying problem, resulting in two- or three-block solutions to the four-block designs.<sup>3</sup>

#### *Hooper Visual Organization Test*

The *Hooper Visual Organization Test* consists of showing the patient 30 pictures of objects that have been cut up and placed in different positions.<sup>134</sup> The patient must visually examine each picture and then decide what it would represent if it were assembled. The patient must write down the name of the object, such as a fish, ball, or key. Most individuals can complete this test in approximately 15 min.<sup>12</sup>

Cognitively intact persons generally fail no more than six items on this test. More than 11 failures usually indicates organic brain pathology. The test appears sensitive to bilateral posterior brain dysfunction or, in some instances, dysfunction of the right frontal lobe. These patients tend to examine only one object singly rather than visually organize the different objects into a cohesive visual organization. Poor performance on this test also can be caused by low intellectual ability, psychiatric disease, or poor effort.

#### *Object Assembly Test*

The *Object Assembly Test* is another subtest of the WAIS.<sup>133</sup> It requires the patient to assemble cardboard figures of familiar objects. There are timed portions to this test, and the patient must form the puzzle parts into a man, a face profile, an elephant, a house, and a butterfly. The patient is not told the name or nature of the object and must identify the object during the assembly process.

The speed component of this test renders it relatively vulnerable to brain damage generally.<sup>3</sup> It tests constructional ability and visuospatial perception and is sensitive to posterior brain lesions, more so on the right side than the left. In terms of internal correlations on the WAIS, the Object Assembly and Block Design tests correlate more highly with one another than do any of the other Wechsler subscale tests.

Patients who have posterior right hemisphere damage typically will perform poorly on this test, and patients with frontal lobe injuries may show poor organization and planning skills in their approach to the test. If the brain injury is significant, the patient may not comprehend the test instructions and possibly could require extra examples, such as described in the test manual.

#### *Visual Form Discrimination Test*

This test consists of a series of three geometric figures that the patient must match to one of four sets of designs.<sup>135</sup> It is a multiple-choice test of visual recognition. Of the four sets of designs, one of the designs is an exact replica of the stimulus figure, while the others may vary to a subtle degree. This is a visual recognition test, and it is sensitive to posterior brain injury, particularly in the left parietal lobe. One of its strengths is that it can be administered to patients who are unable to speak English, as the patient only must point to one of four sets of figures on a sheet of cardboard. Visual memory plays little role in this test. A number of factors may interfere with test performance. These include impaired visual acuity, psychiatric disturbances, visual field defects, and poor motivation. Poor performance on this test alone may be sufficient to provide gross evidence of brain injury.<sup>12</sup>

## **MEASURING SENSORIMOTOR FUNCTION**

### **The Neuroanatomical and Neuroimaging Bases of Sensorimotor Function**

Sensorimotor functions are usually a portion of the cognitive examination. However, their primary role in the assessment of cognition lies in their ability to provide lateralized analysis of the cortex. Therefore, in general, they usually are not given the same weighting or attention in a cognitive examination as the domains of attention, memory, language, visuooperceptual, and executive function. The superior parietal lobule is a major source of projections to the dorsal premotor cortex,

**TABLE 6.15**  
**The Neuroanatomy of Sensorimotor Function**

Function	Purported Location
Coordination of complex movements	Superior parietal lobule projecting to dorsal premotor cortex <sup>136</sup>
Touch localization and active manual exploration	Brodmann's areas 1 and 2, Brodmann's areas 5, 7, and 40, and posterior insula <sup>137</sup>
Complex movement and modulation of sensory guidance, initiation, planning, and learning of complex movement	Premotor area (in Brodmann's area 6), frontal eye fields (in area 6), supplementary motor area (in area 6), supplementary motor area (posterior part of Brodmann's area 44 and perhaps part of Brodmann's area 8) <sup>137,140</sup>
Mental rehearsal of movements	Supplemental motor area <sup>139</sup>

and these play an important role within the coordination of complex movements.<sup>136</sup> The primary somatosensory cortex lies in Brodmann's areas 1 and 2 (also called S1 and S2). The somatosensory association cortex lies within Brodmann's areas 5, 7, and probably also the anterior portion of Brodmann's area 40. The posterior insula is often included in this association cortex as well. The somatosensory association cortex in the human brain plays an essential role in the finer aspects of touch localization and active manual exploration (such as with the *Tactual Performance Test* of the Halstead–Reitan Battery). The somatosensory coordination of reaching and grasping and the encoding of complex somatosensory memories are subserved also.<sup>137</sup>

In the human, an S2 area has been located in a region of the parietal operculum adjacent to the dorsal insula. Functional brain imaging of this area suggests that S2 may participate in pain perception. In some patients, lesions in the region of S2 give rise to a loss of pain perception without impairing discrimination of the other somatosensory modalities. For instance, a thalamic lesion will impair all sensory modalities and a lesion at S1 causes reversed association (loss of discriminative somatosensory modalities without a loss of pain perception). In motor association, areas anterior to M1 project into M1. This premotor cortex contributes a substantial portion of descending corticospinal and corticobulbar fibers, but these are at a lower density than those derived from M1.<sup>138</sup> Lesions in the motor association area produce complex deficits in movement without weakness, dystonia, dysmetria, or hyperreflexia.

In the human, the motor association cortex includes the premotor area (within Brodmann's area 6), the frontal eye fields in Brodmann's area 6, the supplementary motor area in the medial wall of the cerebral hemisphere (mostly in Brodmann's area 6), the supplementary eye fields, the posterior parts of Broca's area (Brodmann's area 44), and perhaps parts of Brodmann's area 8.<sup>137</sup>

Finger movements lead to activation of M1 as well as the supplementary motor area. If the patient imagines movements, the supplementary motor area is primarily activated.<sup>139</sup> The supplementary motor areas of the cortex and the premotor cortex are thought to play important roles in motor planning and response selection. These areas may also play a critical role in the initiation of motor responses and the ability to sustain motor output. Components of the motor association cortex modulate the sensory guidance, initiation, inhibition, planning, and learning of complex movements.<sup>140</sup> [Table 6.15](#) details sensorimotor anatomy.

## The Neuropsychological Measurement of Sensorimotor Function

### *Finger Tapping Test*

The *Finger Tapping Test* is a measure of motor speed and is one of the components of the Halstead–Reitan Battery. It was originally developed by Halstead and improved by Reitan and Wolfson.<sup>72</sup> This is probably the most widely used test of manual dexterity. It consists of tapping a

key with a device that records the number of taps. The score for each hand is the average of five trials. Traumatic brain injury, if it produces motor slowing, often will have an adverse effect on finger-tapping rate. Lateralized lesions usually result in slowing of the tapping rate of the contralateral hand. There are norms for this test based on sex, age, and educational background.<sup>141</sup>

This test is sensitive to unilateral lesions, particularly in the posterior frontal lobes. However, it is sensitive to many conditions besides traumatic brain injury, including AIDS, Huntington's disease, Parkinson's disease, and other neurological or neurodegenerative disorders. It is also susceptible to false positives in severely depressed patients with psychomotor slowing or individuals on medications that produce motor slowing.

#### *Grip Strength Test*

The *Grip Strength Test* is also called the hand dynamometer test. It is used to assess grip strength in each hand.<sup>72</sup> It is a subtest within the Halstead–Reitan Battery. The test is based on the assumption that lateralized brain damage may affect strength of the contralateral hand. It is easily administered in approximately 5 min. However, this is a very effort-dependent test, and there is no method for determining validity. It can be consciously manipulated. Moreover, persons who have orthopedic injuries (e.g., cervical radiculopathy or carpal tunnel syndrome) or arthritis in the hands may perform poorly on this test. It is not a test used alone to detect brain injury or lateralized injury. It is performed with a dynamometer, and the force exerted in kilograms for each hand is averaged for two trials. One generally expects a 10% difference in strength between hands in normal persons, with the dominant hand showing the superior strength.

#### *Grooved Pegboard Test*

This test is a subtest within the *Wisconsin Neuropsychological Test Battery*. It was developed by Kløve in 1963.<sup>142</sup> The test consists of a small board that contains a 5 × 5 set of slotted holes. These function like keyholes, and each peg has a key ridge along one side that requires it to be rotated into position before it may be inserted. It is actually quite a complex test, which makes it very sensitive for measuring general slowing, whether it is due to medication, neurodegenerative disease, Parkinsonism, or other disorders. It can aid in identifying lateralized impairment. The method of scoring is based on the time to completion of the test. Generally, both hands are tested, but only one hand may be used if the examiner only wishes to know about motor speed. If measurements of lateralization of brain injury are required, both hands should be tested. Norms are available for both hands.<sup>3,207</sup>

#### *Finger Localization and Fingertip Number Writing Test*

This is a subtest of the Halstead–Reitan Battery and is part of the *Sensory-Perceptual Examination*. The finger localization portion of this test is a measure of finger agnosia. It is administered by blindfolding the patient and touching her fingers. There is a standardized format for touching fingers, and then the patient must report the name and number of each finger as it is touched. In the fingertip-writing portion, the examiner writes the numbers 3, 4, 5, or 6 in a standardized order, again with the patient blindfolded, until a total of 20 numbers have been written on the fingertips of each hand. The patient must identify which number the examiner has written. A significant number of errors is consistent with sensory impairment of either the peripheral nerves to the fingers or the contralateral parietal lobe. In the examination of a brain-injured patient, assuming peripheral nerve function is intact, this test will identify contralateral parietal lobe dysfunction.<sup>72,143</sup>

#### *Sensory-Perceptual Examination*

This test is a component of the Halstead–Reitan Test Battery.<sup>72</sup> It contains a number of clinical tests to determine tactile stimulation and possible suppression, auditory stimulation and possible suppression, and the visual fields. In the tactile perception test, the patient's hands are placed on a table in front of the examiner with the palms down. The eyes are closed or blindfolded, and the examiner touches either the back of each hand or both hands lightly in a random sequence. After

**TABLE 6.16**  
**Tests of Sensorimotor Function**

Test	Measurement
	<b>Motor</b>
Finger Tapping Test	Manual dexterity and finger motor speed
Grip Strength Test	Lateralized difference in hand strength
Grooved Pegboard Test	Fine motor coordination and manual dexterity
	<b>Sensory</b>
Finger Localization and Fingertip Number Writing Tests	Finger agnosia, fingertip number perception (parietal lobes)
Sensory-Perceptual Examination	Perception of tactile sensation, tactile inattention, auditory suppression, and visual fields

each side has been examined, the examiner then touches either the hand, face, or both hand and face simultaneously and asks the patient to indicate which side was touched. If the patient gives evidence of a suppression error, this suggests a contralateral brain injury.

A similar procedure is used for assessing perception of auditory stimuli. The examiner stands directly behind the patient who has his eyes closed or is blindfolded. A small noise is produced by rubbing the fingers together approximately 6 in. from the patient's left or right ear. This is done for each side to determine if the patient can perceive the auditory stimulus. Following this, the examiner simultaneously rubs the fingers of both hands together near both of the patient's ears, interspersed with auditory stimuli on solely the right or left. If the patient consistently fails to identify the sound arriving at one of the ears on the bilateral stimulation trials, then it is likely that a suppression of the sound in that ear has occurred as a result of injury to the contralateral hemisphere. See [Table 6.16](#) for a listing of commonly used sensorimotor tests.

The last portion of the test includes visual field examination. The examiner sits approximately 4 ft in front of the patient and stretches her arms while the patient's eyes are focused directly on the examiner's nose. The examiner then instructs her to indicate whether she notices anything moving at the periphery of the visual field while focus is maintained upon the examiner's nose. The upper, middle, and lower visual fields are tested while the examiner makes slight movements with her fingers. This examination is performed separately for each side. Interspersed with these unilateral stimulation trials, the examiner makes simultaneous movements of the fingers on both hands, again in the upper, middle, and lower visual fields, to evaluate for suppressions. Mostly, this test proceeds in the same fashion as that which physicians normally use for confrontational visual field testing.

## MEASURING EXECUTIVE FUNCTION

### The Neuroanatomical Bases of Executive Frontal Lobe Function

In [Chapter 2](#), we examined multiple frontal lobe syndromes. However, the concept of executive function is far beyond mere frontal lobe behavior. We also learned in [Chapter 4](#) that executive functions are viewed differently by physicians and neuropsychologists. We have seen that neuropsychologist Lezak conceptualizes four components of executive function: (1) volition, (2) planning, (3) proposive action, and (4) effective performance.<sup>3</sup> Mesulam,<sup>35</sup> a behavioral neurologist, divides the human frontal lobes into three functional sectors:

1. The premotor sector, which includes Brodmann's areas 4 and 6, the supplemental motor area, the frontal eye fields, the supplemental eye fields, and parts of Broca's area. Damage



**TABLE 6.17**  
**The Neuroanatomy of Executive Function**

Function	Purported Location
Bind thoughts, memories, and experiences with visceral and emotional feelings	Orbitofrontal cortex and paralimbic structures (anterior cingulate, paraolfactory gyrus, and ventral and medial frontal lobe) <sup>35</sup>
Working memory	Dorsolateral prefrontal cortex <sup>37</sup>
Response inhibition, flexibility, foresight, and planning	Prefrontal cortex <sup>35</sup>

to this component of the frontal lobes results in weakness, alteration of muscle tone, release of grasp reflexes, incontinence, akinesia, mutism, aprosodia, apraxia, and some motor components of unilateral neglect and Broca's aphasia.

2. The paralimbic sector, which is located in the ventral and medial parts of the frontal lobe and contains portions of the anterior cingulate complex (Brodmann's areas 23 and 32), the paraolfactory gyrus (Brodmann's area 25), and the posterior orbitofrontal region (Brodmann's areas 11, 12, and 13).
3. The heteromodal sector, which contains Brodmann's areas 9 and 10, the anterior portions of Brodmann's areas 11 and 12, and Brodmann's areas 45 and 47. This region receives inputs from all the sensory modalities and from all other heteromodal regions of the brain.

Mesulam further suggests that the frontal cortex is so heterogeneous with respect to structure, connectivity, and physiology that no single descriptor can account for its multiple behavioral functions. It is noteworthy that even massive damage to the prefrontal cortex generally leaves sensation, perception, movement, and homeostatic functions intact. Within the executive relays of the frontal lobe, through its widespread connections, are functional anatomic areas to activate a given network, suppress another network, or orchestrate interactions between networks. The prefrontal cortex plays an important role in inhibiting impulses that are not appropriate for the context and also functions in disengaging stimuli and customary responses in order that alternative responses may proceed to promote flexibility, foresight, and planning.

Many neurons in the prefrontal cortex respond to visual input. However, they seem to have no specificity for color, size, orientation, or movement, but they do have significant behavioral relevance for the visual stimulus.<sup>144</sup> By exploring working memory, it appears that the prefrontal cortex can transform information access from a sequential process, where only one item of data can be managed at a given time, to another pattern where multiple items of data become concurrently accessible (parallel processing).<sup>145</sup> If function allows the focus of attention to move from one to another, a number of variables can be attended and processed simultaneously. It is argued that when these functions are disrupted, mental impairment results, with loss of foresight, strategic thinking, and inability to manage risk.<sup>146</sup> The orbitofrontal cortex in association with other paralimbic components of the frontal lobe enables a person to bind his thoughts, memories, and experiences with visceral and emotional feelings. Damage to this component of frontal lobe function interferes with the ability of emotion and visceral state to guide behavior, especially in the complex and ambiguous situations involving human interaction. The complex neuroanatomical relationships of the frontal lobe are beyond the scope of this text, and the reader is referred to Mesulam or Stuss and Benson for a more definitive and complex overview of frontal function and executive control.<sup>35,155</sup> [Table 6.17](#) reviews executive neuroanatomy.

### The Neuropsychological Measurement of Executive Function

In [Chapter 2](#), it was learned that frontal lobe injury is the most common site of anatomical change following traumatic brain injury. Even nontraumatic brain injury often results in significant changes

**TABLE 6.18**  
**Tests of Executive Function**

Test	Measurement
Behavioral Assessment of the Dysexecutive Syndrome	Measures real-world executive abilities in a more ecologically valid manner
Category Test	Ability to formulate abstract principles based on receiving feedback
Wisconsin Card Sorting Test	Problem-solving skills, cognitive flexibility, ability to maintain conceptual set and concept formation

in executive function.<sup>147</sup> Not only does one see dysfunction of the elements pointed out by Lezak and Mesulam as discussed previously, but alterations of discourse in brain-injured adults are seen as well. In fact, a significant correlation has been noted between scores from the *Wisconsin Card Sorting Test* and measurements of story structure during discourse.<sup>148</sup> Without measures of executive function, it is often difficult to determine the level of cognitive injury a patient has received. For instance, the *Glasgow Outcome Scale* does not detect as many as 25% of patients with severe executive dysfunction following traumatic brain injury<sup>149</sup> (see [Table 6.18](#)).

#### *Category Test*

The *Category Test* is used in the Halstead–Reitan Test Battery.<sup>150</sup> Lezak<sup>3</sup> describes this as a test of abstracting ability. It consists of 208 visually presented items in six sets. Each set is organized on the basis of different principles. From all the tests in Halstead’s battery, this test is considered the most sensitive to the presence of brain damage, regardless of its nature or location. A reevaluation of Halstead’s original data indicates that while the *Category Test*’s greatest sensitivity is to left frontal lesions, in some cases, 35 to 40% of nonfrontal patients also performed abnormally.<sup>151</sup> This test is quite sensitive for detecting brain damage in the frontal lobes with variable specificity. It requires 30 min to 1 h to administer. Severely brain-damaged persons may require longer times. There appears to be considerable variability in the performance of healthy normal controls on this test. This suggests that false positive errors can occur.<sup>12</sup>

#### *Wisconsin Card Sorting Test*

This test was originally developed by Berg<sup>152</sup> and later revised by Heaton et al.<sup>153</sup> There is little question when administering this test that in patients with frontal lobe damage, the frontal patients will make more perseverative errors.<sup>154</sup> The current version of this test consists of 128 cards containing one to four symbols (triangle, star, cross, and/or circle), which are printed in one of four colors (red, green, yellow, or blue). The examiner places four cards in a horizontal array in front of the patient. The patient must match the top card in a pack of 64 cards by placing it directly below one of the four cards lying above. Only minimal instructions are given to the patient, as the preface of the test is to determine if the patient can deduce the underlying sorting principle based on color, form, or number. The patient is given a maximum of 128 cards in which to complete six categories. After the patient has made ten consecutive correct responses, the underlying category automatically changes and the patient is expected to deduce the change. Error scores are kept and perseverative responses are noted.

This test has been shown to be sensitive to dorsolateral lesions in the frontal lobes, but it is relatively insensitive to orbitofrontal lesions.<sup>155</sup> Similar to the *Category Test*, patients with diffuse brain damage may perform as poorly on this test as patients with frontal lobe pathology. However, the *Wisconsin Card Sorting Test* is widely used in PET studies to measure frontal function. The manual contains norms for normal controls, patients with frontal lobe pathology, patients with brain injuries that do not include the frontal lobes, and patients with diffuse brain damage, so the examiner can make some discrimination. Many patients with posttraumatic orbitofrontal syndromes fre-

quently perform well on this test. Poor performance can be caused by visual impairment, color blindness, visual-perceptual difficulties, impaired hearing, psychiatric disease, and poor effort or malingering.<sup>12</sup>

## MEASURING INTELLECTUAL FUNCTIONING

Within the assessment of cognition, particularly when the neuropsychiatric examiner is requesting neuropsychological assessment, it is wise to keep in mind that intellectual assessment alone cannot determine the presence or absence of traumatic brain injury. Verbal IQ, performance IQ, and full-scale IQ have limited predictive ability within the assessment of brain injury. On the other hand, certain patterns within the subscales of intellectual assessment, for instance, with the WAIS-III, may provide useful information in the analysis of cognitive changes following brain injury.

There is no known neuroanatomical site for what is termed *test intelligence*. Moreover, it is not possible at this time to perform brain imaging to determine a location of intelligence. Test intelligence is usually measured by batteries that contain multiple subtests. Therefore, there is no single test instrument capable of comprehensively measuring human intellect. Early in the development of psychology, intelligence was viewed as a unitary capacity. David Wechsler conceived the Wechsler Intelligence Scales as one test with many parts; thus, IQ tests are individually administered test batteries. The calculated IQ scores themselves, however, have no functional utility in neuropsychological prediction.<sup>3</sup> The time required to test individuals with intellectual assessment instruments varies inversely with the severity of injury and directly with the level of intellect. In other words, persons of low intelligence complete fewer items of testing and require shorter test times, and in general, the same can be said for those with severe brain injuries. Table 6.19 lists common adult tests of intelligence.

### Kaufman's Brief Test of Intelligence

*Kaufman's Brief Test of Intelligence* (KBIT) is an individually administered intelligence test for persons whose ages range from 4 to 90. It is useful for assessing verbal and nonverbal abilities.<sup>156</sup> The *Vocabulary* subtest is broken into expressive vocabulary and definitions. Nonverbal abilities are assessed by the *Matrices* subtest, which consists of items involving visual stimuli that require the person being tested to determine the relationship between the stimuli using a multiple-choice format. This test is quick to administer and requires 15 to 30 min, depending on the age, intelligence capacity, and impairment level of the person being tested. Individual subtest scores are converted to standard scores with a mean of 100 and a standard deviation of 15 for both the Vocabulary and Matrices subtests. A composite IQ score is then calculated. There are tables within the manual to enable the examiner to compare the individual's performance on the Vocabulary and Matrices subtests to determine if any differences between the two are statistically significant.

The norms for this test come from a sample of 2022 individuals and were stratified according to U.S. Census data on or about 1990. These data included four variables: gender, geographic region, socioeconomic status, and race or ethnic group. For certain brain-injured patients, this test of intelligence offers an advantage over others. Unlike the Wechsler Scales, it does not require a

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**TABLE 6.19**  
**Tests to Assess Intelligence in Adults**

- Kaufman Brief Test of Intelligence (KBIT)
  - Raven Progressive Matrices Test
  - Test of Nonverbal Intelligence (TONI)
  - Wechsler Adult Intelligence Scale-III (WAIS-III)
-

motor response from the patient. Thus, it is well suited for determining intelligence in brain-injured persons who are physically handicapped or have significant motoric limitations of the dominant extremity. The main limitation of this test instrument is that it provides less of a differentiation between verbal and nonverbal intellectual functions than the Wechsler Scales. It may also produce a spuriously low estimate of verbal intelligence in some persons.<sup>110,157</sup>

### **Raven Progressive Matrices Test**

The *Raven Progressive Matrices Test* was originally developed in England, but it has been used widely in the U.S., as well as many other countries throughout the world, since it is essentially language-free. This test does not require the patient to perform skilled movements or to verbalize responses, but simply to point. Therefore, it can be used to assess persons whose cultural or language background would be disadvantageous if they were administered the Wechsler Intelligence Scales. It also can be administered to individuals with significant motor limitations or those who are hearing impaired.<sup>158</sup>

This test serves to measure inductive reasoning, and it requires the patient to conceptualize spatial, design, and numerical relationships. There are three forms of the test: standard, colored, and advanced. The standard version consists of 60 items, which are grouped in five sets. The patient is to select the correct pattern from either six or eight pictures. Spreen and Strauss<sup>110</sup> find this test particularly useful for persons who are poorly fluent in English or in those who do not understand English. They have also used this test for those who are aphasic or have cerebral palsy. Therefore, while it is not a first-choice test for measuring intellectual functioning, in the severely impaired brain-injured patient, it may be a best second choice.

While this test assesses mainly nonverbal and visuospatial problem-solving skills, the more difficult items contain mathematical concepts that involve analytic reasoning required by the left cerebral hemisphere. Persons with right-sided brain lesions are more likely to show poor performance on the visuospatial tasks, whereas patients with left hemisphere injuries may have greater difficulty with the analytical reasoning portion of the test. This test is not recommended for discriminating right from left brain damage in patients or for assessing individual visuospatial abilities.<sup>3</sup>

### **Test of Nonverbal Intelligence**

The *Test of Nonverbal Intelligence* (TONI) is a language-free measure of abstract problem-solving skills.<sup>159</sup> It is normed for persons ranging from 5 to 85 years. Similar to the Raven Test, it is an untimed test and requires approximately 15 min to administer. The format for administration is completely free of language. No listening, speaking, reading, or writing is required, and the person needs to make only a minimal motor response to the test items.

This test was specifically designed to measure intellectual functioning in individuals who are not functional in English and in those persons who have been raised in non-American cultures. Therefore, when assessing traumatic brain injury in immigrant persons, this may be the preferred intellectual test instrument relative to Kaufman's Brief Test of Intelligence or the Wechsler Intelligence Scales. During testing, the person attempts to identify relationships among abstract figures and then solve problems created by the cognitive manipulation of these relationships. The person must complete patterns by selecting correct responses from among four or six alternatives. The test items contain characteristics of shapes, direction, contiguity, position, rotation, shading, size, figure patterns, links, and movement.<sup>159</sup> The difficulty of test items is increased as the person progresses through the testing. The person must identify the rule or rules that are operating among the figures and thereby select appropriate responses. There are two forms for this test (TONI-1 and TONI-2), and they are useful in situations where the person must be retested at a later date. This, of course, avoids test-retest issues.

Obviously a major strength of this test is its ability to evaluate brain-injured persons for whom the Wechsler Intelligence Scales are not appropriate. It can be administered to brain-injured persons

who have language dysfunction, hearing impairment, poor English skills, or cultural differences. It may be difficult for patients who have significant visual impairment. Thus, a patient who has a visual field cut or a neglect syndrome may not be appropriate for this examination. Moreover, it will not provide a measure of verbal skill, and its ability to measure intellectual functioning is not equivalent to the Wechsler Scales.<sup>12</sup>

### **Wechsler Adult Intelligence Scale-III**

The WAIS-III<sup>133</sup> is the most recent revision of the *Wechsler Intelligence Scale-Revised*. This instrument contains 14 subtests. Eleven of these were retained from the Wechsler Adult Intelligence Scale-Revised. The *Symbol Search Scale* was adapted from the *Wechsler Intelligence Scale for Children-III* (WISC-III). Two new subtests were added: *Matrix Reasoning* and *Letter-Number Sequencing*.

Three functionally distinct factors have consistently emerged in research on all of the published forms of the Wechsler Scales. The first is a verbal factor, usually called verbal comprehension, and it has its highest statistical weightings on the Information, Comprehension, Similarities, and Vocabulary subscales. A second factor, the perceptual organization factor, always statistically loads on the Block Design and Object Assembly subscales, and it statistically contributes some to the *Digit Symbol* subtest and sometimes the *Picture Completion* or *Picture Arrangement* subtests. The third factor, a memory or freedom from distractibility factor, weights significantly on the Arithmetic and Digit Span subscales, and to some extent on the Digit Symbol subscale.<sup>3</sup>

There is some general tendency for verbal scale IQ scores to be reduced relative to performance scale IQ scores when the injury is predominantly or only in the left hemisphere. However, this decline does not occur regularly enough, nor is it typically large enough, for reliable distinctions or predictions to be made.<sup>160</sup> A lower performance scale IQ score is even less useful as an indicator of right hemisphere damage due to the time-dependent requirements of completing the performance scales. Thus, these scales are sensitive to any cerebral disorder that impairs the brain's efficiency, as they call upon more unfamiliar activities than the subtests within the verbal scale test. Confounding reduction of the performance scale IQ score can occur with patients having extensive right hemisphere damage, left hemisphere lesions, bilateral brain damage, certain neurodegenerative disorders, and the cognitive disorders associated with depression.<sup>160</sup> Moreover, a person's inherent intellectual capacity plays a role in the verbal-performance differences, if any. There is a strong tendency for verbal scale IQ scores to be relatively high in those persons whose full-scale IQ scores are in the superior or higher range. This tendency is reversed in favor of higher performance scale IQ scores in those persons whose full-scale IQ scores are below 100.<sup>161</sup>

The WAIS-III contains new index scores that were not present in the prior forms of the Wechsler Scales. These index scores are developed for verbal comprehension, perceptual organization, working memory, and processing speed. The *Verbal Comprehension Index* is composed of the Vocabulary, Similarities, and Information subtests. The *Perceptual Organization Index* is composed of the Picture Completion, Block Design, and Matrix Reasoning subtests. The *Working Memory Index* is composed of the Arithmetic, Digit Span, and Letter-Number Sequencing subtests. The *Processing Speed Index* is based on the Digit Symbol-Coding and Symbol Search subtests.<sup>133</sup>

The WAIS-III has norms for ages 16 to 89 years. This is a substantial lengthening of the upper age limit from the WAIS-R, which includes norms only to age 74. This test contains a powerful and useful function in the assessment of traumatic brain injury in that it is specifically designed to be used in conjunction with the WMS-III. Moreover, from a cultural standpoint, for each age group in the standardization samples of 2450 adults, the proportions of Caucasians, African-Americans, Hispanics, Asians, and Native Americans is based on those same proportions of individuals within each age group of the U.S. population using 1995 Census data. The normative samples also were stratified by educational background ranging from fewer than 8 years of education to greater than 16 years of education.<sup>133</sup>

The disadvantage of the WAIS-III, relative to the WAIS-R, is that the administration time of the third edition appears to have been increased by approximately 30 min. This, of course, is a result of increasing the number of subtests from 11 to 14. This test may require up to 2 h to administer, and it may be particularly difficult for patients who have significant traumatic brain injury, since their performance may deteriorate over time due to mental fatigue while they are taking the test. This test also may not be suitable for individuals with significant motor impairment or for those who have poor English skills. The test is very inflexible in administration requirements also. If a patient is fatigued or anxious during a subtest, a break cannot be taken, or it violates the manner in which the original test norms were obtained. Thus, it may not be particularly suitable for patients who have sustained significant brain damage affecting mental endurance or mood.<sup>12</sup> Moreover, in the standardization sample, 24% of normal individuals who were tested in the development of the WAIS-III had verbal and performance IQ scores that differed by 15 points or more (greater than 1 SD). Since a difference of greater than 1 SD can be found in approximately one of four normal individuals, these IQ scale differences should not be used to determine whether a patient has brain damage.<sup>12</sup> Also, when comparing this test with the WAIS-R, it should be remembered that full-scale IQ determined on the WAIS-III is 3 points less than full-scale IQ determined by the WAIS-R. Moreover, the verbal and performance IQs of the WAIS-III are 1.2 and 4.8 points less than the comparable WAIS-R verbal and performance IQs.<sup>133</sup> Standard scores are classified as very superior (>130), superior (120 to 129), high average (110 to 119), average (90 to 109), and low average (80 to 89).

## CHILD COGNITIVE ASSESSMENT

### MEASURING COGNITIVE DISTORTION

If the physician examiner were to review ordinary texts of pediatric neuropsychology, the issue of poor motivation or malingering is barely discussed, if at all. The general assumption is that young children will not malingering and will do their very best to perform optimally. However, motivation and effort clearly can be altered by the effects of medication, distracters in the environment, or malingering by proxy. This will be discussed in greater detail in the forensic section of this text. If a youngster is being examined for brain injury and the outcome of the examination is extremely important to the parents, covert and even overt signals may have been sent to the child before the examination. Should any questions arise regarding motivation, the VSVT is useful for determining motivation of children, as it is a test based upon probability theory.<sup>21</sup> [Table 6.20](#) lists the valid age ranges for tests commonly used to assess neuropsychological function in children.

### ESTABLISHING A PREINJURY COGNITIVE BASELINE

This may be a bit easier with children than with adults. For instance, children generally are attending educational institutions and the examiner can get access to school transcripts and academic achievement tests. Also, the high school-age child may have completed the ACT, PSAT, or SAT. The scores of these tests can be used to establish an estimated preinjury cognitive baseline if they were administered and completed prior to the time of injury. With a very young child, establishment of a preinjury cognitive baseline proves more difficult. However, by taking an achievement orientation, it is possible to test young children between the ages of 4 and 8 using the *Wechsler Individual Achievement Test-II* (WIAT-II).<sup>162</sup>

### Wechsler Individual Achievement Test-II

WIAT-II is a comprehensive, individually administered test useful for assessing the achievement of children, adolescents, college students, and adults. It is normed on persons aged 4 through adulthood. It was nationally standardized on 5586 individuals, and it uses normative information

**TABLE 6.20**  
**Neuropsychological Test Instruments for Children**

Test Category	Age Range (Years) <sup>a</sup>
Achievement tests (premorbid reading ability)	
Wide Range Achievement Test-III	5–74
Wechsler Individual Achievement Test-II	4–85
Attention tests	
Continuous Performance Test-II	5–90
Kiddie Continuous Performance Test	4–5
Memory tests	
Children’s Memory Scale	5–16
Wide Range Assessment of Memory and Learning	5–17
Language tests	
Boston Naming Test	4–13
Controlled Oral Word Association Test	6–90
Expressive Vocabulary Test	2½–90
Peabody Picture Vocabulary Test-III	2½–90
Token Test	6–13
Visuoperceptual tests	
Hooper Visual Organization Test	5–13
Rey Complex Figure Test	6–89
WISC-III Performance Scales (Block Design, Object Assembly, Picture Completion)	6–16
Sensorimotor tests	
Finger Tapping Test	5–7, 12–80
Grip Dynamometer Test	5–7, 12–80
Executive function tests	
Delis–Kaplan Executive Function System	8–89
Stroop Test	7–80
Trails for Children	8–15
Wisconsin Card Sorting Test	6–89
Intelligence function	
Cognitive Assessment System	5–17
Kaufman Brief Intelligence Test	4–90
Wechsler Intelligence Scale for Children-III	6–16
Wechsler Preschool and Primary Scale of Intelligence-III	2½–7½
Young child’s neuropsychological battery	
NEPSY	3–12

<sup>a</sup> Norms for various ages may be derived from sources other than the published testing manuals.<sup>110,141,207</sup>

based on age and grade. The *Reading* subtests are useful for prediction of preinjury ability. The WIAT-II is composed of four composite scales: Reading, Mathematics, Written Language, and Oral Language. The *Reading Composite Scale* consists of the subtests Word Reading, Reading Comprehension, and Pseudoword Decoding. The *Mathematics Composite Scale* contains the subtests Numerical Operations and Math Reasoning. The *Written Language Composite Scale* contains the subtests Spelling and Written Expression. The *Oral Language Composite Scale* contains the subtests Listening Comprehension and Oral Expression. The scores are presented as standard scores with a mean of 100 and a standard deviation of 15. The WIAT-II measures aspects of the learning process

that take place in the traditional academic setting in the areas of reading, writing, mathematics, and oral language.<sup>162</sup> Therefore, it should provide a reasonably accurate measure of information learned by children and adults prior to brain injury.

## MEASURING ATTENTION IN CHILDREN

Attentional complaints in children following brain injury are very common. However, the pediatric literature is quite limited, and objective measurements of child attention following brain injury are sparse. No childhood studies have provided a comprehensive assessment of attention based on current theoretical models.<sup>163</sup> Children who have sustained moderate to severe traumatic brain injury exhibit significant deficits for sustained and divided attention, and they demonstrate impaired response inhibition. However, they are often relatively intact in their ability to focus attention for the moment.<sup>164</sup> In children ages 3 to 8 years, those who have attentional deficits following brain injury may show a trend toward recovery of arousal and motivation over time. However, their focused attention, impulsivity, and hyperactivity may remain impaired. As noted in [Chapter 2](#), the younger the brain-injured patient at injury, the more likely there will be persisting deficits. This perhaps reflects a relative immaturity of attentional skills at the time of injury.<sup>165,166</sup> Other studies have demonstrated that the greater the severity of injury, the greater the deficit of sustained attention in children following brain injury. This difficulty may impact upon the future development of children as they develop skills dependent on intact attentional capacity.<sup>167</sup>

There have been comparisons made of traumatically brain-injured children with children who have attentional deficits associated with attention deficit hyperactivity disorder. Children with brain injuries were found to suffer from a general slowing of their information processing. This did not correlate with the inhibition deficit that is seen in attention deficit disorder. Thus, the slowing of information processing speed in children seems to be a general consequence of traumatic brain injury in childhood, whereas inhibitory deficits are generally not part of the traumatic brain injury pattern but are specific to attention deficit disorder.<sup>168</sup> However, secondary attention deficit disorder can develop following traumatic brain injury. Those cases seem to occur in youngsters who develop lesions in the right putamen following trauma to the brain.<sup>169</sup>

### Kiddie Continuous Performance Test

The *Conner's Continuous Performance Test-II* works well for youngsters 6 years old and above and, of course, is used in adults, as noted in the adult section above. However, it was determined that the 14-min duration of the CPT-II was problematic for youngsters ages 4 and 5 years. At that age, even children with no signs of attention deficits produced false positives. As a result, the *Kiddie Continuous Performance Test (K-CPT)* was set to run at 7½ min on a computer system.<sup>170</sup> This provided the necessary balance for 4- and 5-year-old youngsters. Moreover, the stimuli used on the K-CPT are a series of pictures that are readily familiar to children of a very young age. Whereas the CPT-II uses letters, these stimuli were inappropriate for very young children.

The K-CPT was specially designed to assist with the assessment of attention disorders in 4- and 5-year-old children. However, even though the K-CPT is appropriate for use with children ages 4 or 5 years, some children with severe cognitive impairment cannot complete this test instrument. If the child cannot understand the simple instructions, he or she is likely to perform poorly on the tasks regardless of whether attention problems are present.

This test is administered on a computer, and it uses a short practice test to familiarize the child with the procedures. Familiar pictures are projected onto the computer screen (e.g., sailboat, horse, scissors, soccer ball, etc.) rather than letters. The child is required to press the space bar or mouse whenever any picture except the soccer ball appears. The K-CPT measures include omission and commission errors, average reaction time, standard error of reaction time for hits, risk taking, perceptual sensitivity, and overall reaction time. Scores can be obtained immediately.



For the child 6 years of age and older, the Continuous Performance Test-II may be administered. The examiner should recall that the Ruff 2 and 7 Test is used for persons ages 16 years and older, and the Brief Test of Attention is used for persons ages 17 and older. The *Seashore Rhythm Test* of the Halstead–Reitan Battery could be used as an alternative to assess attention in persons ages 15 years and above.

## MEASURING MEMORY IN CHILDREN

Traumatic brain injury in children often results in memory deficits. The magnitude of these deficits has been thought to be dependent upon injury severity.<sup>163</sup> However, that dose–response relationship between severity of injury and memory deficit cannot be determined accurately early after injury. This relationship develops over time, with greater memory impairments evident for children with more severe traumatic brain injury by 12 months postinjury.<sup>171</sup> Thus, it is probably best to wait at least a year following brain injury in a child before attempts are made to determine the level of permanent memory impairment. Anderson and others took their data and continued the studies beyond 12 months and found that at 18 months postinjury, there continued to be a dose–response relationship between injury severity and memory dysfunction.<sup>172</sup> Another question of memory injury in young children is whether implicit memory is preserved.<sup>7</sup> Studies suggest that, as with adults, implicit memory (memory for skills and procedures) remains relatively unimpaired following traumatic brain injury, yet children who have sustained brain injury perform significantly poorer on memory measures than control groups.<sup>173</sup>

Many previous studies on brain-injured children have not described the specific memory deficit, as most tasks were not sophisticated enough to differentiate among the various types of memory disorders. Recent studies using the California Verbal Learning Test suggest that deficits occur in a variety of memory components in children, including storage, retention, and retrieval. At the present time, there are no standardized memory instruments other than the *NEPSY* for accurately measuring memory function in children below age 5 following traumatic brain injury.

### Children’s Memory Scale

The *Children’s Memory Scale* (CMS) is a comprehensive learning and memory assessment instrument designed to evaluate learning and memory functioning in individuals ages 5 through 16 years.<sup>174</sup> Nine CMS subtests are used to assess functioning in each of three domains: (1) auditory and verbal learning and memory, (2) visual and nonverbal learning and memory, and (3) attention and concentration. Each domain is assessed through two core subtests and one supplemental test. The core subtest battery can be administered in about 30 to 35 min. The supplemental battery takes an additional 10 to 15 min to administer. There is approximately a 30-min delay between the immediate memory and the delayed memory portion of each subtest. Many portions of the testing are further subdivided by age with three basic age levels: (1) ages 5 to 8, (2) ages 9 to 12, and (3) ages 13 to 16. Eight indices result from the administration of this test; they are presented as standard scores with a mean of 100 and a standard deviation of 15.

The *General Memory Index* globally measures memory function in much the same way that the full-scale IQ score of the WISC-III is viewed as a global measure of general intellectual ability. The *Attention/Concentration Index* assesses the ability to sustain and direct attention and concentration, processing speed, and working memory. The *Verbal Immediate Index* measures immediate and working memory span for auditory verbal material. The *Visual Immediate Index* measures immediate and working memory span for visual and nonverbal material. The *Verbal Delayed Index* measures the ability to consolidate, store, and retrieve newly learned auditory verbal material. The *Visual Delayed Index* assesses the ability to consolidate, store, and retrieve newly learned visual and nonverbal material. The *Delayed Recognition Index* enables one to determine whether impaired performance on the Verbal Delayed Index is the result of an encoding and storage deficit or a

retrieval deficit. The *Learning Index* is a summation of the child's performance across three learning trials of the Word Pairs (verbal) subtest and the Dot Locations (visual) subtest.

## Wide Range Assessment of Memory and Learning

The *Wide Range Assessment of Memory and Learning* (WRAML) allows the examiner to evaluate a child's ability to actively learn and memorize a variety of information.<sup>175</sup> The WRAML is normed for children ages 5 through 17 years. The structure of the test is based upon three major divisions. The first division makes a distinction between memory and learning. The second division evaluates competencies in both verbal and visual modalities. The third division evaluates delayed recall. There are three verbal, three visual, and three learning subtests that yield three indices: (1) *Verbal Memory Index*, (2) *Visual Memory Index*, and (3) *Learning Index*. When combined, the nine subtests yield a *General Memory Index*. Standard scores and percentiles are derived from the subtests and allow an age-based comparison of performance. The normative data are divided into two main age groups, children ages 5 to 8 and children ages 9 and older.

## MEASURING LANGUAGE IN CHILDREN

Language skills undergo rapid development during the early childhood years, and by the time children begin school, they are competent communicators with well-established syntactic, semantic, and pragmatic abilities for their age. However, as with most aspects of pediatric neuropsychology, little is known about the effects of traumatic brain injury on the acquisition of these language skills during the early childhood years. Morse and others<sup>176</sup> studied brain-injured children between 4 and 6 years of age. Their results indicated that children who had sustained severe brain injury performed most poorly among neuropsychological tests on language tasks. When receptive syntax ability alone is studied, brain-injured children perform significantly worse than controls on syntax comprehension.<sup>177</sup> Written language production seems to be impaired in children who are brain-injured as well. The output of written language is negatively correlated with severity of injury. The efficiency and completeness of language production by writing seem to be affected the greatest, and a moderate correlation is found between measures of written language and other neuropsychological functions.<sup>178</sup>

The development of pragmatic communication skills seems to be truncated in brain-injured children. Injuries sustained at an earlier age consistently predict poor performance on language tasks, complicating the ongoing development of generalized and higher-order communicative skills such as negotiating requests and hinting to others.<sup>179</sup> Deficits in pragmatic communication ability have a significant negative impact on functional outcome from traumatic brain injury, particularly during adolescence when sophisticated social communication skills are developing. Turkstra and others studied this hypothesis in adolescents and found that brain-injured adolescents had much more difficulty negotiating within language, hinting, describing simple procedures, and understanding sarcasm than their non-brain-injured controls.<sup>180</sup> In association with linguistic processing difficulties and errors of pragmatic language, reduced articulatory speed and increased pausing are often found in brain-injured children. This reduction in speaking rate may be present more than 1 year after traumatic brain injury.<sup>181</sup> The negative effects upon linguistic processing in head-injured children are best detected by examining the discourse of children. A consistent pattern of generally poor discourse is found among children injured below age 5. There is no evidence that lesion focus correlates with this finding.<sup>182</sup> During the exposition of a narrative story, children with traumatic brain injury are significantly more dysfluent than their age-matched controls, and this produces a striking burden upon the listener.<sup>183</sup> These discourse difficulties seem quite persistent. Ewing-Cobbs and others evaluated children 3 years after brain injury. These youngsters were 1 to 8 years of age at the time of their injuries, and 3 years later, the discourse deficiencies persisted and were most pronounced at the level of cognitive organization of the text. Moreover, these

youngsters produced fewer words and utterances than a group of siblings on a story retelling task, and their stories were characterized by fewer elements of meaning across sentences.<sup>184</sup> The non-dominant aspects of language seem equally impaired in brain-injured children. For instance, children who have sustained brain injury show less sensitivity than controls in how emotions are expressed within narratives. In particular, children are less able than controls to identify deceptive emotions within stories (dysprosody).<sup>185</sup>

The Boston Naming Test, COWA, and Token Test can be used to assess language in youngsters. The Boston Naming Test has norms for children as young as 5 years, and the COWA and Token Test have norms for ages as low as 6 years. These test instruments were discussed more fully in the above adult cognitive assessment section. Language testing of children below age 5 or 6 will be discussed next within the discussion for the NEPSY.

### **Expressive Vocabulary Test**

The *Expressive Vocabulary Test* (EVT) is an individually administered assessment of expressive vocabulary and word retrieval for children and adults ages 2½ through 90 years.<sup>187</sup> This test has been conormed with the *Peabody Picture Vocabulary Test-III* (PPVT-III).<sup>186</sup> The EVT measures expressive vocabulary knowledge with two types of items — labeling and synonyms. Word retrieval is evaluated by comparing expressive and receptive vocabulary skills using standard score differences between EVT and PPVT-III.<sup>187</sup> The conorming of the EVT and PPVT-III provides a very useful anterior and posterior language assessment in very young children and allows direct comparisons of expressive and receptive vocabulary.

The EVT is an untimed test that can be completed in about 15 min. The younger the child, generally the shorter the testing time. Examinees are administered only items that most closely approximate their ability levels. The EVT does not require the child to read or write or give a lengthy oral response. EVT results can be reported as standard scores (with a mean of 100 and a standard deviation of 15) that range from 40 to 160. These standard scores can allow comparisons to be made between EVT scores and scores earned on tests of oral language, academic achievement, and cognitive ability. If needed, EVT scores can be expressed as percentiles, normal curve equivalents, stanines, and test–age equivalents.

### **Peabody Picture Vocabulary Test-III**

This test is designed for persons aged 2½ through 90+ years. It serves two purposes: (1) as an achievement test of receptive (auditory) vocabulary attainment for standard English, and (2) as a screening test of verbal ability. It was standardized nationally on a stratified sample of 2725 persons, including 2000 children and adolescents. Raw scores can be converted to standard scores, percentiles, stanines, normal curve equivalents, and age equivalents.<sup>186</sup>

This test instrument is very easy to administer and is highly reliable, even at the youngest ages. It is extremely useful in testing preschool children. Because no reading or writing is required, it can be used in children who have written-language difficulty or impairment of the writing hand. For individuals with language impairments, particularly those with expressive vocabulary problems, it provides a measure of linguistic potential because it is a pure measure of receptive vocabulary. It may be used in children who are withdrawn or those who have significant cognitive impairment because there is no need to speak or interact verbally with the examiner. Even children who are hemiparetic and language impaired can be tested reliably with this instrument.

## **MEASURING VISUOPERCEPTUAL ABILITY IN CHILDREN**

Children who are brain-injured and sustain impairments in the visuoperceptual domains may also demonstrate weaknesses in spatial abilities, social judgment, or other nonverbal functions. Moreover, children may demonstrate weaknesses in the visuoperceptual area within the context of

relatively intact elementary verbal skills. Routine vision screening generally confirms that impairments in visual acuity or other primary sensory capacities are not present.<sup>163</sup>

The Block Design, Object Assembly, and Picture Arrangement subtests of the WISC-III may be used for assessing visuo-perceptual and visuo-spatial skills. The analogs of these tests used for adults have been discussed previously in the adult cognitive measurement section. The essential findings in adults are generally the same as in children. However, these WISC-III subtests have been specifically normed upon children, and the children's version should be used. The WISC-III is normed for measuring cognitive function in children ages 6 through 16 years, 11 months.

### **Hooper Visual Organization Test**

This test consists of showing children 30 pictures of objects that have been cut up and placed in different positions. Norms exist in order to assess children as young as age 5 years.<sup>134</sup> The child is required to visually examine each picture and decide what it would be if it were assembled and write down the name of the particular object, such as fish, ball, or key. Test items are arranged in increasing difficulty, and most children can complete the test in approximately 15 min. It is sensitive to posterior brain damage. Poor performance on this test can be due to poor visual acuity, low intellectual functioning, psychiatric disease, and poor effort.<sup>12</sup>

### **Rey–Osterrieth Complex Figure Test**

The *Rey–Osterrieth Complex Figure Test* consists of instructing the patient to copy a complex geometric figure onto a sheet of white paper. The amount of time taken initially to copy the figure is recorded. Standard procedures usually have the person draw the figure again from memory after a delay of 3 min and again after 30 min or 1 h. Norms are available on this test in order to measure children as young as 6 years. A scoring system was developed by Taylor that was adapted from the original work of Osterrieth.<sup>188</sup>

Traumatically brain-injured patients, including children, have difficulty on recall trials of the complex figure test. Even patients with mild head injuries may show significant deficits on 3-min recall trials within the first 2 years of injury. Moderately to severely injured patients have been shown to have impaired functioning more than 2 to 5 years after trauma. However, clearly there is a memory component to this test as well as a visuo-perceptual component, and visual memory is one element being measured, among others.<sup>3</sup>

This test has some discriminating ability for lesion location. Patients with posterior brain damage, particularly on the right side, are more likely to have problems with spatial organization, whereas patients with frontal lobe pathology are more likely to have difficulty in the planning and organization of their drawing. Patients with right hemisphere damage tend to perform more poorly on the recall section than patients with predominantly left hemisphere brain damage.<sup>12</sup> This test is easy to administer and score, but nonneurological etiologies can produce impaired scores.

### **Measuring Sensorimotor Function in Children**

Children, following traumatic brain injury, show alterations of both sensory and motor skills. However, there are very few research studies comparing traumatically brain-injured children with controls regarding their sensorimotor function. Moreover, norms on children are noticeably lacking. The Grip Strength and Finger Tapping Tests discussed above can be used in children ages 6 to 8 and ages 12 and older, if the norms of Spreen and Strauss are used.<sup>110</sup> However, as we will see next, the NEPSY can be used for sensorimotor function assessment in children ages 3 to 12.

Levin and Eisenberg<sup>189</sup> found that approximately 25% of children with severe traumatic brain injuries displayed deficits on tests of stereognosis, finger localization, and graphesthesia. Timed fine motor skills also seem degraded in youngsters following traumatic brain injury. In the studies

of Bawden et al.,<sup>190</sup> the performance of children with severe injuries declined proportionately as the demand for speed increased. As would be expected, children with mild and moderate injuries were less affected by demands for speed.

## MEASURING EXECUTIVE FUNCTION IN CHILDREN

As with adults, children who sustain traumatic brain injury, particularly the frontal parts, frequently demonstrate executive dysfunction. However, the studies of children with these disorders are minimal. Two tests noted above can be used in school-age children, as norms exist for their interpretation. The Wisconsin Card Sorting Test can be used to assess frontal function, particularly the dorsolateral brain areas, in children as young as 6 years. The Stroop Test has norms available for measuring response inhibition in children as young as 7 years. The *Delis–Kaplan Executive Function System* (D-KEFS) has norms beginning at age 8 for children.<sup>191</sup> For very young children, the NEPSY measures executive function in youngsters as young as 3 years of age.

Levin and others have found that children with traumatic brain injuries display deficits on various tasks meant to assess executive functions. These include the *Tower of London* (in the Delis–Kaplan test), which measures planning skills, and the *Controlled Oral Word Association Test*, which measures verbal fluency. The *Twenty Questions Test* (see the D-KEFS section next) measures concept formation and mental flexibility and has been used to assess executive function in children as well.<sup>192</sup> Levin's group also has measured the magnitude of deficits within executive function tasks and found a correlation with the volume of lesions in the frontal lobes, but very poor or no correlation with lesion volume outside frontal lobe areas when using tests specifically designed to measure planning skills, verbal fluency, concept formation, and mental flexibility.<sup>193</sup>

### Delis–Kaplan Executive Function System

The D-KEFS was standardized on a nationally representative stratified sample of 1750 children, adolescents, and adults, ages 8 to 89 years. Stratification was based on age, sex, race, ethnicity, years of education, and geographic region. The 2000 U.S. Census figures were used as target values for composition of the D-KEFS normative sample.<sup>191</sup> The D-KEFS consists of nine subtests, each of which may stand on its own merits independently: (1) Trailmaking Test, (2) Verbal Fluency Test, (3) Design Fluency Test, (4) Color–Word Interference Test, (5) Sorting Test, (6) Twenty Questions Test, (7) Word Context Test, (8) Tower Test, and (9) Proverb Test. Raw scores are converted to scaled scores, with a mean of 10 and an SD of 3.

The key objective of the D-KEFS is to provide psychologists with a larger and more diverse armamentarium of executive function tests for assessing the complex and multifactorial domain of cognition in a more comprehensive manner. The overall philosophy of this testing system uses three approaches: (1) relatively new tests that were developed by the authors, (2) modification of tasks that have been used previously in past experimental studies but not developed into standardized clinical instruments, and (3) modifications of existing clinical instruments. Historically, the Wisconsin Card Sorting Test has been the gold standard of executive function tests.<sup>153</sup> However, Kaplan<sup>208</sup> has argued that the use of a single-score method such as that used in the Wisconsin Card Sorting Test for quantifying performance on a cognitive instrument will mask the multiple natures of cognitive function that are required for successful performance. She argues that the single-score method is especially problematic with executive function tasks because such tests typically tap a host of fundamental and higher-level cognitive skills. This is purportedly avoided in the D-KEFS.

Particularly with children, the D-KEFS instruments measure several key components of executive function. These include (1) initiation of problem-solving behavior, (2) verbal concept-formation skills, (3) nonverbal concept-formation skills, (4) transfer of concepts into action, (5) abstract expression of conceptual relationships, (6) flexibility of thinking, and (7) flexibility of behavioral response.

## MEASURING INTELLECTUAL FUNCTIONING IN CHILDREN

Intellectual deficits are found in children who sustain traumatic brain injuries whether they are compared with normal controls or with children who have received orthopedic trauma not involving the head. The magnitude of the deficits is generally directly proportional to injury severity. IQ scores that reflect nonverbal skills, relative to verbal skills, are particularly likely to be depressed.<sup>163</sup> While it is not an inviolate finding, performance intelligence on standard IQ tests in children seems more vulnerable to change following head injury than the verbal portions. This dissociation probably reflects different demands of the two major IQ scales. Performance IQ subtests are more likely to require fluid problem-solving skills, and they generally involve speeded motor input and timed performance, whereas verbal IQ subtests are more likely to measure previously acquired verbal knowledge, and they make few demands for responses requiring speed or motor control.<sup>163</sup>

IQ scores tend to increase from an injury baseline over time following traumatic brain injury in children. The largest increases occur among children who are more severely injured. The greatest improvement in IQ scores is immediately after injury, and the scores tend to plateau after approximately 1 to 2 years. Improvements have been shown to occur for periods up to 5 years. However, even with substantial recovery, IQ scores often continue to be depressed relative to preinjury intelligence, particularly among children with severe injuries.<sup>194–196</sup> If it is necessary for the examiner to determine if there have been practice effects from prior intellectual assessments administered to the child, the current best reference for determining potential changes is found in the recent work by McCaffrey et al.<sup>206</sup>

### Cognitive Assessment System

The *Cognitive Assessment System* (CAS) has been used to evaluate children and adolescents with traumatic brain injury. Children with traumatic brain injury earned significantly lower scores in the domains of planning and attention than matched control groups. The results of studies using this test instrument are consistent with previous medical literature demonstrating poor performance on measures of attention and executive function among children who have experienced traumatic brain injury.<sup>197</sup> The Cognitive Assessment System is an assessment battery designed to evaluate cognitive processing in children ages 5 through 17 years. This test is based upon the PASS theory (planning, attention, simultaneous, and successive). These four processing areas of cognitive function comprise the four scales that make up the CAS.

The CAS has two forms: a standard battery and a basic battery. Each of the two forms is composed of planning, attention, simultaneous, and successive scales. In the standard battery, these scales are defined by three subtests each. In the basic battery, these scales are composed of two subtests each. Each subtest yields a scaled score with a mean of 10 and a standard deviation of 3, similar to that derived for the subtests of the Wechsler Intelligence Scale for Children-III (WISC-III). The subtest scaled scores within each PASS scale are combined to yield a standard score with a mean of 100 and a standard deviation of 15. The standard battery consists of 12 subtests, and the basic battery consists of 8 subtests; both yield a full-scale standard score that is derived from the sum of the subtest scaled scores.<sup>198</sup>

The *Planning* subtests contain three test components: (1) matching numbers, (2) planned codes, and (3) planned connections. The *Simultaneous* subtests contain three test components: (1) non-verbal matrices, (2) verbal-spatial relations, and (3) figure memory. The *Attention* subtests are composed of (1) expressive attention, (2) number detection, and (3) receptive attention. The *Successive* subtests contain four components: (1) word series, (2) sentence repetition, (3) speech rate that is normed for ages 5 to 7 only, and (4) sentence questions that are normed for ages 8 to 17 only.

The materials and instructions for each subtest are divided into age-appropriate item sets. Younger children (ages 5 to 7) are administered different item sets than are older children (ages 8

to 17). The test is very explicit in that it requires subtests to be administered as they were during the standardization data collection and in the order prescribed in the manual. Administering the tests out of order may invalidate the results. The logic for this is that the Planning subtests are administered first because they are the least structured, giving the child maximum latitude to solve them in any manner thought best. This is in contrast to the Attention subtests, which are highly structured and have instructions that impose considerable constraints on the child.

The standard scores from the CAS are presented in the same manner as deviation IQs are presented following administration of the WISC-III. The classifications of the descriptive categories are also the same as those of the WISC-III. For instance, an attained standard score of 130 and above is classified as very superior, whereas 120 to 129 is superior. High average classification is made for standard scores 110 to 119, average for scores 90 to 109, low average for scores 80 to 89, below average for scores 70 to 79, and well below average for scores 69 and below. The standardization sample percentiles for each classification range fit closely to the theoretical normal distribution.<sup>198</sup>

### **Wechsler Intelligence Scale for Children-III**

This is the third edition of the *Wechsler Scales for Children*. The psychometric standards for the Wechsler Scales probably exceed those of any other psychological test developed to date for the measurement of intellectual functioning in adults or children. It is an individually administered clinical instrument for assessing the intellectual ability of children ages 6 through 16 years, 11 months,<sup>199</sup> and it retains the essential features of the original WISC.<sup>200</sup>

The WISC-III includes changes in the test materials and administrative procedures from those of prior test editions. These have been introduced to make the testing experience more interesting to children. The pictorial stimulus materials are now printed in color, and the recommended order of administering the subtests has been changed so that the child's introduction to the testing situation takes place gradually and with less stress. Entirely new items have been added to replace dated ones and to replace items that analyses indicated were unfair to particular groups of children. The *Verbal* subtests are titled Information, Similarities, Arithmetic, Vocabulary, and Comprehension. The *Performance* subtests are titled Picture Completion, Coding, Picture Arrangement, Block Design, and Object Assembly. Two supplementary scales exist: the *Digit Span* subtest and the *Mazes* subtest. *Symbol Search* is a third subtest that may be interchanged for the *Coding* subtest if the examiner wishes. The supplementary subtests are not used to establish the norms for the verbal and performance IQs, and they are not needed to obtain these scores. The manual recommends that they may be administered when time permits and if the examiner wishes to obtain a richer representation of the child's abilities.<sup>199</sup> Digit Span may substitute for a Verbal subtest and Mazes for a Performance subtest, if one of the standard subtests is somehow invalidated or, for appropriate reasons, cannot be administered to the child.

In addition to the verbal, performance, and full-scale IQ scores, four factor-based index scores can be calculated: (1) verbal comprehension, (2) perceptual organization, (3) freedom from distractibility, and (4) processing speed. These factor-based scales, like the IQ scales, have a mean of 100 and a standard deviation of 15. The scores for the subtest scales have a standard deviation of 3 and a mean of 10 (exactly as the WAIS-III scores).

David Wechsler did not originally intend his scales to be used as neuropsychological instruments. However, they were found to be very useful and are integral parts of most neuropsychological evaluations of adults or children.<sup>201</sup> For instance, Kaplan and others<sup>202</sup> developed the WAIS-R as a neuropsychological instrument. They view the qualitative interpretations of test performance, analysis of errors, and testing of limits as important as or more important than the IQ scores themselves. Some neuropsychologists may use the WISC-III as a neuropsychological test instrument, but when that test is performed, the IQ scores are not used for assessing brain injury, but various subscale scores may be so used.

## MEASURING COGNITIVE INJURY IN THE VERY YOUNG CHILD

As noted earlier, the research base for neuropsychological assessment of traumatically brain-injured children is very weak relative to the databases available for adult patients. Moreover, the younger the child, the more sparse are the databases of assessment techniques. There has been a recent addition to the techniques available for measuring cognition of very young children. The NEPSY<sup>203</sup> was introduced in 1998.

The NEPSY is a comprehensive instrument that was designed to assess neuropsychological development in preschool and school-age children. The authors chose NEPSY as an acronym formed from the words *neuropsychology* and *psychology*. The subtests of this instrument are designed specifically for children between the ages of 3 and 12 years. Compared with other neuropsychological tests for children, the NEPSY is unique in that not only can it measure cognitive function of very young children, but the subtests were also standardized on a single sample of children and administered in conjunction with a number of other validity measures, including the *Wechsler Preschool and Primary Scale of Intelligence-Revised*, the WISC-III, and the WIAT. A broad range of subtests is included in the NEPSY to assess neuropsychological development in five functional domains: (1) attention/executive functions, (2) language functions, (3) sensorimotor functions, (4) visuospatial processing, and (5) memory and learning.

One of the major purposes for developing the NEPSY was to create an instrument that could be used for follow-up of children with congenital or acquired brain damage (including traumatic brain injury). Recovery of function in children who sustained traumatic brain injury needs to be evaluated over time in order to identify improving functioning, as well as persistent deficits that may require attention. Particularly in a psychoeducational framework, the NEPSY may be used to adapt interventions to the child's changing needs.

Much of the inspiration for the NEPSY was Luria's approach to assessing cognitive function in adults who had sustained brain damage.<sup>204</sup> Luria's work stimulated a Finnish version of the NEPSY developed in the 1980s.<sup>205</sup> The initial process of adapting the Finnish *NEPSY* for publication in the U.S. began in the spring of 1987. The U.S. pilot version was administered to 160 children in New York, New Jersey, Connecticut, and Pennsylvania during the fall of 1987. A tryout phase (1990 to 1994) was undertaken, and some subtests were eliminated while others were modified and new subtests were developed. The U.S. national tryout was undertaken in 1991–1992 and was administered to a sample of 300 children between the ages of 2 and 12. The sample was further stratified by race/ethnicity, gender, parent education, and geographical region. The review of these data established the age range for the present NEPSY at 3 to 12 years, and the subtests designed for 2-year-olds were eliminated. The standardization and validation phase was conducted by The Psychological Corporation from 1994 to 1996. The standardization version of the NEPSY was composed of 38 subtests and administered to 1500 children between the ages of 3 and 12. This sample was again stratified by age, race/ethnicity, gender, parent education, and geographic region. Oversampling was included for minority groups. Validation studies were carried out with clinical populations. Following the standardization and validation of data, the final selection of the subtests for each of the five functional domains was made.<sup>203</sup>

The NEPSY provides standard scores for the five domains noted previously. These are composite scores derived from specified subtests in each of the domains. The mean of the core domain scores is 100 with a standard deviation of 15. The subtest scaled scores within each core domain score have a mean of 10 and an SD of 3. The standard scores allow the NEPSY core domain scores and subtest scaled scores to be compared with other types of normalized scores (e.g., WISC-III or CAS scores). Supplemental scores are also available that enable the examiner to evaluate a child's performance in more detail and to identify factors that could account for or contribute to the child's poor performance. Qualitative observations are also recorded, much in the same manner that Luria emphasized during his career. The reader is referred to the NEPSY manual<sup>203</sup> for a more complete



understanding of the only multidomain neuropsychological test instrument developed to date for very young children.

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# 7 Behavioral Assessment Following Traumatic Brain Injury

## INTRODUCTION

It is ironic that improved medical management of the acute aspects of traumatic brain injury (TBI) has increased the number of survivors and, as a result, the number of severely handicapped individuals, many of whom will become burdens to their families, rehabilitation facilities, or social services.<sup>1</sup> The social outcome of such injuries is also very significant. More and more studies are suggesting that emotional, behavioral, or other psychosocial changes are more disturbing for relatives, and more difficult for the community to accept, than other forms of physical handicap such as cerebral palsy or quadriplegia.<sup>2</sup> The multiple characteristics of behavioral and psychosocial changes following brain trauma include reduced tolerance to stress, increased emotional lability, verbal or physical threatening or aggressive behaviors, a dysfunction of previous social skills, inappropriate behavior, and lack of concern or denial of the feelings of others. Behavioral disorders are far more likely to interfere with integration of the patient into the family and society than are adverse cognitive outcomes following TBI. Abnormal behaviors often lead to interference with rehabilitation attempts.

Earlier studies have noted that brain injury patients and controls did not differ in regard to preinjury psychopathology or social dysfunction as measured by standard instruments.<sup>3</sup> When brain-injured patients were examined for psychopathology 6 weeks after their injury, 39% of head injury patients were identified with psychiatric disorders, compared with only 4% of control patients. Those patients who developed depression or anxiety were, on average, 10 years older and were more likely to be women than were the control patients. In fact, Robinson and Jorge have recently argued the importance for all clinicians to understand that structural brain lesions, particularly from traumatic brain injury, are associated with lifelong depressive disorders and other behavioral disturbances.<sup>4</sup> Thus, this chapter will focus upon the adverse behaviors following traumatic brain injury that are most likely to interfere with life function in patients and also the substantial impact upon family and caregivers that arises from the effects of traumatic brain injury.

## THE ADULT

### EFFECTS UPON AFFECT AND MOOD

Holsinger et al. noted that the risk of depression remains elevated for decades following head injury and seems to be the highest in those who have had a severe head injury. They evaluated the lifetime rates of depressive illness 50 years after closed-head injury in male World War II veterans who served during 1944–1945 and were hospitalized at that time for a head injury, pneumonia, laceration, puncture, or incision wounds.<sup>5</sup> They found an odds ratio of 1.63 for the appearance of major

depression in head-injured veterans vs. controls. They noted in their studies that the increase in depression could not be explained over the lifetime of the veteran by history of myocardial infarction, history of cerebrovascular accident, or history of alcohol abuse. The lifetime risk of depression increased with severity of the head injury. In reviewing mood disorders following traumatic brain injury over the short term, it appears that these disorders are quite persistent and that, over a 1-year period, little change may be seen in the level of severity of the depression.<sup>6</sup> Approaching the 1-year interval from a different angle, a depressive illness was present in 14% of traumatic brain injury patients compared with 2.1% of the general population. Thus, in comparison with the general population, a higher proportion of adult patients developed psychiatric illness, specifically depression, 1 year after traumatic brain injury.<sup>7</sup> Others have noted that following traumatic brain injury, the most frequent Axis I diagnoses were major depression and other select anxiety disorders such as posttraumatic stress disorder (PTSD) and obsessive-compulsive disorder (OCD). Psychiatric comorbidity is high in association with depression.<sup>8</sup> It is imperative that traumatic brain injury patients be assessed for depression. Depression can, and often does, impede the achievement of optimal functional outcome, whether in the acute or chronic stages of recovery.<sup>9</sup>

As stressed in [Chapters 3 and 4](#), assessment for suicide potential should be made of every brain-injured patient examined for neuropsychiatric purposes. Posttrauma suicide risk seems to be increased by the connection between psychosocial disabilities as an outcome of brain injury and mood disorders. In fact, psychosocial disabilities appear more strongly associated to mood disorders than they do to physical disabilities.<sup>10</sup> A recent report<sup>11</sup> studied a consecutive series of patients admitted with stroke, traumatic brain injury, myocardial infarction, or spinal cord injury. This study included almost 500 patients who were psychiatrically examined. Seven and three-tenths percent of patients with acute medical illness had clinically significant suicidal ideation. Twenty-five percent of patients with major depression and concurrent physical illness developed suicidal ideation. The prognosis was good for those patients who were detected and treated, and the most important factor in preventing suicide among this population appeared to be the early treatment of depressive disorders.<sup>11</sup> Leon-Carrion et al. have noted that during the recovery period following traumatic brain injury, the risk of suicide is high. The profile of these patients reveals an emotional person with cognitive difficulties demonstrating problems with reality interpretation. The patients try to understand what is happening around them, but are unable to cope. These patients often demonstrate concrete thinking, although they have difficulty solving problems, and they have few intellectual resources to cope with their surroundings. They are particularly unable to distance themselves from the emotional aspects of situations.<sup>12</sup> The reader may wish to review a suicide prevention strategy recently developed specifically for families and patients following traumatic brain injury.<sup>13</sup>

## MEASURING MOOD CHANGES

### Beck Anxiety Inventory

The *Beck Anxiety Inventory* (BAI) is designed to measure subjective symptoms of anxiety in adolescents and adults. It is a self-administered inventory and contains 21 descriptive symptoms of anxiety that the patient rates on a 4-point scale: 0 — not at all; 1 — mildly, it did not bother me much; 2 — moderately, it was very unpleasant, but I could stand it; and 3 — severely, I could barely stand it. Scoring is performed by adding the raw scores for each of the 21 symptoms; the maximum score the patient can achieve on this test is 63 points. Minimal anxiety ranges from scores of 0 to 7 points, mild anxiety ranges from scores of 8 to 15 points, moderate anxiety ranges from scores of 16 to 25 points, and greater than 26 points is consistent with severe anxiety.<sup>14</sup>

This inventory provides only an estimate of overall severity of anxiety. Since the test contains only 21 items, its discriminating power is thus weak as far as psychological tests go. Therefore, it is recommended that this test instrument be administered in association with the *Beck Depression Inventory-II* (BDI-II) or the *Beck Hopelessness Scale*, as this will provide a more comprehensive

assessment of the patient's subjective emotional difficulties. The examiner is warned that there are no internal validity controls on this test instrument. Therefore, the individual's score on the Beck Anxiety Inventory must be consistent with other personality tests noted below that contain internal validity controls, such as the *Minnesota Multiphasic Personality Inventory-2* (MMPI-2), *Millon Clinical Multiaxial Inventory* (MCMI-III), or *Personality Assessment Inventory* (PAI). Moreover, this test may not be appropriate for patients who have sustained severe traumatic brain injuries, as their organic denial may interfere with awareness of their emotional problems.<sup>15</sup>

### **Beck Depression Inventory-II**

Like the BAI, this test instrument is based upon the original work of Aaron Beck, M.D.<sup>16</sup> The Beck Depression Inventory (BDI) contains 21 forced-choice statements regarding depressive symptoms. It is useful for measuring the severity of depression in adults and adolescents age 13 years and older. The BDI-II was developed to correspond with diagnostic criteria in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV).<sup>17</sup> The BDI-II is an outgrowth of the original BDI, which became the BDI-1A. For the new, revised version of the BDI-II, four of the original items (weight loss, body image change, somatic preoccupation, and work difficulty) were dropped and have been replaced by four new items (agitation, worthlessness, concentration difficulty, and loss of energy) in order to index symptoms more typical of severe depression or depression warranting hospitalization. Two other items were changed to allow for increases as well as decreases in appetite and sleep. Many of the statements and their alternatives were reworded. Unlike the BDI-1A, the BDI-II constitutes a substantial revision of the original BDI.<sup>18</sup>

This test is easy to administer and requires about 5 to 10 min. It is also easily scored, but it should be used only by professionals who are well schooled in the assessment of depressed persons. Patients with severe closed-head injuries may not test as depressed because they may be unaware of their cognitive deficits in a fashion similar to that noted previously for the BDI. For those performing forensic examinations, one of the major limitations of this test is that individuals involved in litigation who are being evaluated by the courts may purposely test as severely, if not profoundly, depressed, because of the test's obvious face validity for depression.<sup>15</sup> The statistical bases for this test instrument are much stronger than for the two previous versions of the BDI.

### **Millon Clinical Multiaxial Inventory-III**

The MCMI-III is a personality inventory containing 175 questions. Unlike during the administration of the MMPI-2, the questions and the patient's responses are contained within the same booklet. This test is designed to be used with patients who are ages 17 or older. Unlike the MMPI and its versions, it attempts to directly assess preexisting personality traits or disorders, and as a consequence, it may be valuable in forensic assessment of brain injury cases where prior personality function may be an issue.<sup>15</sup>

The 175 total test items of the MCMI-II is far less than the 567 items of the MMPI-II. It has been produced to reduce objectionable statements. The reading and vocabulary skill levels are approximately eighth grade. The test is constructed as an operational measure of personality syndromes derived from the theory of personality and psychopathology developed by Theodore Millon.<sup>19</sup> The MCMI-III includes changes to comport more closely to the diagnostic criteria contained in the DSM-IV.<sup>17</sup> Software is available from the manufacturer to allow a computer-generated interpretive narrative report, or the test may be mailed to the manufacturer for grading. However, as discussed in the forensic section of this book, that may not be advisable in forensic assessments.

The MCMI-III has been shown to be a valid test. However, the cross-validation sample techniques were developed by its authors. It has a limited database relative to the extraordinarily long and thorough analysis of the MMPI and subsequent revisions. Like the MMPI test instruments, the MCMI-III was not designed to identify or diagnose brain injury. Its primary value in the assessment

of traumatic brain injury lies in its ability to describe the various emotional and adjustment problems seen in patients following brain trauma. Moreover, as noted in [Chapters 2 and 3](#), it may be useful in determining if premorbid personality dysfunction has been exacerbated by the effects of traumatic brain injury. However, the examiner is cautioned to not assume that the Axis II profile produced by this test, which purports to describe premorbid or long-standing personality traits, accurately reflects those traits after a significant duration of time has elapsed between the time of the injury or the accident and when the patient is actually tested. For example, Spordone and Saul<sup>15</sup> point out that item 43, “My own bad temper has been a big cause of my troubles,” was designed to identify long-standing antisocial or borderline personality traits. If after sustaining a traumatic brain injury that results in poor frustration tolerance, irritability, and aggressive outbursts toward others, the patient recognizes this problem and responds “yes,” the patient is likely to be diagnosed as having long-standing antisocial or borderline personality traits. As we saw in [Chapter 2](#), this very well could be “acquired sociopathy,” often seen following orbitofrontal brain trauma. Thus, great skill is required in drawing conclusions of Axis II profiles on the MCMI-III and relating those to the presence of premorbid personality dysfunction. Such a determination should not be based solely on the results of the MCMI-III, and this personality delineation will require a thorough investigation of prior academic, legal, medical, military, and occupational records as well as a face-to-face examination before an assessment of this nature is complete.

### **Minnesota Multiphasic Personality Inventory-2**

The original Minnesota Multiphasic Personality Inventory (MMPI) was published in 1943 after extensive research studies at the University of Minnesota. It was developed by psychologist and psychiatrist Hathaway and McKinley, respectively.<sup>20</sup> All versions of the MMPI contain three validity scales, the L (lie), the F (frequency), and the K (defensiveness). The MMPI-2 contains the more recently added scales VRIN (inconsistency), TRIN (response bias), and Fp (psychopathology). A patient’s profile on these scales can provide valuable insights as to whether the patient is exaggerating, denying psychological problems, defensive, seeking out help for emotional problems, or faking a mental disorder. The use of these validity scales generally requires consultation with a psychologist who is expert and trained in the MMPI instruments. A more extensive review of specific applications of these scales to faking and symptom magnification is provided in the forensic portions of this text.

The MMPI-2 contains 10 clinical scales:

- 1 — Hs: hypochondriasis
- 2 — D: depression
- 3 — Hy: hysteria
- 4 — Pd: psychopathic deviate
- 5 — Mf: masculinity/femininity
- 6 — Pa: paranoia
- 7 — Pt: psychasthenia
- 8 — Sc: schizophrenia
- 9 — Ma: mania
- 0 — Si: social introversion

This test may be scored by using special templates over the patient’s answer sheet or by entering the patient’s raw scores into computer software produced by the University of Minnesota Corporation. The psychologist can examine the relative elevations of each of these scales in relationship to the others and determine the clinical significance of the patient’s profile, as well as judge the overall test responses for validity. The content scales can provide an adjunct to the traditional empirically derived clinical scales. The reader who wants a more thorough understanding of

MMPI-2 scoring and analysis should consult with some of the standard texts on the matter, such as Graham.<sup>21</sup>

The MMPI-2 has been administered to individuals with moderate to severe traumatic brain injury.<sup>22</sup> Individuals following brain trauma tend to show elevated scores on the schizophrenia (8) and mania (9) scales. However, patients who have sustained mild traumatic brain injury tend to show elevations on scales 1, 2, and 3 (hypochondriasis, depression, and hysteria). Elevation on scale 1 and a low score on scale 5 tend to predict low likelihood for resumption of employment following a traumatic brain injury. The MMPI-2 was not designed specifically to diagnose brain damage. Unfortunately, some psychiatrists and psychologists rely on the patient's MMPI-2 profile to diagnose brain damage or organicity.<sup>15</sup> This should not be done. However, a patient's profile on the MMPI-2 may be used to determine the presence of significant emotional problems that may account, at least in part, for relatively poor performance on neuropsychological testing or be an outcome of traumatic brain injury itself. Thus, the MMPI-2 appears to have usefulness for measures of outcome following traumatic brain injury, but it lacks specificity for the diagnosis of traumatic brain injury.

When administering the MMPI-2, it should be remembered that another person is not to be interposed between the test questions and the patient. In other words, the examiner, or a surrogate, should not read the test items to the patient. If the patient's reading ability (a sixth-grade reading level is required to understand MMPI-2 items) is insufficient to take the test unaided, special auditory tapes containing an oral repetition of the test items can be obtained from the test manufacturer. This is a perfectly valid way to administer the test to those with poor reading skills. Moreover, there are available Spanish language and French language editions if required. It is probably wise in clinical situations to measure reading recognition with the *Wide Range Achievement Test-III* or other similar test instrument before administering the MMPI to ensure minimal reading proficiency. For forensic assessment, as discussed more fully later in the text, it may be necessary to further measure reading comprehension as well as recognition of language.

If using a language version other than English, the norms may not be appropriate for the patient and psychological consultation may be required to determine if appropriate norms are being used. The patient's responses to languages other than English may reflect cultural factors that were not part of the original database for the MMPI-2, even though it is demographically correct and corresponds to the average demographics of the U.S. in 1989.<sup>23</sup> Many psychologists numerically score this test on a computer, which actually is probably more reliable than hand scoring using the templates. However, care must be exercised when using the narrative descriptive scoring procedures in addition to numerical scoring, and it is recommended that the narrative descriptors never be used alone without extensive face-to-face evaluation of the patient. Moreover, the examiner should not rely solely on the MMPI-2 to determine whether an individual has psychological or psychiatric impairment. The neuropsychiatric examination of traumatic brain injury should be based on a detailed clinical and background history, behavioral observations, interviews with collateral sources if needed, brain imaging and neurological examination, and a thorough review of medical and psychiatric records.

### **Personality Assessment Inventory**

The *Personality Assessment Inventory* (PAI) was developed by Morey,<sup>24</sup> and it is a self-administered objective test of personality and psychopathology. Unlike the MMPI, this test is based upon clinical syndromes and is more consistent with contemporary diagnostic practices.<sup>25</sup> The PAI is useful for patients from ages 18 through adulthood. There is no data to support the interpretation of the test scores for adolescents, unlike the Minnesota Multiphasic Personality Inventory-A (MMPI-A). This test has a wider range of utility at the lower end of the intellectual and educational scales, as the reading level necessary to take the PAI is at the fourth grade. The test usually can be administered in 45 to 60 min, unlike the 1½ h or more generally required for the MMPI-2. That is because this test contains 344 test items, compared with the 567 test items for the MMPI-2. There are 4 validity

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**TABLE 7.1**  
**Adult Behavioral Tests That Are Useful in**  
**Traumatic Brain Injury**

**Mood/Affect**

Beck Anxiety Inventory  
Beck Depression Inventory-II  
Millon Clinical Multiaxial Inventory-III  
Minnesota Multiphasic Personality Inventory-2  
Personality Assessment Inventory  
State-Trait Anxiety Inventory

**Aggression**

Aggression Questionnaire  
Buss-Durkee Hostility Inventory  
State-Trait Anger Expression Inventory

**Emotional Intelligence**

Behavioral Assessment of the Dysexecutive Syndrome  
Bar-On Emotional Quotient Inventory

**Neurobehavioral Function**

Neurobehavioral Functioning Inventory

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scales, 11 clinical scales, 5 treatment scales, and 2 interpersonal scales. The clinical scales contain a number of subscales. [Table 7.1](#) describes the validity and clinical scale components.<sup>15</sup>

It is being argued more and more in the psychological literature that the PAI is psychometrically superior to the MMPI-2 and more clinically relevant. The test questions are more straightforward than those on the MMPI-2. However, like the MMPI-2, this test instrument was not designed to establish the presence of brain damage, and it should not be used for this purpose. Patients with impaired cognitive abilities as a result of brain trauma should be tested with caution, and it may not be appropriate for patients who are confused or have significant psychomotor retardation.<sup>15</sup> As with the caveat noted above for the MMPI-2, determination of the psychological or psychiatric state should not solely rest on the use of this test instrument and a thorough examination should be made concurrently. A computerized interpretive profile and narrative report are commercially available from the test manufacturer. The test can be computer scored.

### **State-Trait Anxiety Inventory**

Construction of the *State-Trait Anxiety Inventory* (STAI) began in 1964 with the goal of developing a single set of items to provide objective measures of state and trait anxiety. The concepts of state and trait anxiety were first introduced by Cattell.<sup>26</sup> State anxiety and trait anxiety are analogous in certain respects to kinetic and potential energy. The anxiety state, like kinetic energy, refers to a palpable reaction or process taking place at a given time. On the other hand, anxiety traits, like potential energy, refer to individual differences in reactions.<sup>27</sup>

The STAI was designed to be self-administering. It contains 40 items, with 20 items on Form Y-1 and 20 items on Form Y-2. The patient circles one of four responses to each question based on the following categories: almost never, sometimes, often, and almost always. The test may be administered to adults ranging in age from 19 to 69 years. Normative data for Form Y are from working adults, college students, high school students, and military recruits. Form X norms are available within the Form Y manual for male neuropsychiatric patients, general medical and surgical patients, and young prisoners. However, these norms are not based on representative or stratified

samples. One useful function of the STAI is for following patients during treatment. Since it only takes 6 to 7 min to administer this test, it can be used serially to evaluate levels of anxiety throughout the rehabilitation and treatment process.

## AGGRESSION

The Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation performed a survey of its members to determine whether physiatrists formally measured agitation following brain injury. The majority of physiatrists surveyed did not formally identify or measure agitation in any scientific sense.<sup>28</sup> The neuropsychiatric examiner is not likely to deal with agitation chronically, as aggression is probably more frequently encountered than agitation. However, it is important to be aware that this does occur acutely following traumatic brain injury at a high rate and is seen both in the neurosurgical unit and the rehabilitation unit. A simple rating scale (*Agitated Behavior Scale*) is used to measure agitated behavior; it was developed at the Ohio State University Department of Physical Medicine and Rehabilitation.<sup>29</sup> The examiner should also review past medical records to determine the level of agitation following traumatic brain injury, as there is some relationship between severity of injury and severity of agitation. Irritability following brain injury has been studied, and acute-onset irritability is found at a higher frequency in patients who have left cortical lesions. On the other hand, delayed-onset irritability is seen in patients who have poor social functioning and a greater impairment in activities of daily living regardless of lesion location. These findings suggest that post-brain-injury irritability may have different causes and require different treatment planning than that found in the acute stage.<sup>30</sup>

Disinhibited aggressive behavior occurs following traumatic brain injury. The exact incidence is not well known (see [Chapter 2](#)). The disinhibited behavior is often called *impulsive aggression*. Where this has been studied, a higher incidence of premorbid aggressive behavior is noted, and the aggressive persons generally are younger. They also had more preinjury impulsive, irritable, and antisocial features than nonaggressive controls.<sup>31</sup> A review of data from the Viet Nam Head Injury Study revealed that patients with frontal ventral medial lesions consistently demonstrated more aggressive and violent tendencies than control patients or patients with lesions in other brain areas. The optimistic news from this study is that most of the aggression was by verbal confrontation rather than physical assault. However, this type of behavior did have a significant adverse impact and disruptive influence upon family activities.<sup>32</sup> When one looks at outcomes of traumatic brain injury regarding criminal activity, there is noted to be a direct relationship between the level of alcohol use and the level of criminal arrest rates.<sup>33</sup> In this particular study, a relatively high incidence of heavy drinking both before and after injury was found among patients with a history of criminal arrest. One additional finding was that those persons with relatively high levels of aggressive behaviors and arrests had a strong association with a greater likelihood of psychiatric treatment. Should it become necessary, a framework of evaluation is used to determine the relevance of the association of traumatic brain injury and the ultimate commission of a crime.<sup>34</sup> This will be discussed more fully in the forensic sections of this text. However, for clinical evaluations, neuropsychiatric assessment often requires a detailed determination of aggression risk before placing patients into the home or other care facilities.

## MEASURING AGGRESSION

### Aggression Questionnaire

The *Aggression Questionnaire* (AQ) is an updated version of the *Buss–Durkee Hostility Inventory*. Dr. Buss contributed to the development of the Aggression Questionnaire more than 40 years later.<sup>35</sup> It is a brief measure consisting of only 34 items scored on five scales: Physical Aggression (PHY), Verbal Aggression (VER), Anger (ANG), Hostility (HOS), and Indirect Aggression (IND). An AQ total score is also provided, along with an Inconsistent Responding (INC) Index score as a validity

indicator. The individual taking the test rates the item description on a scale from 1 (not at all like me) to 5 (completely like me). The items on this test instrument can be read and understood easily by any person with at least a third-grade reading ability. The norms are based on a standardization sample of 2138 persons ranging in age from 9 to 88 years. The Inconsistent Responding Index, although unlikely to uncover sophisticated fakers, may help to identify unusual levels of inconsistency in item responses that can result when a test taker attempts to “fool the test.” It is also useful to detect persons who are careless in completing the form or who lack consistent attention as a result of brain injury.

The AQ total score is based on the person’s responses to all 34 AQ items. It is a good summary measure of the general level of anger and aggression the individual has reported. Statistically, the AQ total score is most closely associated with the PHY and ANG subscale scores. When the AQ total score is high, it is important to examine the individual’s subscale scores and other information available to the examiner to understand what kind of experiences the individual has reported and to assess the level of risk for aggression. If the picture is dominated by high levels of anger and hostility, for example, but relatively low levels of physical or verbal aggression, the implications for follow-up assessment and intervention are likely to differ from what is called for when the picture is dominated by high levels of physical or verbal aggression.<sup>35</sup> As for the subscales, it should be noted that those who obtain high PHY scores tend to justify their aggressive acts in their own minds. They perceive themselves as being provoked by others, and they are more likely than others to respond aggressively when they feel ashamed or humiliated. Low PHY scores may indicate a relative absence of physically aggressive behavior and a relatively strong ability to control physically aggressive impulses.

Individuals with high scores on the VER scale are commonly aroused to anger by situations they perceive to be unfair. Persons with a preexisting antisocial personality will tend to obtain high scores on the VER scale. Low VER scores are obtained by individuals who do not perceive themselves as argumentative. The ANG subscale describes aspects of anger. Persons who score high on the ANG scale may benefit from relaxation training, as well as cognitive-behavioral and other arousal-reducing strategies or psychotherapy. Thus, this scale may be useful to predict those who might respond to treatment techniques aimed at reducing anger. The HOS subscale is most closely associated with pervasive social maladjustment, as well as severe psychopathology. It is probably wise to review this scale with elements on the MMPI-2. Predictors of violence from the MMPI-2 subtests are more fully explained in the forensic sections of this text. Persons with elevated HOS scores are more likely to demonstrate affective disturbance and social isolation. Extremely low HOS scores are consistent with individuals who feel comfortable in their current social surroundings. The IND scale measures the tendency to express anger and actions that avoid direct confrontation. Youngsters who score high on IND may be identified as oppositional or avoidant, and they often have disrupted peer relationships. Adults with antisocial personality characteristics tend to obtain high IND scores. People with low IND scores are likely to be willing to use direct confrontation to resolve conflicts in their lives.

With respect to psychiatric disturbances, individuals with anxiety disorders often obtain elevated VER and HOS scores in combination. Persons identified as antisocial will often have high VER, HOS, and IND scores relative to other AQ scores. Children who have ADHD may obtain high scores on both the PHY and HOS scales.<sup>35</sup>

### **Buss–Durkee Hostility Inventory**

The Buss–Durkee Hostility Inventory was originally published in 1957, and it still has some usefulness in the evaluation of hostile behaviors.<sup>36</sup> This inventory contains 75 items from an original inventory of 105 items. The 75 items were determined following measures of internal consistency that rejected 30 of the original items. The questions are answered in a true–false format. Factor analyses on college men and women revealed two factors: an attitudinal component



of hostility (resentment and suspicion) and a motor component (assault, indirect hostility, irritability, and verbal hostility). This inventory is still used today but only in limited forms, as the AQ is supplanting it.

### **State–Trait Anger Expression Inventory-2**

The original *State–Trait Anger Expression Inventory* (STAXI) was published in 1988.<sup>37</sup> The new version, the STAXI-2, provides concise measures of the experience, expression, and control of anger. The STAXI-2 was developed for two primary reasons: (1) to assess the components of anger for detailed evaluations of normal and abnormal personality, and (2) to provide a means of measuring the contributions of various components of anger to the development of medical conditions, particularly hypertension, coronary heart disease, and cancer.<sup>38</sup>

Anger expression and anger control within the STAXI-2 instrument are conceptualized as having four major components: (1) Anger Expression-Out involves the expression of anger toward other persons or objects in the environment; (2) Anger Expression-In is anger directed inward; (3) Anger Control-Out is based on the control of angry feelings by preventing the expression of anger toward other persons or objects in the environment; (4) Anger Control-In is related to the control of suppressed angry feelings by calming down or cooling off when angered. Thus, since anger following traumatic brain injury is so pervasively destructive to relationships and family dynamics, this instrument may prove useful for the assessment of traumatically brain-injured persons who are being considered for family or personal psychotherapy to reduce hostility.

Separate norms are provided for females and males in three age groups: 16 to 19 years, 20 to 29 years, and 30 years and older. Appendix A of the manual also provides percentiles based on scores of a psychiatric patient sample. T-scores are provided with a mean of 50 and a standard deviation of 10, similar to the T-scores used for the MMPI-2. Guidelines exist for interpreting high scores on the STAXI-2 scales and subscales. The STAXI-2 consists of six scales, five subscales, and an Anger Expression Index, which provides an overall measure of the expression and control of anger. Persons taking the test rate themselves on a 4-point scale that assesses either the intensity of their angry feelings at a particular time or how frequently anger is experienced, expressed, suppressed, or controlled. Completion of the STAXI-2 generally requires 12 to 15 min. If an examinee does not understand an item, it is acceptable for the psychologist to provide simple definitions of the words or issues of concern. If 10 or more of the 57 items are missing, the protocol should be considered invalid. The test instrument enables the examiner to determine state and trait anger vs. anger expression and the ability to control oneself when angry. [Table 7.1](#) lists adult tests that are useful for behavioral evaluation following brain injury.

### **EFFECTS OF BRAIN INJURY UPON SEXUALITY**

A review of the literature in this area will generally find that sexual concerns have been neglected in much of the posttraumatic head injury and rehabilitation literature. Authors do report that the sexual sequelae after head injury include impulsiveness, inappropriateness, change in sex drive, reduction in sexual frequency, global sexual difficulties, and specific sexual dysfunctions.<sup>39</sup> Over 50% of individuals who suffer traumatic brain injury are reported to demonstrate a decrease in sexual arousal postinjury. Crowe and Ponsford<sup>40</sup> studied this in males and determined that men following brain injury have difficulty developing sexual imagery. Their results indicate that sexual arousal disturbances may exist above and beyond the disturbances of affect that have been associated with frontal injury from trauma. Interestingly, other researchers<sup>41</sup> have found that patients with frontal lobe lesions following brain injury reported an overall higher level of sexual satisfaction and functioning than those individuals with other than frontal lobe lesions.

Efforts have been made to predict sexual adjustment following traumatic brain injury. This has proved most difficult. For instance, when professionals are queried regarding sexual dysfunction

in their patients, physical changes are not identified as the primary obstacle preventing persons from achieving sexual satisfaction following traumatic brain injury. Rather, the cognitive and emotional sequelae of brain injury seem more important from the professional's perspective.<sup>42</sup> A Swedish study<sup>43</sup> noted that a high degree of physical independence and maintained sexual ability were the most important predictors for sexual adjustment following brain injury. Preinjury factors predicting successful sexual functioning following traumatic brain injury were not identified. Unfortunately, at this time in the treatment and rehabilitation of brain-injured patients, the causes and effects of sexual functioning after brain injury are very confusing. The medical literature does not clarify this confusion, and one cannot accurately differentiate between primary and secondary sexual problems following traumatic brain injury.<sup>44</sup>

One of the more complicated issues facing the neuropsychiatric examiner is that of sexually aberrant behavior following traumatic brain injury. Studies are scant on this matter as well and offer multiple theories and treatments.<sup>45-48</sup> Sex offending is a significant clinical problem among a small minority of men following traumatic brain injury. These men often have an absence of alcohol or preinjury histories of sexual offending, which suggests that the brain injury itself is a significant etiological factor underlying the offense. Simpson's Australian group<sup>49</sup> has studied this issue even further and noted that the sexually aberrant behavior in brain-injured persons correlates with a higher incidence of postinjury psychosocial disturbance in areas of nonsexual crime and failure to return to work. These rates were much higher than in a matched control group of other brain-injured persons who were not sexually aberrant. There were no significant differences between the two groups in the incidence of premorbid psychosocial disturbance or postinjury brain imaging findings or neuropsychological findings. Thus, they caution against simplistic explanations of sexually aberrant behavior as being the product of damage to frontal lobe systems or the result of a premorbid psychosocial disturbance. They further caution that results of neuropsychological examination alone cannot be considered conclusive when examining a brain-injured person who then develops sexually aberrant behavior.<sup>50</sup>

## **PSYCHOSOCIAL FUNCTIONING**

The psychosocial problems of decreased social contact, depression, and loneliness that occur for many persons suffering from traumatic brain injury create a major challenge for enhancing efforts at community reentry. These psychosocial problems remain a persistent long-term problem for the majority of individuals with severe traumatic brain injury. The problems of social isolation and decreased leisure activities create a renewed dependence of the survivor upon the family to meet these needs. This is particularly true since individuals who experience severe traumatic brain injury are at high risk for a significant decrease in their friendships and social support.<sup>51</sup> The goal of human rehabilitation is independent living. The National Council on the Handicapped<sup>52</sup> defines this as managing one's affairs, participating in day-to-day community of life in the manner of one's own choosing, fulfilling a range of social roles, including productive work, and making decisions that lead to self-determination.

One of the major factors that interfere with psychosocial functioning following traumatic brain injury is social competence. This is a range of behaviors that underlie communication between persons.<sup>53</sup> Problems with emotional control interfere with social competence. Kersel and others followed severe traumatic brain injury victims for 1 year postinjury. Problems with emotional control were found to be most distressing for patients. When these individuals were compared with their preinjury social functioning, they revealed a loss of employment at a 70% rate. Thirty percent of individuals had returned to live with their parents, and breakdown of relationships occurred for almost 40%.<sup>54</sup> Remarkably, when the study period is increased to 10 to 20 years, persons with traumatic brain injury in their families may need professional assistance to maintain a reasonable psychosocial quality of life. Severe traumatic brain injury seriously affects psychiatric symptomatology, which directly impacts the family and social domains. High rates of depression, psycho-

motor slowness, loneliness, and family member sense of burden are found at 10 years and beyond in many patients who have sustained traumatic brain injury.<sup>55</sup>

Measuring psychosocial recovery after traumatic brain injury has proved complex. Grant and Alves looked at this problem more than 15 years ago and found multiple confusing approaches in the medical literature.<sup>56</sup> The Department of Medicine at the University of Sydney has looked at the difficulties with psychosocial measurement for a number of years.<sup>57</sup> They have recently promoted the *Sidney Psychosocial Reintegration Scale* (SPRS), an instrument developed to quantify disability and handicap in persons with traumatic brain injury. The SPRS is a 12-item questionnaire measuring three domains of everyday living commonly disrupted after severe TBI. These include occupational activities, interpersonal relationships, and independent living skills. By statistical analysis, they demonstrated that the SPRS was sensitive to group differences on the *Glasgow Outcome Scale* (see [Chapter 1](#)) and to changes occurring during the period of active recovery. They found the SPRS to have sound psychometric properties, being a reliable, stable, sensitive, and valid instrument useful for both clinical and research settings. On the other hand, often the neuropsychiatric examiner is asked to predict psychosocial adjustment after TBI. That presents a more complex challenge. This has also been evaluated at the University of Sydney,<sup>58</sup> and their studies revealed that within the neuropsychological domain, the variable measuring behavioral regulation of abilities was the most significant (see the section on “Measuring Aspects of Emotional Intelligence Following Brain Injury”). Neurophysical impairments in memory functioning predicted successful occupational activities. Chronicity, cognitive speed, and behavioral regulation predicted success in interpersonal relationships. Neurophysical impairments, behavioral regulation, and memory functioning predicted independent living skills. When the Glasgow Outcome Scale is used for prediction of functioning, it also demonstrates predictive and concurrent validity of neuropsychological, psychosocial, and vocational functioning 6 months after injury.<sup>59</sup> The UCLA Brain Injury Research Center demonstrated a systematic decrease in mean neuropsychological test performance as a function of increasing Glasgow Outcome Scale severity, as well as an increased prevalence of symptoms of depression and lower ratings on measures assessing employability and capacity for self-care. Their study indicated that Glasgow Outcome Scale Category 4 (moderate disability) lacked sufficient discriminability (see [Chapter 1](#) for the Glasgow Outcome Scale). However, even with attempting to measure psychosocial function and outcome and to assist victims of traumatic brain injury, current community supports are often inadequate to deal with the complex array of neurologic and psychiatric difficulties. McAllister has outlined some principles helpful in the evaluation of the behaviorally challenged brain-injured patient in the community.<sup>60</sup>

## **DRIVING BEHAVIORS FOLLOWING TRAUMATIC BRAIN INJURY**

Believe it or not, it is difficult to find evidence that there is a significant worsening of driving skill following traumatic brain injury in those persons who are still functional enough to drive. A study from Norway found a higher number of traffic accidents after brain injury, but the difference was not significant. Those persons who did have an increased rate were generally young males who had deficits in cognitive and executive functions.<sup>61</sup> The University of Washington Study looked at a large cohort of eligible drivers in the state of Washington from 1991 to 1993. The relative risks of any subsequent crash or receipt of a driving citation were no greater for those who sustained a stroke or traumatic brain injury than for nonhospitalized individuals, nor were the risks of experiencing two or more of these events in the 12 months after hospitalization significantly elevated. These results did not support the hypothesis that individuals who have sustained a brain injury are at increased risk of motor vehicle crashes.<sup>62</sup> It may be that the reason for this somewhat surprising finding is that procedural memory is affected so little following traumatic brain injury (see [Chapters 2, 4, and 6](#) regarding memory dysfunction following traumatic brain injury). However, the evaluation of driving skill following brain injury must be obtained on an individualized basis, as some individuals are quite dysfunctional in driving behavior following a traumatic brain injury.

There is evidence that, in some individuals, attention can be substantially impaired following traumatic brain injury, and the attentional dysfunction may affect driving skill. Recent research suggests that the attentional deficit causes impairment in the driver's ability to cope with time pressure.<sup>63</sup> In terms of assessment, some evidence suggests that the *Useful Field of View* (UFOV), a measure of visual information processing, is a good predictor of vehicle crash risk in older adults. Recent research suggests that traumatic brain injury survivors have higher UFOV scores than young adults, which indicates a greater functional loss of peripheral vision in these individuals. Previous studies in older adults have shown that people with UFOV deficits are more likely to experience vehicle crashes.<sup>64,65</sup> Further recent research suggests that virtual reality testing may provide an innovative medium for direct evaluation of basic cognitive function such as divided attention and its impact on driving. These have previously not been available through traditional neuropsychological measures, which may not have ecological validity relative to driving.<sup>66</sup> If neuropsychological assessment of certain targeted functions that may be important for driving such as attention and vigilance are used, it is recommended that on-road evaluation also be provided as a supplement in cases with ambiguous test findings.<sup>67</sup>

## TRAUMATIC BRAIN INJURY AND IMPACT UPON EMOTIONAL INTELLIGENCE

Emotional intelligence is a term of art rather than a cognitive or behavioral domain. Sternberg<sup>68</sup> attempted to identify the operations used in solving standard intelligence tests in hopes that this would describe the intelligence of daily living. Howard Gardner made further attempts at this discovery with his theory of multiple intelligences.<sup>69</sup> He noted that damage to the frontal lobes of an adult exerts only relatively minor effects on the individual's ability to solve problems such as those found on a standard intelligence test, but it may wreak severe damage on the person's personality. The individual may no longer be recognizable as the same person known by others before the injury. In fact, Gardner believes that this kind of injury can cause a pathology of personhood.

Daniel Goleman<sup>70</sup> brought to public awareness the concept of emotional intelligence. He describes emotional intelligence as abilities representing five main domains:

1. *Knowing one's emotions.* This includes self-awareness and recognizing a feeling as it happens.
2. *Managing emotions.* This is the ability to handle feelings so they are appropriate and build upon self-awareness. In particular, this describes the capacity to soothe oneself and to shake off rampant anxiety, gloom, or irritability. People who are poor in this ability are constantly battling feelings of distress.
3. *Motivating oneself.* This has been described in [Chapters 2](#) and [4](#) in association with executive function. Self-motivation is part of self-mastery and creativity. The ability to exercise self-control and delay gratification and stifle impulsiveness underlies accomplishment of every sort.
4. *Recognizing emotions in others.* Empathy builds on emotional self-awareness and is the fundamental "people skill." This may be a feature of right brain nonverbal function discussed in [Chapter 4](#), but people who are empathic are more attuned to the subtle social signals that indicate what others need or want.
5. *Handling relationships.* The art of relationships is, in large part, a skill at managing emotions in others. This is the ability that underlies popularity, leadership, and interpersonal effectiveness.

Antonio Damasio has reviewed the famous story of Phineas Gage.<sup>71</sup> A short review of the alteration of Gage's emotional intelligence after the tamping rod was blown through his brain offers a fascinating 150-year-old review of traumatic brain injury and the impact it has upon emotional intelligence. Dr. Harlow spent much of his life exploring the behavior of Phineas Gage after his

injury.<sup>72</sup> Following injury, Gage could touch, hear, and see and was not paralyzed. He did lose vision in the left eye as the tamping rod passed under zygomatic bone, severing the optic nerve, and exited the posterior frontal skull. He is described as walking firmly and using his hands with dexterity, and he had no noticeable difficulty with speech or language. Prior to his injury, he was described as having “temperate habits” and “considerable energy of character.” Following the accident, he was described as “fitful, irreverent, indulging at times in the grossest profanity which was not previously his custom, manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no longer arranged than they are abandoned.” He was so different in his personality following his injury that his railroad employers had to terminate his employment shortly after he returned from sick leave, for they “considered the change in his mind so marked that they could not give him his place again.” The change in his employment status was not due to lack of physical ability or skill; it was due to a change in behavior and emotional intelligence.

Emotional intelligence has been studied scientifically to see its relationship to everyday life. There is a large scientific basis that affirms the ecological validity of emotional intelligence.<sup>73</sup> Research with brain-damaged patients shows that people who cannot experience affective reactions because of isolated frontal lobe damage also tend to make disastrous social decisions, and their social relationships suffer accordingly, even though intellectual abilities remain unimpaired. Adolphs and Damasio<sup>74</sup> have posited that affective processing is an evolutionary antecedent to more complex forms of information processing. They believe that higher cognition requires the guidance provided by affective processing. Traumatic brain injury often injures affective processing, as we have seen previously in this text.

Bar-On has argued that emotional intelligence is critical to human self-actualization.<sup>75</sup> He has conducted extensive research on emotional intelligence, and his cross-cultural findings strongly suggest that the best predictors of self-actualization are the following factors and facilitators, which he lists in their order of importance: happiness, optimism, self-regard, independence, problem solving, social responsibility, assertiveness, and emotional self-awareness. Many of these behavioral descriptors and facilitators are altered following traumatic brain injury.

## MEASURING ASPECTS OF EMOTIONAL INTELLIGENCE FOLLOWING BRAIN INJURY

### Behavioral Assessment of the Dysexecutive Syndrome

This test was developed to predict problems in everyday functioning arising from impaired executive function. The test battery consists of a collection of six novel tests and a questionnaire. These are similar to real-life activities likely to be problematic for persons who have impaired executive ability. The entire test can be administered in approximately 30 min, so it requires little time for completion. Some of the test items have timed components:

1. *Rule Shift Cards Test*: This examines the patient’s ability to respond correctly to a rule and shift from one rule to another.
2. *Action Program Test*: This requires the patient to obtain a cork from within a tube without using any of the objects in front of the patient. The patient is not allowed to lift the stand, the tube, or the glass beaker containing water and must perform the activity without touching the lid with his fingers.
3. *Key Search Test*: This requires the patient to develop a strategy to locate lost keys in an imaginary large, square field.
4. *Temporal Judgment Test*: This section contains four open-ended questions.
5. *Zoo Map Test*: On this test, the patient is shown how to visit a series of designated locations on a map at a zoo and must follow certain rules.

6. *Modified Six Elements Test*: In this section, the patient must perform three tasks, each of which is divided into two parts. The patient must attempt some portion within each subtest within a 10-min period without violating any rules.
7. *Dysexecutive Questionnaire*: This 20-item questionnaire samples the patient's emotional and personality changes, motivational changes, behavioral changes, and cognitive changes.

Sporidone and Saul<sup>15</sup> believe that this test is a useful tool for the evaluation of impaired executive functions in traumatically brain-injured patients. It is particularly useful in those persons who appear to be cognitively well preserved and function well in highly structured settings. In fact, research has shown that this test is a better predictor of a patient's executive function in real-world situations than is the *Wisconsin Card Sorting Test*.<sup>76</sup> The test apparently is able to differentiate patients with neurological disorders as a result of closed-head injuries from normal, healthy control patients. The test also seems to correlate well with behavioral ratings of executive functions made by the patient's family or significant others. However, in terms of statistical analysis, the test-retest reliability is low. This is because in general, there is a tendency for traumatically brain-injured patients to improve on follow-up testing. This may not be a sensitive test for patients who have sustained only a mild traumatic brain injury or patients who are depressed, have significant hearing or visual impairments, or are significantly anxious.<sup>15</sup>

### **Bar-On Emotional Quotient Inventory (EQ-i)**

The evolution of the *Bar-On Emotional Quotient Inventory* (EQ-i) began in 1980 with the independent development of a theoretically eclectic and multifactorial approach to operationally defining and quantitatively describing emotional intelligence.<sup>77</sup> The EQ-i has been used to evaluate the emotional intelligence of people suffering from severe medical problems such as heart disease, cancer, and AIDS. However, since there is no significant database on the EQ-i in traumatic brain injury at this time, its primary usefulness is to determine the effect of traumatic brain injury upon emotional intelligence, particularly in the regulation of emotions, and then apply this information to psychotherapy directed at individuals coping with the outcomes of brain injury. This is primarily true in an effort to apply emotional intelligence to the improvement of health and mental function.<sup>78</sup> It is recommended that the EQ-i be used as part of a larger evaluation process as delineated within this text.

Within the EQ-i, there are 15 conceptual components of emotional intelligence that are measured by the subscales. These include:

1. *Emotional self-awareness*: This is the ability to recognize one's feelings. It also is used to differentiate between feelings, to know what one is feeling and why, and to know what caused the feelings. This lack of ability is termed *alexithymia* (inability to express feelings verbally).<sup>79</sup>
2. *Assertiveness*: This subscale measures the ability to express feelings, beliefs, and thoughts and defend one's rights in a nondestructive manner. This ability is very difficult for traumatically brain-injured persons to manage due to the poor modulation of affect following some traumatic brain injuries.
3. *Self-regard*: This measures the ability to respect and accept oneself as basically good. Following traumatic brain injury, self-esteem is often impaired, particularly due to problems of interpersonal relatedness.
4. *Self-actualization*: This pertains to the ability to realize one's potential capacities. As the person rehabilitates, this subscale may be useful in monitoring general improvement during rehabilitation or psychotherapy.
5. *Independence*: The ability to be self-directed and self-controlled in one's thinking and actions and to be free of emotional dependency is an important aspect of this subscale. Traumatic brain injury often robs people of their independence, and this subscale is a

useful factor in measuring improved independence through cognitive rehabilitation and psychotherapy.

6. *Empathy*: To be aware of, to understand, and to appreciate the feelings of others is the core of empathy. Being empathetic means being able to “emotionally read” other people. Since traumatic brain injury often interferes with right cerebral processing and the nonverbal aspects of interpreting other people, this is an important subscale for determining the impact of behavioral disturbance related to right cerebral hemisphere injury.
7. *Interpersonal relationships*: This involves the ability to establish and maintain mutually satisfying relationships that are characterized by intimacy and by giving and receiving affection. Since relationships are often traumatically influenced following brain injury, this subscale may tap into the variables involved in problematic relationships following traumatic brain injury.
8. *Social responsibility*: The ability to demonstrate oneself as a cooperative, contributing, and constructive member of one’s social group is manifested as social responsibility. This component of the EQ-i relates to the ability to do things for and with others, accepting others, acting in accordance with one’s conscience, and upholding social rules. As discussed earlier in this text, “acquired sociopathy” may occur due to infraorbital brain injury, and this subscale may assist in the measurement of poor social outcomes following infraorbital injury.
9. *Problem solving*: Problem solving is multiphasic in nature and is the ability to identify and define problems as well as to generate and implement potentially effective solutions. Due to the significant aspects of frontal injury in traumatic brain injury, this aptitude often is impaired.
10. *Reality testing*: Following brain injury, many persons appear paranoid due to difficulty with reality testing. This subscale measures the ability to assess the correspondence between what is experienced and what objectively exists. It involves “tuning in” to the immediate situation, attempting to keep things in the correct perspective, and experiencing things as they really are, without excessive fantasizing or daydreaming about them. These abilities often are seriously impaired following traumatic brain injury, and this subscale assists in the assessment of those functions.
11. *Flexibility*: The ability to adjust one’s emotions, thoughts, and behavior to changing situations and conditions is consistent with flexibility. As noted in [Chapter 6](#), cognitive flexibility is often impaired following traumatic brain injury to frontal brain systems. This subscale may assist in the delineation of behaviors affected by lack of flexibility.
12. *Stress tolerance*: Many persons following traumatic brain injury will tell their treaters and therapists that they cannot deal with stressful situations. This subscale measures the ability to withstand adverse events and stressful situations without “falling apart” by actively and positively coping with stress. It may assist therapists and rehabilitation counselors in assessing a brain-injured patient’s ability to tolerate stressful situations.
13. *Impulse control*: This is the ability to resist or delay an impulse, drive, or temptation to act. [Chapter 2](#) explained the difficulties of persons with orbitofrontal brain trauma. This subscale may help delineate behaviors associated with inferior frontal brain injury.
14. *Happiness*: Many brain-injured patients tell their therapists, counselors, and physicians how unhappy they are following brain injury. This unhappiness spills over into family relationships. This subscale in the EQ-i measures the ability to feel satisfied with one’s life, to enjoy oneself and others, and to have fun.
15. *Optimism*: Optimism is the opposite of pessimism, which is a common symptom of depression and a common feature of suicidal people. It is the ability to look at the brighter side of life and to maintain a positive attitude, even in the face of adversity. This subscale may be useful to assist in the screening of persons who are having substantial behavioral difficulty with affect regulation.

The EQ-i takes about 30 to 40 min for most people to complete, as it is short and contains only 133 items. A significantly brain-injured person may require a longer time. There are no imposed time limits for completing the EQ-I, but patients should complete the inventory in one sitting. Professionals using the EQ-i can obtain software support from the test manufacturer to assist in scoring and display of the results.

There are validity controls within the EQ-i. The omission rate is tabulated and should be near 0%. If more than 6% of answers are omitted, the results are considered invalid. An Inconsistency Index will identify those persons who cannot maintain their concentration or comprehension well enough to complete the test. The Positive Impression and Negative Impression Scales will identify those persons who are excessively optimistic or attempting to make themselves appear worse than they are. If either the Positive or Negative Impression Scale score exceeds 2 standard deviations from the mean, the protocol is invalid. The age range for this test instrument is persons 16 years of age and older. The reading level required is approximately sixth or seventh grade (12 to 13 years of age). Even though the reading level is this low, the EQ-i should not be administered to youngsters under the age of 16. There is a child and adolescent version currently in development.

## THE CHILD

### EFFECTS UPON AFFECT AND MOOD

Research studies and reviews of the pediatric literature demonstrate high rates of new psychiatric disorders following pediatric traumatic brain injury.<sup>80</sup> ADHD and depressive disorders are the most common lifetime and new diagnoses in children following traumatic brain injury. When looking at depressive symptoms specifically, these seem mainly related to socioeconomic status. An inverse relationship exists between the frequency of depression in children and the level of socioeconomic prosperity.<sup>81</sup> However, when one reviews the large groups of studies of head-injured children, there is a significant paucity of data about mood and depression in these children. Most of the data are concerned with cognitive function rather than mood function.<sup>82-85</sup> Max and others<sup>86,87</sup> have documented the development of depression and other psychiatric disorders in children and adolescents following traumatic brain injury, and their findings are consistent with the findings of other researchers that have noted substantial behavioral disorders in brain-injured children.

### MEASURING MOOD CHANGES IN CHILDREN

#### Adolescent Psychopathology Scale

The *Adolescent Psychopathology Scale* (APS) was developed and standardized for use in the clinical assessment of adolescents ages 12 to 19 years. The APS consists of 346 items and requires approximately 45 to 60 min to complete. A significantly impaired adolescent may take somewhat longer for completion. The standardization sample of this test instrument does not include individuals under the age of 12 years or over the age of 19 years. Therefore, this test should not be used for children or young adults outside those age ranges.<sup>88</sup> Reading level requirements are at about the third-grade level. However, the test author advises that years of completed education is not a reliable indicator of reading ability, and it is recommended that the youngster be administered an appropriate reading test such as those described in [Chapter 6](#).

This is a self-report measure of psychopathology, and the test instrument has been devised to comport with the majority of DSM-IV Axis I clinical disorders and five of the DSM-IV Axis II personality disorders. The APS was designed specifically for adolescents, and it is not a downward extension of adult scales from other test instruments. It assesses four broad content domains: (1) clinical disorders, (2) personality disorders, (3) psychosocial problems, and (4) response style indicators. The APS further provides the perspective of internalizing and externalizing domains, which are based on a factor analysis of the scales. Specific analytical procedures for performing this



function are contained within the technical manual, and a well-trained psychologist experienced with this test instrument should have no difficulty with interpretation. The clinical disorder scales deal with 20 DSM-IV diagnoses: ADHD, conduct disorder, oppositional defiant disorder, adjustment disorder, substance abuse disorder, anorexia nervosa, bulimia nervosa, sleep disorders, somatization disorder, panic disorder, OCD, generalized anxiety disorder, social phobia, separation anxiety disorder, PTSD, major depression, dysthymic disorder, mania, depersonalization disorder, and schizophrenia. The personality disorder scales evaluate pervasive aspects of inner sense, feelings, affect, and thoughts, as well as behaviors that deviate significantly from normal characteristics of adolescence. The five personality disorder scales include avoidant personality disorder, obsessive-compulsive personality disorder, borderline personality disorder, schizotypal personality disorder, and paranoid personality disorder.

The psychosocial problem content scales function primarily as targets for intervention. These scales are categorized along the internalizing–externalizing dimension noted previously. The psychosocial problem content scales include self-concept, psychosocial substance use difficulties, introversion, alienation–boredom, anger, aggression, interpersonal problems, emotional lability, disorientation, suicide, and social adaptation. A number of these problems are important in the assessment of children following traumatic brain injury, and they would include the anger, aggression, emotional lability, and suicide scales.

The response style indicator scales are used for validity checks. They include four scales: (1) lie response, (2) consistency response, (3) infrequency response, and (4) critical item endorsement. The Lie Response Scale assesses the adolescent's openness and willingness to give honest answers. The Consistency Response Scale measures the youngster's understanding of item content and serves as a potential screener for random responding or inattention. Inattention could occur due to poor reading comprehension or serious brain injury, and that should be kept in mind. The Infrequency Response Scale contains items that generally are not endorsed by normal adolescents. They represent unusual and bizarre behaviors, affect, and cognition. The Critical Item Endorsement Scale consists of 63 of the 346 items on the APS. They are designated as critical items for their ability to differentiate clinical from nonclinical individuals.

### **Behavior Assessment System for Children**

The *Behavior Assessment System for Children* (BASC) is a multimethod, multidimensional approach to evaluating the behavior and self-perceptions of children ages 2½ to 18 years. The BASC has five components, which may be used individually or in any combination. These are (1) a self-report scale, in which the child can describe his or her emotions and self-perceptions; two rating scales, (2) one for teachers and (3) one for parents, which gather descriptions of the child's observable behavior; (4) a structured developmental history; and (5) a form for recording and classifying directly observed classroom behavior.<sup>89</sup>

The author has used this instrument in his practice for a number of years, as he has most of the other instruments discussed in this text. Through trial and error, it has been learned that the teacher section of the BASC correlates poorly with measurements made in the doctor's office. It seems that teachers are significantly concerned about identifying a child with special needs, as that child will then require government-mandated programs. Therefore, the author has discovered that unless the child is so observably brain injured that no one can miss it, teachers are loathe to describe the child's behavior as being significantly different following a brain injury. Moreover, they see potential risk in that they might be pulled into a legal situation. Therefore, it is not recommended that the teacher forms be used with this test instrument in the assessment of traumatically brain-injured children, as the results may be spurious.

Norms are representative of the general population of children for that age and sex. There are separate-sex norms for males and females. The test authors point out, for example, that although raw score ratings on the Aggression Scale tend to be higher for males than females, use

of separate-sex norms removes this difference and produces distributions of normative scores that are the same for both sexes. Whereas the Teacher Rating Scales should not be used generally for brain injury assessment, the Parent Rating Scales (PRSs) and the Self Report of Personality (SRP) are useful.

The SRP consists of statements that are responded to as true or false. It takes about 30 min to complete and has forms at two age levels: 8 to 11 years, and adolescents from 12 to 18 years. The child level has 12 scales and the adolescent level has 14 scales. Both levels have identical composite scores: school maladjustment, clinical maladjustment, and personal adjustment. An overall composite score, the Emotional Symptoms Index, is obtained.

The PRSs are a comprehensive measure of a child's adaptive and problem behaviors in the community and home settings. The PRSs use a four-choice response format and take 10 to 20 min for the parent to complete. There are three forms at three age levels: the preschool child, the child, and the adolescent. The PRSs produce a clinical profile that delineates the following behaviors: (1) hyperactivity, (2) aggression, (3) conduct problems, (4) anxiety, (5) depression, (6) somatization, (7) atypicality, and (8) withdrawal. Two composite scores are also generated that measure whether the child is externalizing or internalizing problems. Scales 1, 2, and 3 measure internalization of problems, and scales 4, 5, and 6 measure externalization of problems.

Since the BASC has the PRSs as well as the self-report from the child, interesting contrasts or difficulties within family structures due to the brain injury may often be determined. The weakness of this test is that currently the basic structure of clinical descriptors is based on the DSM-III-R rather than the more contemporary DSM-IV.

### **Minnesota Multiphasic Personality Inventory-Adolescent**

The *Minnesota Multiphasic Personality Inventory-Adolescent* (MMPI-A) was developed because many studies of the MMPI test instruments have demonstrated the importance of using adolescent norms for young people. The use of adult norms applied to adolescents tends to overpathologize or make adolescents appear more disturbed than they actually are. Thus, the MMPI-A is an outgrowth of the MMPI Adolescent Project Committee of the University of Minnesota, which was specifically appointed to develop the MMPI-A.<sup>90</sup>

The MMPI-A contains 478 items. All the basic clinical scale items, as well as those that are unique to the adolescent form, appear among the first 350 questions. Thus, scores for F2, F, VRIN, TRIN, the content scales, and the supplementary scales are not obtainable in the first 350 items, but require complete administration of the test booklet. The clinical sample for the normative base included 420 boys and 293 girls, ages 14 to 18. It is recommended that the MMPI-A be used with 14- to 18-year-olds. The grade level of the clinical sample ranges from 7 to 12, and all normative subjects were enrolled in school, although some were attending school in a psychiatric treatment facility. When scored on the basis of the original MMPI norms, this clinical sample produced clinical scale profiles that were very similar to those of the previous clinical sample used by Marks et al.<sup>91</sup> to develop the MMPI code-type data for adolescents.

It is thought to be possible that bright, mature 12- or 13-year-old adolescents can comprehend and respond validly to the MMPI-A. However, ethically it must be reported by the examiner that these age levels are outside the normative database. Also, for adolescents age 19, the MMPI-2 should be used rather than the MMPI-A. For 18-year-olds, the maturity level allows the clinician to make some judgment about whether to use the MMPI-A or the MMPI-2 during examination.

An essential requirement is adequate English language reading comprehension. This could prove especially troublesome for a youngster who was learning disabled or had ADD prior to traumatic brain injury. Alternative test instruments may be required for this group of youngsters. Some brain-injured youngsters may be too easily distracted, hyperactive, or impulsive to complete 478 items in a single testing session. Thus, frequent breaks may be required. The majority of

MMPI-A items are at the fifth- to seventh-grade reading level. The author recommends that all adolescents be screened for reading skill prior to administration of the test instrument.

The validity indicators contain some differences from those of the MMPI-2. Those that are similar to the MMPI-2 are the Cannot Say, L, F, K, VRIN, and TRIN scales. Two new validity scales, F1 and F2, are unique to the MMPI-A. The Cannot Say measures the total number of items that the adolescent failed to answer true or false. The L scale may be used as a measure of naive defensiveness in adolescents. F, the Infrequency Scale, is divided into a 33-item F1 scale and a 33-item F2 scale. The F1 scale is a direct descendant of the traditional F scale from the original MMPI. The F2 scale consists of items that occur in the latter half of the test booklet. Thus, the F1 and F2 scales for the MMPI-A may be used in an interpretive strategy similar to the one recommended for the F and Fb scales in the MMPI-2. Because all the F1 scale items appear in the first 350 items of the MMPI-A booklet, this measure provides a method for evaluating the acceptability of the response pattern for the basic MMPI-A scales. The F2 scale operates like the Fb scale of the MMPI-2 in that it provides an index of the acceptability of the test record in relation to the MMPI-A content and supplementary scales. F1 will enable the psychologist to determine the likelihood of significant symptom magnification or even malingering of psychological problems.

The K scale is a basic validity indicator in the MMPI-A, but few descriptors are available from the normative samples. The test manufacturer recommends that interpretation of K profiles with elevated T-scores (> 65) include a cautionary statement about the possibility of a defensive test-taking attitude. The test authors recommend that TRIN should be used to clarify elevations on this scale and psychological consultation will be necessary to complete this analysis. The VRIN and TRIN scales are new validity scales developed with the second edition of the MMPI-2. They are quite different from the traditional L, F, and K scales. VRIN and TRIN scores indicate the tendency of a person to respond to items in ways that are inconsistent or contradictory. TRIN is made up exclusively of pairs that are opposite in content. Thus, this scale can be used to determine whether the adolescent is acquiescent or nonacquiescent to true or false responses. VRIN is useful to determine if the adolescent is answering the questions carelessly or is confused. Moreover, it can be useful for determining symptom magnification or malingering. A high F1 with a normal or low VRIN is consistent with the adolescent understanding the responses and deliberately skewing the responses of the test items to represent either symptom magnification or malingering. A high elevation on VRIN accompanied by a high elevation on F1 may be consistent with a disorganized or confused adolescent who cannot attend to the test items or comprehend the test items. Psychological consultation is required for the neuropsychiatric examiner to fully use the validity scales on the MMPI-A.

The MMPI-A contains 10 clinical scales. These have the same names as the MMPI-2 or the original MMPI scales, and they include:

- 1 — Hs: hypochondriasis
- 2 — D: depression
- 3 — Hy: hysteria
- 4 — Pd: psychopathic deviate
- 5 — Mf: masculinity/femininity
- 6 — Pa: paranoia
- 7 — Pt: psychasthenia
- 8 — Sc: schizophrenia
- 9 — Ma: hypomania
- 0 — Si: social introversion

As is true for the interpretation of the MMPI and MMPI-2 with adults, the adolescent MMPI-A interpretation is often done by code type. The only published empirically developed code type for the MMPI-A was by Marks et al.<sup>91</sup> Archer and Klinefelter published code-type frequency data for

1762 adolescent patients who received the original form of the MMPI and were scored using the Marks et al. norms and the MMPI-A norms.<sup>92</sup>

The scoring and interpretation of the MMPI-A have options specific for adolescents that are not present for the adult interpretive schemes. For instance, the potential for school problems can be determined by two of the MMPI-A content scales (A-SCH and A-LAS). Several other MMPI-A scales also include school problems (see the MMPI-A 1992 manual). Scale 0 (Si) and its subscales are helpful for describing problems of social relationships. These of course occur very frequently in adolescents following traumatic brain injury. Predictions about family problems can be made from the A-FAM scale. Alienation (A-ALN) and cynicism (A-CYN) are covered by the MMPI-A content scales. Negative peer group influences can be inferred from elevations on the PRO scale, given its item content. The IMM scale also provides information relating to interpersonal style and capacity to develop meaningful relationships. Elevations on the A-TRT scale can be interpreted as an indication of the presence of negative attitudes toward mental health treatment that may interfere with building a therapeutic relationship.<sup>90</sup> As with the adult MMPI-2, psychological consultation is recommended when using the MMPI-A.

### **Multiscore Depression Inventory for Children**

The *Multiscore Depression Inventory for Children* (MDI-C) is a 79-item questionnaire in the form of brief sentences presented in a true–false response format. The administration time is about 15 to 20 min. This test instrument is standardized for ages 8 to 17, and it allows children to indicate their own feelings and beliefs about themselves. It is an unusual test in that it is the first behaviorally oriented test for children that was written by children in their own words.<sup>93</sup> The MDI-C is reportedly useful both as a screening instrument and to identify high-risk children within clinical assessments. It yields scores on eight scales, as well as a total score measuring the general severity of depression. It may be scored on a computer, by sending the score sheet by fax to the manufacturer, or by mail-in scoring.

The MDI-C scales are anxiety, self-esteem, sad mood, instrumental helplessness, social introversion, low energy, pessimism, defiance, and total. The Anxiety Scale measures cognitive and somatic aspects of anxiety. The Self-Esteem Scale reflects children's perceptions of themselves. The Sad Mood Scale is basically what it says. The Instrumental Helplessness Scale measures children's perceptions of their abilities to manipulate social situations in order to receive ordinary benefits. The Social Introversion Scale reflects the tendency to withdraw from social situations and social contact. The Low Energy Scale measures cognitive intensity and somatic vigor. The Pessimism Scale gauges children's outlook to the future. The Defiance Scale measures irritability and other behavior problems. The Total Scale sums all 79 items, including a Suicide Risk Indicator, and is an overall measure of depression. The scale items have a third-grade reading level. Most children have few problems understanding the content, since children wrote it. There are scales to determine faking good and faking bad as response biases. Children are more likely to have a defensive response or a "faking good" response, as they may be worried how adults or professionals will react to their problems. Children with high scores on the Infrequency Index are either "faking bad" or suffering extreme forms of depression. This instrument includes scales that address features widely agreed to accompany depression or contribute to it. The scores are displayed as T-scores exactly analogous to the T-score presentation with the MMPI-A. On this test instrument, the most reliable and valid measure of depression in a child is the total score of the MDI-C. This score is a measure of severity of childhood depression. Children with total scores greater than 65T have sad or blue moods often. They may be irritable, helpless, hopeless, and lack energy. Vegetative signs of depression may be present. On the subscale for suicidal ideation, children with total scores above 65T should be carefully evaluated for suicidal behaviors and ideas. Item 45 from this test instrument contains a Suicide Risk Indicator ("I have a suicide plan."). Furthermore, the test manufacturer recommends evaluating the child's answers to item 5 ("I think about death a lot."),

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**TABLE 7.2**  
**Child Behavioral Tests That Are Useful in Traumatic Brain Injury**

<b>Mood/Affect</b>
Adolescent Psychopathology Scale (12–19 years)
Behavioral Assessment System for Children (2½-18 years)
Minnesota Multiphasic Personality Inventory-Adolescent (14–18 years)
Multiscore Depression Inventory for Children (8–17 years)
State–Trait Anxiety Inventory for Children (9–12 years)

<b>Neurobehavioral Function</b>
Neurobehavioral Functioning Inventory (16–82 years)

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item 11 (“I hate myself.”), item 26 (“I do not want to live.”), item 36 (“I worry about death.”), and item 56 (“No one would care if I died.”).

### **State–Trait Anxiety Inventory for Children**

The *State–Trait Anxiety Inventory for Children* (STAIC) was initially developed as a research tool for the study of anxiety in elementary school children. It is comprised of separate, self-report scales for measuring two distinct anxiety concepts: state anxiety (S-Anxiety) and trait anxiety (T-Anxiety). This measurement is very similar to the adult test described previously (STAI). The original STAIC was constructed to measure anxiety in 9- to 12-year-old children, but it may also be used with younger children who possess average or above-average reading ability and with older children who are below average in their reading ability.<sup>94</sup>

The S-Anxiety Scale measures transitory anxiety states. These are subjective, consciously perceived feelings of apprehension, tension, and worry that vary in intensity and fluctuate over time. On the other hand, the T-Anxiety Scale measures a relatively stable individual difference in childhood anxiety proneness. High T-Anxiety children are more prone to respond to situations perceived as threatening with elevations in S-Anxiety intensity than low T-Anxiety children. Thus, this test instrument may be useful in the highly traumatized child who is also being screened for possible posttraumatic stress disorder.

No internal validity scales are used for this test instrument. There are foreign language adaptations and translations of the test that are available from the manufacturer. There are a wide variety of languages available, including Hindi, Chinese, Czech, German, Greek, Hebrew, Japanese, Russian, Spanish, and Turkish, among others. A Spanish language version is also used in Puerto Rico, Mexico, and the Mexican-American population of Texas. The East Indian versions have been standardized with college students at Punjab University in India. The scores are provided as T-scores with the usual mean of 50 and a standard deviation of 10. [Table 7.2](#) lists behavioral tests that are useful during evaluation of children.

### **AGGRESSION**

As noted previously, with regard to personality changes following traumatic brain injury, the labile subtype is the most common and the disinhibited aggressive subtype is the second most common.<sup>95</sup> In adults, the impulsive aggressive types have a higher incidence of premorbid aggressive behaviors.<sup>31</sup> For children who are aggressive following traumatic brain injury, while they have cognitive deficits as well, their psychosocial adjustment is quite poor if aggressive traits are present. Traumatically brain-injured children demonstrate significantly lower levels of self-esteem and adaptive behavior, and have high levels of loneliness. Their maladaptive behaviors often contain aggressive and antisocial behaviors. These, in turn, have significantly detrimental effects on children’s abilities

to learn and be maintained in a classroom setting.<sup>96</sup> The treatment for children with brain injuries is quite complex, and generally requires interventions by child psychiatrists and other specialists in child behavior. Specific interventions include contingency management, stimulus control, problem solving, social skills training, relaxation training, anger management, and parent–child training.<sup>97</sup>

## **PSYCHOSOCIAL FUNCTIONING IN BRAIN-INJURED CHILDREN**

There is substantial evidence that head-injured children are not representative of the pediatric population with respect to psychosocial variables.<sup>98</sup> Children who grow up with physical disabilities or illness face challenges when trying to fit in with peers.<sup>99</sup> Those children who have brain injuries may face even greater social challenges due to the direct impact of the injury or illness on brain regions that subserve abilities critical for social interaction, such as the ability to discern affect expressed nonverbally or to generate and implement effective strategies to deal with different interpersonal situations (right hemisphere-controlled nonverbal communication and executive function).<sup>100</sup> Children with traumatic brain injuries are at risk for both acute and chronic social problems.<sup>101,102</sup> Traumatically brain-injured children seem as capable of judging the appropriateness of their behaviors in generating response options as typically developing peers. However, individual children following traumatic brain injuries are more likely to demonstrate social problems related to deficits of emotional intelligence, as discussed previously. The Social Knowledge Interview has been used to identify children with social skills deficits following traumatic brain injuries in order to develop effective rehabilitation strategies for them.<sup>100</sup>

Many times, when children are traumatically brain-injured, they sustain other bodily injuries as well. The psychosocial impact of pediatric injuries can be quite substantial upon the child and the child's family. While orthopedic injuries alone cause caregiver burdens and family stresses, the co-occurrence of traumatic brain injuries and orthopedic injuries plays a significantly negative role in family adjustment, particularly in children ages 6 to 12 years. Moreover, in those families who were dysfunctional prior to the trauma, traumatic brain injuries and concurrent orthopedic injuries have a magnified impact.<sup>103</sup>

## **THE DYNAMICS OF TRAUMATIC BRAIN INJURY WITHIN THE FAMILY OR WITH SIGNIFICANT OTHERS**

### **THE ADULT**

In the mid 1970s, the internationally recognized brain trauma center in Glasgow became concerned with the psychological effects of head injury. When their findings were first published in 1983,<sup>104</sup> they noted that the psychiatric consequences of head injury had been largely neglected. They further noted the lack of information regarding head injury in children and the largely neglected area of family and social consequences. More recent studies indicate that severe traumatic brain injury is a source of considerable caregiver morbidity, even when compared with other traumatic physical injuries. Caregivers of the severe traumatic brain injury group have persistent stress associated with the patient's injury. The risk of clinically significant psychological symptoms for caregivers of brain-injured children is twice that for caregivers with orthopedically injured children.<sup>105</sup>

The impact upon interpersonal relations in families following traumatic brain injury is substantial. Reviews of the literature in this regard document the considerable problems acquired brain injury causes for the survivor's family and other close relationships, and the correspondingly significant inflated rate of separation and divorce.<sup>106</sup> A study in the U.K. assessed the extent to which brain injury affects marriages and close relationships. This study evaluated 131 adults with traumatic brain injuries in order to determine the incidence of divorce or separation. Forty-nine percent of the sample reported divorce or separation from their partners 5 to 8 years following their traumatic brain injuries. Factors that positively predicted separation or divorce were the level of

severity of injury and the shortness of the relationship prior to the injury.<sup>107</sup> Risk to relationships within family systems has been assessed in Australia, and findings were similar to those in the U.K. The severity of the injury and residual neurobehavioral function both inversely predicted family and relationship functioning when studied by Douglas and Spellacy.<sup>108</sup>

Caregiver stress often manifests itself as depression. Neurobehavioral disturbance in the person with the injury is the strongest predictor of whether the caregiver will develop a psychiatric disorder. Moreover, the level of social support shows a direct and linear relationship to family functioning.<sup>109</sup> The prevalence of major depression is high in caregivers of individuals who have sustained brain injuries. If left untreated, the depression may interfere with the capacity to provide care and with the rehabilitation process. The neuropsychiatric examiner should carefully assess both the current and preaccident mood states of primary caregivers where appropriate.<sup>110</sup> Some evidence suggests that the greater the number of adverse events or effects occurring in the patient as well as the patient's impact upon the family, the more likely the caregiver is to develop depression.<sup>111</sup> Some evidence also suggests that the rate of depression in caregivers may exceed 50% when measured on the Beck scales.<sup>112</sup> Other studies have shown that the rate may be as high as 60% in caregivers attempting to meet the needs of a traumatically brain-injured loved one.<sup>113</sup>

It has been questioned whether injured persons can accurately assess their cognitive and behavioral states relative to what their caregivers observe. Models to test this have used the *Neurobehavioral Functioning Inventory* (NFI), which is comprised of six scales with items describing symptoms and daily living problems. The findings indicate general agreement between family members and patients regarding everyday problems within the patient. Use of this inventory by a group at the Medical College of Virginia found that the results did not support that patients tend to underestimate their difficulties, and the agreement level between patient and family related to injury severity and outcome seemed fairly good.<sup>114</sup> Attempts to directly measure caregiver distress have used the *Caregiver Appraisal Scale*. Preliminary support for using this instrument in caregivers of adults with traumatic brain injuries was obtained, and it demonstrated adequate concurrent validity.<sup>115</sup>

In performing a clinical examination of a patient and family, the question is how to help. In order to help, one must first determine the nature of care needs, the stress and burden experienced in the family or caregiving home, and how individuals caring for the injured party cope with caregiving demands.<sup>116</sup> The main goal for intervention is to intercede in a way that will reestablish life cycle trajectories for caregivers, as well as reintegrate affected individuals and their families into a larger social system.<sup>117</sup> One potentially useful intervention has been the development of a mentoring program where individuals who have been through the stresses of caring for a traumatic brain-injured person mentor individuals with newly injured family members.<sup>118</sup> Lastly, during evaluation of brain-injured patients and their families, the particular gender difference for brain-injured women must be considered. A woman's roles as wife, mother, and daughter are likely to result in a different constellation of family dynamics when traumatic brain injury is introduced, compared with that of the male. This gender difference has been little studied, and further research is necessary.<sup>119</sup>

## THE CHILD

There is good evidence that family functioning influences behavioral adjustment and adaptive function in brain-injured children.<sup>120</sup> Parents of children who have sustained traumatic brain injuries report higher levels of psychological symptoms than parents of children with orthopedic injuries. Traumatic brain injury in a child is a source of considerable caregiver morbidity, even when compared with other traumatic injuries.<sup>105,121</sup> Parents of children who suffer brain injuries are often surprised by the extent to which work and family finances are disrupted. They have significant difficulty maintaining regular work schedules, and injury-related financial problems are common. The highest risk for work and financial problems occurs in families of children with severe injuries who have between four and nine impairments or among those parents whose children were hospitalized for more than 2 weeks and then not discharged to home.<sup>122</sup> If either parent is significantly

distressed at 6 months following the child's brain injury, this predicts that the child herself will have significant behavioral problems at 12 months postinjury.<sup>123</sup> Interestingly, since siblings are rarely caregivers, no statistical differences were found in depressive symptoms, self-concept, or behavior between siblings and their classmates of those youngsters who had a brain-injured sibling in their home 3 to 18 months after injury.<sup>124</sup>

The strongest influence on family functioning after childhood traumatic brain injury is preinjury family functioning. One great stressor in this regard is the development of "novel" psychiatric disorders in the child. As noted previously in this text, traumatic brain injury in childhood predicts a much higher likelihood of developing psychiatric injury than in children who have not had brain injury (see [Chapter 2](#)). These factors play some role in predicting which families are at increased risk for family dysfunction after a child traumatic brain injury.<sup>125</sup> Moreover, the lower the functioning of a family prior to the child's brain injury, the more significant will be the recovery problems of the child following injury, as one might expect.<sup>126</sup> Thus, the neuropsychiatric examiner when examining a brain-injured child, should carefully determine, if possible, the levels of family burden from internal dysfunction within the family that were present prior to the child's injury.<sup>127</sup> Lastly, what happens to children and family dynamics when a parent is brain-injured? Data indicate that parents with traumatic brain injuries provide less goal setting, less encouragement of skill development, less emphasis on obedience to rules and orderliness, less promotion of work values, less nurturing, and lower levels of active involvement with their children after injury. However, spouses of individuals with traumatic brain injuries, compared with their counterparts, reported less feelings of warmth, love, and acceptance toward their children. Parental traumatic brain injury has select consequences for all family members, particularly, their children.<sup>128</sup>

## MEASUREMENT OF PATIENT NEUROBEHAVIORAL FUNCTION WITHIN THE FAMILY

### Neurobehavioral Functioning Inventory

The *Neurobehavioral Functioning Inventory* (NFI) was developed in three phases. It grew out of the 105-item *Brain Injury Problem Checklist*, developed in the 1980s. This inventory was based on face validity and organized into five categories: somatic, cognitive, behavior, communication, and social problems. Patients and family members rated the frequency of patient problems. The present NFI consists of two forms, one for patients and one for family members or other knowledgeable informants. Both forms consist of 76 items on a 5-point Likert scale that measures the frequency of behaviors exhibited by the patient. The Likert-type response choices include never, rarely, sometimes, often, and always.<sup>129</sup>

It is essential to attain responses from both the patient and a relative. Differing perspectives may be useful to the examiner. When more than one informant is available, the examiner may consider soliciting the opinion of the person who knows the patient best. This usually will be the primary caregiver, but examiners may wish to solicit responses from different family members and compare their answers. The age range for administration is 16 to 82 years. However, this inventory has an interesting component in that it is standardized to accept responses from patients who were ages 4 to 81 at the time of their injury. The standardization sample was also multiethnic and comprised of varying levels of brain injury severity existing between 0 and 195 days postinjury.

The NFI contains six clinical scales: (1) depression, (2) somatic, (3) memory and attention, (4) communication, (5) aggression, and (6) motor. The data are presented as T-scores with a mean of 50 and a standard deviation of 10. The examiner may find it useful to look at responses to individual test items, as they offer a wealth of information regarding overall neurobehavioral functioning.



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# 8 Neurobehavioral Analysis and Treatment Planning Following Traumatic Brain Injury

## INTRODUCTION

The first seven chapters of this text have formed the bases upon which the database is collected for a neurobehavioral analysis following a neuropsychiatric examination of a traumatically brain-injured patient. [Table 8.1](#) outlines a suggested schema whereby the examiner performing a neuropsychiatric assessment can systematically collect data for analysis to determine and establish the neuropsychiatric deficits in a patient following traumatic brain injury (TBI) and to further assist in the treatment planning necessary for addressing the cognitive and behavioral issues resulting from trauma to the brain. By no means is this approach the only methodology whereby a proper neuropsychiatric assessment of a brain-injured person can be completed. However, it is empirically tested by more than 20 years of systematic assessment of injured persons, and it represents a database of more than 2500 traumatically brain-injured people.

## ANALYSIS OF THE DATA

### THE INJURY RECORD

As with most journeys, it is best to start at the beginning. Most instances of brain trauma in the U.S. are managed initially by an emergency medical squad.<sup>1</sup> Within the emergency medical squad records, one generally will find the *Glasgow Coma Scale* (GCS). However, it is not always stated as such explicitly. The examiner should look for either “GCS” or “EVM.” As noted in [Chapter 1](#), the initial neurological status of the patient will be represented as a score of 3 to 15, with 3 representing the most severe presentation and 15 the most promising. One should also look for a trauma score. This often will be represented in a triage form, and it is more reliable than the original trauma score proposed by the American College of Surgeons in 1981.<sup>2</sup> The triage form of the Revised Trauma Score (RTS) is scored from 12 (best possible score) to 0 (worst possible score). [Table 8.2](#) represents the triage RTS, which is no longer copyrighted and is found on the vast majority of emergency medical squad or ambulance run sheets currently used in the U.S.

The ambulance run sheet or emergency medical squad records should be further reviewed to determine if the patient was intubated at the scene due to respiratory distress or failure. Moreover, review should establish whether intravenous fluids were required and whether manitol or furosemide, or other mechanisms, were used at the scene to lower intracerebral pressure. Certain lazaroids (21-aminosteroids) and other experimental agents such as free radical scavengers may be used in some trauma protocols that are currently using experimental substances, under trauma center control, in the initial management of the brain-injured patient in an effort to reduce brain morbidity. Prehospital care of the head-traumatized patient remains in flux and is constantly

**TABLE 8.1**  
**Schema for Clinical Neuropsychiatric Data Collection and Review**

Ambulance report:	Review for level of consciousness, Glasgow Coma Scale
Emergency department:	Review for Glasgow Coma Scale, trauma score, and level of mental/cognitive function; What injuries were documented? What is the result of neuroimaging?
Hospital record:	Review ICU records, neurosurgical/neurological consultation, and any subsequent neuroimaging. Is there evidence of secondary causes of brain injury?
Rehabilitation record:	Review deficits at admission and Rancho level at discharge; review reports of speech/language, occupation therapy, and physical therapy assessments. Was neuropsychological assessment performed?
Outpatient treatment:	Review continuing treatments by physiatrists, neurologists, psychiatrists or psychologists. Is speech/language therapy still in place?
Neuropsychiatric examination:	Review history, mental status examination, neurological examination, brain imaging, standardized mental assessment, and laboratory testing.

**TABLE 8.2**  
**Revised Trauma Score**

Glasgow Coma Scale (GCS)	Systolic Blood Pressure (SBP)	Respiratory Rate (RR)	Score
13–15	> 89	10–29	4
9–12	76–89	> 29	3
6–8	50–75	6–9	2
4–5	1–49	1–5	1
3	0	0	0

*Note:* RTS score = GCS + SBP + RR; best score = 12; worst score = 0.

improving. The combination of rapid triage and transportation by appropriate medical personnel to a neurotrauma center has been shown to reduce mortality in almost every country where it has been instituted.<sup>3,4</sup> If possible, the examiner should review whether extraction from a vehicle was required and attempt to determine the nature of the trauma. This in no way is meant to imply that a neuropsychiatric examiner should be an accident reconstruction specialist, but clearly it is worthwhile knowing what level of trauma was sustained by the body of one's patient, as that often is a direct indicator of the severity of injury. It is one thing to trip and fall over an object on the ground and strike one's head; it is another to fall from a five-story building under construction while one is employed as an ironworker. Moreover, how the person was transported to the hospital emergency department is also valuable information. Most large trauma centers send helicopters for severely injured persons and transport less severely injured persons by motor vehicle, even if helicopter service is available.

The next record to review is that of the emergency department of the hospital receiving the traumatized patient. Again, the examiner should review the GCS, if available. It is important to determine if there has been a change, either up or down, in the severity level of the GCS. Obviously, if one's GCS improves during transport from the accident scene to the emergency department, this generally accrues to the benefit of the patient and the eventual outcome. On the other hand, a deteriorating GCS or RTS may be of prognostic importance and also may indicate evolving morbidity from such issues as epidural hematoma or cerebral edema (see Case 2). Generally, the emergency department will order computed tomography (CT) head imaging, and the examiner should review this. The history and physical examination should be noted, and in particular, the

examiner should determine if focal neurological signs were present. Issues of secondary brain injury may be established at this point in the record by reviewing for the presence of hypoxemia, blood loss, or hypotension. Moreover, the emergency department will generally establish the presence of other comorbid conditions that may both indicate the level of trauma sustained by the patient and be a harbinger of potential complications. Thus, it is important to establish if the brain-injured patient sustained multiple orthopedic injuries, injury to internal organs, or other causes of secondary injury, as described in [Chapter 2](#). Again, the examiner should determine whether intubation was required. Often, a brain-injured person does not require intubation at the accident scene, but due to deteriorating neurological status, reduction of respiratory drive systems, or chest trauma, intubation is later required.

Most modern emergency departments follow algorithms for the management of head-injured persons.<sup>5</sup> Most emergency departments will order a blood alcohol level and urine drug screen for abused substances at the time the patient presents. The examiner should take note of these findings, as the issue of substance abuse and subsequent neuropsychiatric treatment planning may be dictated by the results of these tests.

As discussed in [Chapter 5](#), the single most important radiological study to obtain in a patient with a severe head injury is a CT scan of the head.<sup>6</sup> While the neuropsychiatric examiner is not expected to order the emergency department films and review them after the injury, it is generally an important element of a complete neuropsychiatric examination of post-brain trauma to review radiological reports obtained at the initial trauma evaluation. Depending on the level of brain imaging required by the neuropsychiatric examination, the films themselves may prove to be important for comparisons. Other markers of significant brain trauma noted in the emergency department may be found within angiography and magnetic resonance imaging (MRI) records, transcranial Doppler studies, blood transfusion, hyperventilation, hypertonic fluid infusion, fluid resuscitation, and pharmacological paralysis procedures.<sup>5</sup>

After the patient leaves the emergency department, if admitted to the hospital, he or she is generally housed in a general surgical unit, specialized trauma unit, or neurosurgical intensive care unit (ICU). The level of hospital care available at the receiving institution, and the availability of neurosurgeons and other support staff, generally will dictate within which care setting the patient is managed. It is important for the neuropsychiatric examiner to review intensive care monitoring records of patients admitted to the hospital. As noted in [Chapter 1](#), issues such as vascular failure, intracranial hypertension, and brain shift or herniation may dictate high levels of cardiopulmonary, fluid and electrolyte, and nutritional and metabolic management of the patient. Records should be scanned to determine if these support modalities were required, as, again, these are important markers as to the severity of the actual head injury and markers of contributing factors that may impact prognosis and healing.<sup>7-9</sup> Moreover, as noted in [Chapter 5](#), it is important to review whether electrophysiological monitoring was performed in the trauma unit and whether seizures were noted requiring either antiseizure prophylaxis or use of antiepileptic drugs (AEDs).

In the hospitalized patient, the records following the ICU should be reviewed for ancillary therapies that may point to significant neurological or neurocognitive deficits present in the patient after injury. For instance, was speech–language therapy required? Was there need for occupational therapy intervention? Was physical therapy required due to hemiparesis, spasticity, or orthopedic injury? Was psychiatric intervention required because of severe behavioral difficulties, agitation, or aggression? Was neurological assessment or neurophysiological monitoring required due to peripheral nerve injuries or seizures? What was the patient’s level of self-care?

A patient injured severely enough to require rehabilitation following the acute care hospitalization is generally transferred to the brain injury rehabilitation unit, if available. These records should be scrutinized carefully, as they often contain a wealth of information that directly applies to neuropsychiatric assessment and treatment planning. Most brain injury units will provide an initial evaluation, usually by a physiatrist. This physician will take a complete history, review the pre-rehabilitation hospital records, and perform what necessary physical and laboratory assessments are



required. The neuropsychiatric examiner should review the rehabilitation record to determine if the patient required audiometry or vision screening. Brain injury physiatrists are skilled at determining cognition levels, and these data usually are entered into the patient record (see information on the Rancho Scale in [Chapter 1](#)). Most brain injury treatment units supply occupational, physical, and recreational therapies, and these treatments or assessments should be reviewed. A careful cataloging of residential skills and activities of daily living is usually performed within brain rehabilitation units, and family interactions are generally documented. These records should be reviewed. While cognitive therapy is often the major modality provided in a brain injury rehabilitation unit, the speech and language pathologist also plays a crucial role. There is some overlap, of course, between these two disciplines and their approaches to the brain-injured patient. Specific recommendations for care or treatment programming will be found usually in the nursing record and should provide significant assistance to the neuropsychiatric examiner during evaluation.<sup>10</sup>

The rehabilitation record will usually contain significant process notes and documentation of the procedures used during cognitive rehabilitation. Cognitive rehabilitation can be divided generally into two levels: functional and generic. At the functional level, the patient is trained in activities necessary to the orderly execution of practical daily living skills, such as dressing or preparing meals. At the second level, generic cognitive skills such as attention, memory, and executive functions are remediated.<sup>11,12</sup>

The rehabilitation medical record for the severely traumatized patient will also contain important information as to the level of spasticity in the motor-impaired patient; the presence of swallowing, feeding, or reflux difficulties; bowel and bladder management; respiratory management; heterotopic ossification; central dysautonomia; posttraumatic epilepsy; neuroendocrine disorders; communicating hydrocephalus; and musculoskeletal complications.<sup>13</sup> Careful review of the rehabilitation record regarding the aforementioned issues will be most helpful to the neuropsychiatric examiner within the overall assessment of the patient, as well as useful in treatment planning for postinjury neuropsychiatric issues.

## THE NEUROPSYCHIATRIC EXAMINATION DATABASE

### History

In [Chapter 3](#), we learned the importance of taking a comprehensive adult brain injury or child brain injury history. During analysis of the data, the neuropsychiatric examiner should review the history she obtained and catalog the posttrauma neurocognitive and behavioral symptoms expressed by the patient. The current activities of daily living should be reviewed to enable the examiner to understand the impact of the injury upon the patient's daily functioning. It is important to categorize the symptoms expressed by the patient that are referable to cognitive dysfunctions. These include complaints within the domains of attention, speech and language, memory and orientation, visuospatial and constructional functions, executive abilities, affective and mood changes, thought processing, and risk of harm to self or others. Since the neuropsychiatric examiner may be well down the line in the list of care providers to the brain-injured patient, it is important also to review what behavioral treatments may have occurred in the patient prior to the neuropsychiatric examination.

The past medical history will help the examiner focus upon past or current medical problems that may impact treatment planning. Illnesses such as hypertension or diabetes may have a direct impact on brain functions. The past neuropsychiatric history may uncover such issues as childhood attention deficit disorder or preinjury antisocial behaviors or substance abuse. A preinjury history of epilepsy clearly may have a major impact upon neuropsychiatric strategies and treatment planning. The development of the family history helps to organize data analyses around possible genetic factors that may play a role in the treatment of traumatic brain injury (e.g., a strong family genetic loading of Alzheimer's disease). The social history provides the examiner with a scaffolding to understand and predict future psychosocial problems. Demographics and social factors are highly

important in the analysis of problems that develop following brain injury, as noted in [Chapter 7](#). The review of systems will enable the examiner to focus thinking on current symptomatic issues that may impact treatment. Many of these posttrauma problems will have been discovered during review of the rehabilitation records in the more severely injured patient. In those patients who did not require hospitalization or brain trauma rehabilitation, more careful attention to the current review of systems by the neuropsychiatric examiner will be required.

With the child, a more neurodevelopmental analysis of data clearly is required. It is probably important for the neuropsychiatric examiner to access the child's pre- and postinjury academic records. Not only do these records help establish cognitive and behavioral baselines, but they are critical in the longitudinal evaluation of academic functions of the child. As noted in [Chapter 6](#), many substantial issues may arise within the education of children following traumatic brain injury. The examiner should pay careful attention to the history of behavioral and neurocognitive symptoms following brain injury. As noted in [Chapters 3, 4, and 6](#), these disorders play a different role in the developing child than similar disorders would play in the rehabilitation or treatment of an adult patient. Children are "works in progress," and as previously noted in this text, the younger the child, the more at risk the child is for adverse cognitive and behavioral outcomes following traumatic brain injury.

A present analysis of the child's activities of daily living, including those within school settings, is important to establish in the data analysis. This data can be analyzed vis-à-vis the past pediatric history and past neurodevelopmental history. It is important to remember that children with learning disorders or preinjury psychiatric conditions, such as attention deficit disorder with hyperactivity, are more vulnerable to the deleterious effects of traumatic brain injury than children with more normal preinjury neuropsychiatric profiles. The genetic family history of the child is as important as it is for the adult. Review of systems of the child is taken in a straightforward manner. The social history of the child may play particular prominence in treatment planning, as the child is a dependent person by definition and actuality. Thus, the examiner should search for the complicating factors of divorce, neglect, poverty, poor nutrition, poor housing, etc.

## **Mental Status Examination**

Within [Chapters 2 and 4](#), we learned of particular neuropsychiatric disorders that may present following traumatic brain injury. The adult mental status examination should provide the examiner face-to-face evidence of changes consistent with potential neuropsychiatric disorders that then will require further elucidation using neurocognitive and behavioral measures. From the standpoint of analysis, the mental status examination following brain injury can be divided into two basic components: cognitive and behavioral. The examiner should review his notes and data acquired during the face-to-face mental examination and determine which domains of cognitive function appear abnormal. Thus, a careful analysis of the cognitive aspects of the adult mental status examination should reveal to the examiner the patient's appearance, level of consciousness, attentional abilities, speech and language function, memory and orientation abilities, visuospatial and constructional abilities, and executive function. Discerning intellect on a face-to-face mental examination can be performed only in the most general sense, and specific measures are required to identify intellectual changes, as noted in [Chapter 6](#). However, by reviewing the data obtained using techniques from [Table 4.13](#), the examiner can categorize the impairments present, at least in a qualitative sense. The cognitive data collected from the mental status examination is then used with measurements obtained during the neurocognitive examination to describe the patient's cognitive deficits.<sup>14,15</sup>

As the examiner reviews the adult mental status examination data, the presence of obvious behavioral abnormalities should be apparent. Changes in affect and mood as well as alterations in thought processing, content, and perception should be readily discernible. As discussed in [Chapter 4](#), it is important to determine possible risks to self or others. Following the face-to-face adult mental

status examination, the neuropsychiatric examiner should be able to formulate qualitative problems in the realms of affect and mood, aggression and hostility, formal thought impairment, perceptual distortions, and suicidal or homicidal risk. The qualitative determination of deficits in these areas then can be compared with specific measurements of adult behavior, as noted in [Chapter 7](#).<sup>15-17</sup>

A review of the cognitive data collected during the child examination is performed in the same manner as that in which the mental status data from the adult examination are processed. The analysis of the data, of course, is subject to the age level of the child at the time of examination.<sup>18,19</sup> As with the adult, the examiner should review the mental status examination for childhood cognitive domains of attention, speech and language, memory and orientation, visuospatial and constructional abilities, and executive function. The deficits noted on face-to-face examination should be recorded and compared with measurements made during the neurocognitive examination of the child. Obviously, the neurocognitive measurements should confirm or refute apparent mental status deficits.

Behavioral assessment of the child during face-to-face mental status examination should focus upon affect and mood as well as thought processing, content of thought, and perception. As we learned in [Chapter 4](#), stability of moods normally is evident in children by ages 2 or 3 years. For screening mood in children, Weinberg et al.'s simple questions are included in the "Affect and Mood" subsection under "The Child Mental Examination" section in [Chapter 4](#).<sup>20</sup> Evaluation of the thought process and content of thought in the very young child is extremely difficult. The examiner may have to fall back on the collection of historical data from parents or caretakers of the child in order to determine the presence of thought distortions. The use of the *Kiddie Formal Thought Disorder Rating Scale* (K-FTDS) may assist to determine thought pathology in the very young child (see the "Thought Processing, Content, and Perception" subsection in "The Child Mental Examination" section in [Chapter 4](#)).

## Neurological Examination

The examiner should review her notes of the neurological examination. It will be necessary to correlate any focal neurological deficits with the neuroimaging, neurocognitive, and behavioral data. For instance, an obvious hemiparesis should correlate with dysfunction of neurocognitive modalities and behaviors known to be regulated or induced in the contralateral cerebral hemisphere. Cranial nerve dysfunctions may be related to facial and skull trauma and bear no direct relationship to intracerebral lesions. The presence of abnormal involuntary movements may be a marker for subcortical injury. Obviously, if the patient has an upper motor neuron lesion, one would expect spasticity and hyperreflexia in the contralateral arm and leg. However, it is not the role of the neuropsychiatric examiner, in most instances, to delineate neurological deficits. This function is best left to neurosurgeons, neurologists, and psychiatrists. However, it is important for the examiner to make note of focal neurological deficits, as she may have a direct impact upon the cognitive and behavioral treatment planning process and the needs of the patient. "The Adult Neurological Examination" section in [Chapter 4](#) provides further delineation of potential neurological deficits seen following traumatic brain injury.

With the child, "The Child Neurological Examination" section in [Chapter 4](#) outlines possible neurological deficits seen following traumatic brain injury. Again, the neuropsychiatric examiner should use neurological deficits in children as markers for injury and leave her expert delineation and treatment to those physicians better qualified to deal with focal neurological deficits in children. On the other hand, as with the adult, these deficits may have substantial impact upon the neuropsychiatric treatment planning process. Since most brain-injured children are of school age, clearly there could be an impact upon the educational process from these deficits, and the neuropsychiatric examiner must take this into account. It is also noteworthy that the physically injured child presents special challenges to parents or caregivers, and these may have a different importance within the treatment planning process than would those same deficits in the adult.<sup>21</sup>

## Brain Neuroimaging

Review of imaging obtained at the time of the acute brain trauma has been discussed previously within the review of past medical records. Imaging obtained by the neuropsychiatric examiner will, in most instances, follow the general guidelines of [Chapter 5](#). It is incumbent upon the examiner to review imaging obtained during the course of the neuropsychiatric examination with the radiologist, nuclear medicine physician, or neurologist who assisted with interpretation of the structural or functional neuroimaging or the electroencephalogram (EEG). It is important for the examiner to note whether there have been interval changes in the neuroimaging since the original accident. For instance, has a previous subdural hematoma resolved? Is there evidence of neurodegeneration, encephalomalacia, or hemosiderin deposits present during the neuropsychiatric examination that were not observable at the time of the acute injury? Has sufficient time elapsed following the trauma that now there is evidence of atrophy or *exvacuo* ventricular dilatation? Is there evidence of subdural hygromas or *exvacuo* areas following necrosis of brain tissue? These findings, if present, should alert the examiner to review the cognitive examination and the behavioral examination to determine if structural or functional changes presented by neuroimaging correlate with cognitive or behavioral changes.

The determination of neuroimaging abnormalities within the neuropsychiatric examination is important for treatment planning and prognostic considerations. It may be necessary to assist the patient with disability determination, workers' compensation, or the Social Security Administration. Notable areas of injury found within the neuroimaging examination may prove helpful in explaining cognitive and behavioral deficits that require pharmacologic, psychotherapeutic, or family or caregiver interventions.

In most instances, within the acute care of the traumatized patient, CT imaging, rather than MRI, will have been obtained. As noted in [Chapter 5](#), the neuropsychiatric examiner is most likely to obtain an MRI of the patient's brain within the context of the structural neuropsychiatric examination, in particular if this has not been obtained previously. If the MRI insufficiently assists the examiner with explaining neuropsychiatric deficits, it may be necessary to proceed to single-photon emission computed tomography (SPECT) or positron emission tomography (PET) imaging. If these images are obtained, they should be reviewed carefully to determine if functional disturbances are present in areas not detected by MRI or CT. Moreover, the functional imaging should be compared to the structural imaging to determine if there is a greater extent of functional damage peripheral to the structural damage (see [Chapter 5](#)). This is usually the case. An EEG should be obtained by the neuropsychiatric examiner if the patient has posttraumatic seizures or if the neuropsychiatric history suggests markers of functional organic disturbances in awareness.<sup>16,22</sup>

## Neurocognitive Measures

Some neuropsychiatric examiners are quite facile with neuropsychological and neurocognitive test data, whereas others are not. Those who are inexperienced in this area should consult with the psychologist or neuropsychologist who undertook the neuropsychological measures requested by the neuropsychiatric examiner. A short consultation with the psychology professional will be of marked assistance to the neuropsychiatric examiner with limited experience in the use of neuropsychological test data and neuropsychological consultation. For those more experienced psychiatrists and neuropsychiatrists, a review of the psychological test data and comparison vis-à-vis the neuropsychiatric history, mental status examination, neurological examination, and brain imaging data will be required.

The psychiatrist or physician examiner should first review measures of cognitive distortion to ensure that optimal effort and motivation were provided during the neuropsychological assessment. If the effort was subpar, an explanation for this must be sought before further interpretation of the neuropsychological test data is undertaken. Assuming that the patient provided optimal cognitive

effort, it is then necessary to establish in some way a preinjury cognitive baseline. As was discussed in [Chapter 6](#), with the adult patient this can be determined with reading tests such as the *Wechsler Test of Adult Reading*.<sup>23</sup> Moreover, it is useful for the neuropsychiatric examiner to evaluate the preinjury work product of the patient. For instance, determining what level of professional or employment endeavors the patient functioned within prior to injury is very important, and this provides substantial data for the examiner to use in the overall neuropsychiatric assessment. As noted in [Chapter 6](#), if possible, one can get ACT or SAT scores of adult patients produced while they were undergoing secondary education. In some instances, brain trauma patients have been intellectually tested previously. In most instances, the examiner will not have available preinjury neuropsychological data. However, many times, by the time the neuropsychiatric examination is performed, the patient will have been tested neuropsychologically within a rehabilitation treatment program or on an outpatient basis by a neuropsychologist consulting to a psychiatrist or neurologist. These data should be secured, as they are quite useful and enable the neuropsychiatric examiner to determine interval cognitive changes. One expects, within the normal progression of brain trauma healing, that neuropsychological test scores improve rapidly in the first 6 to 12 months postinjury and then plateau. On the other hand, if there is substantial deterioration of neuropsychological performance within the year following brain injury, and assuming the patient provided optimal neuropsychological effort, then other substantial causes of impact upon neuropsychological performance, such as the development of depression, should be explored.

Once the examiner has determined that cognitive effort was optimal during testing and after the examiner has further established the preinjury cognitive status of the patient, then specific focus upon neuropsychological domains should be undertaken. The examiner should review specifically the neuropsychological test data measuring attention, memory, language, visuospatial abilities, sensorimotor function, executive function, and intellectual functioning. The findings from these examinations are then collated into the entire database of the neuropsychiatric examination.

With regard to the analysis of child neurocognitive data, establishing the preinjury cognitive state in the very young child is problematic. However, as noted in [Chapter 6](#), the *Wechsler Individual Achievement Test-II* may be used to establish reading ability in children ages 4 and up.<sup>24</sup> Other efforts at establishing preinjury cognitive ability may include interviewing preschool teachers, reviewing drawings and creative endeavors produced by the child prior to the brain injury, and, of course, taking a history from parents regarding the child's cognitive skills prior to injury (see [Chapter 3](#)). Following establishment of the preinjury cognitive ability, the various neurocognitive testing domains of the child should be reviewed. These include attention, memory, language, visuospatial ability, sensorimotor function, executive function, and intellectual functioning. The findings from these data should be collated into the database established by history, mental status examination, neurological examination, brain imaging, and behavioral evaluation.

## **Behavioral Measures**

As one approaches the behavioral assessment, it is a good time to review the entire neuropsychiatric database for comorbid or premorbid conditions that may play a role in the behavioral difficulties expressed by the patient. In particular, is there preinjury evidence of prior brain injury, learning disability, or substance abuse? Are current expressed behaviors such as impulsivity, hyperactivity, and risk taking a result of the traumatic brain injury, or are they merely premorbid characteristics?<sup>25-27</sup> When these parameters have been established, the examiner should then attempt to categorize the behavioral difficulties expressed by the patient into neuropsychiatric or psychiatric syndromes (see [Chapter 2](#)). It is important to delineate the various syndromes and comorbidities discretely, as differentiation is important for treatment planning and selection. A careful review of the neuropsychiatric history, mental status examination, and ancillary psychological testing should be of assistance to the examiner for separating the patient's psychopathology into discrete entities for further treatment analysis.

When assessing the behavioral problems of the child following brain injury, an approach similar to that of the adult can be undertaken. However, the historical database should be expanded to include teachers, parents, pediatricians, and other caregivers who may provide valuable insights into behavioral changes, if any, observed in the child following traumatic brain injury. While the face-to-face mental examination and ancillary psychological testing may be very important to the overall analysis, gathering the observations of others in the child's life cannot be overstated in terms of their importance to the neuropsychiatric analysis.

### **Impact of the Brain Injury upon Caregivers**

If the purpose of the neuropsychiatric examination is to provide data for treatment planning, the family and other caregivers of the injured patient cannot be overlooked. Quality treatment cannot take place effectively if caregivers are too stressed to assist the patient. Kay and Cavallo<sup>28</sup> have outlined five important impacts of brain injury upon the family system:

1. Traumatic brain injury inevitably causes profound changes in every family system.
2. These changes dramatically influence the functional recovery of the person with brain injury.
3. The impact of the brain injury continues over the life cycle of the family, long after the initial adjustment to disability is made.
4. The lives of individual family members may be profoundly affected by a brain injury in another family member.
5. Family assessment and intervention are crucial at all stages of rehabilitation and adjustment after brain injury, even when a pathological response is not present in the patient.

Thus, the examiner should review carefully the family system for elements of stress within it. As has been noted elsewhere in this text, the burden upon families comes greatest from the behavioral, affective, and personality changes seen following brain injury. Cognitive deficits cause an intermediate burden. The physical injuries of the patient are the least burdensome when compared to behavioral and cognitive impairments.<sup>29</sup> Lezak further provided observations on what it is like for family members living with a brain-injured person who has undergone substantial personality change.<sup>30</sup> She described the personality change impacting the family as (1) an impaired capacity for social perceptiveness; (2) a stimulus-bound behavior or concrete thinking; (3) an impaired capacity for control and self-regulation; (4) emotional alterations, including apathy, irritability, and sexual changes; and (5) an inability to profit from experience and a tendency to repeat maladaptive patterns.

When the caregiver is the spouse, the traumatically brain-injured patient can extract an enormous cost from his or her spouse. Life is no longer the same for either spouse. It has been noted that health care professionals often fail to recognize the spouse's need for individual psychotherapy, or even to consider the stress the spouse may be under.<sup>31</sup> It is very important to determine the level of stress within the spouse, particularly if the examiner is also providing treatment over the long term. [Table 8.3](#) summarizes a schema for collecting neuropsychiatric data to determine deficits during a traumatic brain injury neuropsychiatric assessment.

## **ESTABLISHING NEUROPSYCHIATRIC DEFICITS**

The classic formulation following medical examination is an appraisal of four aspects of the neuropsychiatric assessment: symptoms, signs, measurement, and differential diagnosis. It is also wise to be able to support a conclusion that a particular neuropsychiatric deficit is present. The clinical assessment can be further refined using a biopsychosocial model that applies particularly appropriately to traumatic brain injury. The biopsychosocial model integrates clinical data from

**TABLE 8.3**  
**Systematizing Clinical Neuropsychiatric Deficits**

	<b>Cognitive</b>	<b>Behavioral</b>
History:	Are there symptoms of inattention, speech/language dysfunction, memory impairment, disorientation, visuospatial/constructional dysfunction, or sensorimotor or executive dysfunction? Is there a preinjury learning disorder, psychiatric or neurologic illness, or substance abuse disorder?	Are there symptoms of affective/mood changes, aggression/agitation, thought/perception dysfunction, high-risk behaviors/disinhibition, or altered emotional intelligence? Is there a preinjury learning disorder, psychiatric or neurologic illness, or substance abuse disorder?
Mental status examination:	Are there signs of altered consciousness, inattention, speech/language dysfunction, memory impairment, visuospatial/constructional inability, sensorimotor impairment, or executive impairments?	Are there signs of abnormal affective modulation, abnormal thought processing or content, abnormal perceptions, or admissions of suicidal ideations or plans?
Neurological examination:	Are there abnormalities in function of cranial nerves, motor/sensory abilities, tendon reflexes, muscle strength/tone, cerebellar ability, or posture/gait?	Are there abnormalities of neurological function?
Brain imaging:	Are there abnormal CT or MRI images from the acute care setting? What are the structural and functional imaging findings from the neuropsychiatric evaluation? Do they correlate with cognitive deficits (e.g., frontal lobe injury and deficits of working memory, executive function)?	Are there abnormal CT or MRI images from the acute care setting? Do structural or functional image abnormalities from the neuropsychiatric evaluation correlate with behavioral abnormalities (e.g., infraorbital brain injury and orbital frontal disinhibition syndrome or aggression)?
Cognitive measures:	Is there good effort during testing? If so, are there quantitative impairments of attention, speech/language, memory, sensorimotor function, visuospatial/constructional skill, executive functions, or intellectual functions?	
Behavioral measures:	Is there evidence of symptom magnification or malingering? If not, does psychological testing confirm the presence of depression, mania, anxiety, or psychosis? Is there test confirmation of aggression or self-destructive ideas?	
Family/caregiver interviews:	Does the family or caregiver report impairments in the patient's ability to understand, follow directions, pay attention, remain oriented, use language, or to remember, plan, organize, or complete activities of daily living? Is the family/caregiver stressed or depressed by the patient's cognitive impairments?	Does the family or caregiver report behavior problems in the patient such as aggression, anger, depression, euphoria, anxiety, delusions, perceptual distortions, disinhibition, apathy, hypersomnolence, or suicidal ideas or plans? Is the family/caregiver stressed or depressed by the patient's behavior?

three interrelated domains: (1) biological disturbances in brain function; (2) the patient's emotional and psychological reaction to impairments in cognition and behavior, including the presence of denial or acceptance of these deficits; and (3) the disruptions of interpersonal relationships, family interactions, and work capacities.<sup>32</sup> Thus, the classic formulation method for developing a medical diagnosis is enlarged within the neuropsychiatric model for the clinical assessment of traumatic brain injury. Lezak takes a slightly different approach, as she is establishing only one portion of the neuropsychiatric assessment — neuropsychological deficits.<sup>33</sup> She notes that one distinguishing characteristic of neuropsychological assessment is its emphasis on the identification and measurement of psychological deficits, for it is primarily in deficiencies and dysfunctional alterations of cognition, emotionality, and self-direction in management that brain damage is manifested behaviorally. Yet, brain damage always implies behavioral impairment. The neuropsychiatric examiner must remember that, in some patients, the loss of function, or neuropsychiatric deficit, may be subtle. It may become apparent only following complex assessment or under cognitive and behavioral demands requiring judgment or within emotionally charged conditions. In other patients, the direct behavioral or cognitive effects of the impairment may be so slight or ill defined as to be unobservable under ordinary clinical conditions. The patient may report vague, unaccustomed, and unexpected frustrations or uneasiness, while family and friends report puzzlement at the patient's depression, irritability, or anger. The systemization in [Table 8.3](#) is then used to develop the treatment planning strategies for the patient who has sustained a traumatic brain injury.

## NEUROPSYCHIATRIC TREATMENT PLANNING

Neuropsychiatric treatment planning of the traumatically brain-injured patient is not intended to be comprehensive or all-inclusive. For instance, a totally comprehensive plan is outside the scope of neuropsychiatric evaluation and treatment; it would include planning for the treatment of pain associated with brain injury, assisting posttraumatic headaches, treating and rehabilitating cognitive dysfunction, providing treatment for behavioral changes, improving speech and language disorders following brain injury, managing multifactorial problems with drug therapy, and dealing with spasticity, orthopedic impairment, and neuromuscular disorders following brain injury. Neuropsychiatric evaluations and treatment, in general, deal with the behavioral and cognitive changes in the patient and the impact these impairments may have upon the brain-injured patient and his family or caregiver.

Attempting to treat the brain-injured patient in a professional vacuum is not productive for either the doctor or the patient. One major source of assistance for both the physician and the patient is the Brain Injury Association of America. This organization is an advocacy group for persons who have sustained traumatic brain injury, and it is an educational and resource center for patients and physicians alike. It is located in Alexandria, Virginia, and it provides a wealth of information, particularly to patients, at its Web site: <http://www.biausa.org>. It can be recommended to patients that they subscribe to the publication of this organization, *Brain Injury Source*, which is published on a quarterly basis and provides important educational services for patients and families challenged by traumatic brain injury. [Table 8.4](#) summarizes neuropsychiatric treatment planning.

## PHARMACOLOGIC MANAGEMENT OF TRAUMATIC BRAIN INJURY SYMPTOMS

The majority of pharmacologic interventions following traumatic brain injury are implemented during the acute phase of recovery, particularly within the neurosurgical ICU, trauma center, or rehabilitation facility. However, pharmacologic agents may be necessary for longer periods of time or may be added at a later point in the recovery to treat new or persistent difficulties. The potential side effects of these agents must be weighed against the benefits.<sup>34</sup> The examining physician must remember that the brain has been injured and, obviously, using pharmacologic agents with central



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**TABLE 8.4**  
**Neuropsychiatric Treatment Planning after TBI**

Cognitive:	<ol style="list-style-type: none"><li>1. Identify specific cognitive domains (attention, language, memory, visuospatial, sensorimotor, executive, or intellectual) found to be impaired during the neuropsychiatric examination.</li><li>2. Review school records and work products, if possible.</li><li>3. Determine appropriate pharmacologic, cognitive, behavioral, and family interventions.</li></ol>
Behavioral:	<ol style="list-style-type: none"><li>1. Identify specific disorders of mood, thought, or perception found to be impaired during the neuropsychiatric examination.</li><li>2. Identify whether patterns of aggression, anger, disinhibition, impaired emotional intelligence, or self-destruction are present.</li><li>3. Determine appropriate pharmacologic, psychotherapeutic, behavioral, and family interventions.</li></ol>
Family/caregiver:	<ol style="list-style-type: none"><li>1. Identify specifically from family interviews the impact of the patient's TBI upon the family or caregiver system.</li><li>2. Determine if the spouse, parent, or caregiver is in need of pharmacologic, social, or psychotherapeutic assistance.</li><li>3. Provide appropriate intervention to improve the patient's family/caregiver support and to assist the caregiver if stressed or depressed.</li></ol>
The child:	<ol style="list-style-type: none"><li>1. Identify specific cognitive, behavioral, and caregiver impairments from the neuropsychiatric examination.</li><li>2. Review preschool or school performance, if possible.</li><li>3. Provide appropriate pharmacologic, psychotherapeutic, cognitive, behavioral, educational, social, and parental interventions.</li></ol>

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nervous system (CNS) affects may produce deleterious as well as beneficial effects. These concerns are even more significant when medications are used in older adults. That being said, the physician or neuropsychiatric examiner should be aware that very few evidence-based studies have measured the effects of psychotropic agents in traumatically brain-injured patients. Most pharmacologic information in the medical literature is anecdotal or based on small sample sizes. American medicine is constantly stressing the need for Class I evidence (e.g., randomized controlled trials). At the time of this writing, there is limited Class I evidence to assist the physician in choosing pharmacologic agents in an attempt to treat behavioral or cognitive symptoms following traumatic brain injury.

### Antidepressants

Recall in [Chapter 2](#) that depression occurs in approximately 25% of patients following traumatic brain injury, and that the risk of depression remains elevated for decades following head injury.<sup>35,36</sup> Depression is rarely, if ever, immediately present following injury and tends to appear within the first year after injury. Moodiness and irritability give way to a deeper and more pervasive feeling of sadness, discouragement, and despair. The patient is caught in a vicious cycle as the depressed state begins to work against the process of natural recovery. The prolonged disability compounds the depression. It is at this point that a treating physician considers an aggressive course of antidepressant treatment.<sup>37</sup>

In general, the more modern antidepressants developed and released from 1988 (e.g., fluoxetine) to the present time are preferable because they do not have the anticholinergic properties of the earlier tricyclic antidepressants. Thus, the physician does not have to worry about exaggerating cholinergic dysfunction by muscarinic blockade. There is no current pharmacologic evidence that one antidepressant is superior to another in treating the mood changes following traumatic brain injury. The choice of an antidepressant depends predominantly on the desired side effect profile.<sup>38</sup>

A number of trials have reviewed the use of antidepressants in traumatic brain injury, including tricyclic antidepressants and the newer serotonin reuptake inhibitors (SSRIs). However, none of these trials meet Class I evidence standards for psychopharmacologic research.<sup>39–43</sup> It must be

remembered that antidepressants may increase the likelihood of epileptic seizures in patients who have sustained brain injury. In particular, maprotiline and bupropion have been associated with a greater risk of seizures than other antidepressants.<sup>44,45</sup> While it is rare, on some occasions, electroconvulsive therapy (ECT) may be required for severe depression following brain injury that remains unresponsive to pharmacologic management. In the case of depression following acute traumatic brain injury, it can be used effectively.<sup>38,46</sup> Very few studies of ECT in traumatic brain injury have been published, and almost all are single-case studies.<sup>47</sup>

When prescribing antidepressants to a brain trauma patient, drug interactions must be kept in mind. Other psychotropic agents with anticholinergic effects may be additive to those of antidepressants such as the tricyclic compounds and mirtazepine. Moreover, antiepileptic drugs with liver induction properties, such as phenytoin, carbamazepine, and phenobarbital, may decrease the plasma levels of antidepressants below the therapeutic range. On the other hand, fluoxetine may increase the plasma level of phenytoin.

The strategy for using antidepressants in traumatically brain-injured persons generally follows the usual algorithms for choosing medications in other idiopathic mood disorders.<sup>12</sup> Thus, the treater generally should begin an attempt to elevate mood using serotonin reuptake inhibitors as a first-line antidepressant drug because of their low side-effect profile. Traumatic brain injury patients often have an increased susceptibility to antidepressant side effects. Moreover, the serotonin-enhancing properties of SSRIs may have an unpleasant activating effect that will produce agitation or anxiety in the patient. There is evidence that these agents may have a particular tendency to produce a manic episode following traumatic brain injury.<sup>48</sup> Also, it is well known that SSRI antidepressants may produce a robust antidepressant response that is later followed by an apparent relapse. As with the treatment of idiopathic depression, at this point, the practitioner should increase the dosage of the SSRI and thereby recapture, in some instances, therapeutic remission. If the patient does not respond to an SSRI, desipramine and nortriptyline are good second-line choices because of their relatively low anticholinergic profiles.<sup>12</sup> The dosing strategy must be modified following traumatic brain injury. Due to the heightened sensitivity to medication side effects, the medication should be initiated at one-half to one-third of the usual starting dosage. A longer time interval between increases of dosage also is generally recommended. [Table 8.5](#) summarizes antidepressant use following TBI.

In judging response to medications, the treater should remember that those patients with persistent postconcussion syndromes and an associated major depression may demonstrate an improvement in mood state following the use of antidepressants. On the other hand, the physical complaints often expressed by these patients (e.g., headaches) and the cognitive deficits (i.e., attention, mental processing speed, or memory) often do not improve. Thus, the clinician must carefully assess the symptomatic profile of the patient, as a good mood response to antidepressants may be colored or contaminated by a poor cognitive response. At this juncture, if this occurs, the treater may have to consider the various augmentation strategies often necessary when treating

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**TABLE 8.5**  
**Antidepressant Approaches to TBI**

- Following TBI, the risk of depression remains elevated for decades.<sup>35,36</sup>
  - Most depressions begin the first postinjury year.<sup>37</sup>
  - The choice of antidepressant depends predominantly upon the desired side effect profile.<sup>38</sup>
  - Maprotiline and bupropion carry enhanced seizure risk.<sup>44,45</sup>
  - ECT may be required in extreme cases.<sup>38,46</sup>
  - SSRIs can, in some, produce excessive activation, irritability, or mania.<sup>48</sup>
  - Start low and go slow to reduce side effect risk.
  - Mood disorders may respond, whereas cognitive symptoms may not.
-

depressed patients, such as the use of buspirone, lithium salts, or atypical neuroleptics, or the addition of thyroid hormone.<sup>49</sup>

## Antiepileptic Drugs

Antiepileptic drugs (AEDs), prescribed following traumatic brain injury, are generally used for three clinical indications: (1) posttraumatic seizures, (2) behavioral dyscontrol syndromes (see [Chapter 2](#)), and (3) neuropathic pain (i.e., peripheral nerve dysfunction) or central pain disorders (due to central nervous system pain generators).<sup>34</sup> Twenty years ago, it would not have been expected that antiepileptic drugs would be routinely used within psychiatric medical practice. They are now frequently prescribed, and they clearly are broad-spectrum psychotropics.<sup>49</sup>

The primary neuropsychiatric use of AEDs is for behavioral dyscontrol, particularly aggression. If associated with disinhibition, AEDs may represent an appropriate interventional choice.<sup>34</sup> Moreover, the obvious manifestations of post-brain injury mania (see [Chapter 2](#)) may respond to the AEDs, particularly valproic acid and carbamazepine.<sup>12,50</sup> Valproic acid, in particular, is used more and more frequently in psychiatry as a first-line agent in mania.<sup>51</sup> The reader is also referred to an excellent recent review of the use of antiepileptics in traumatic brain injury with particular emphasis upon psychiatric usage.<sup>52</sup>

The mechanism of action of these drugs remains under study. Phenytoin and carbamazepine inactivate voltage-dependent sodium channels. Thereby, they reduce neuronal excitability. This effect is shared by valproate, oxcarbazepine, felbamate, gabapentin, lamotrigine, and topiramate.<sup>53</sup> Benzodiazepine and barbiturate anticonvulsants interact with the GABA<sub>A</sub> receptor. Activation of the GABA<sub>A</sub> receptor results in an inward chloride flux. Felbamate has some interaction with GABA<sub>A</sub> receptors, and valproate, gabapentin, primidone, tiagabine, and topiramate influence chloride influx within the cell by increasing the availability of GABA<sub>A</sub>.

Ethosuximide suppresses the spike waveform of epilepsy of the absence type by inhibiting calcium conductants across low-threshold (T-type) channels. Valproate may also exert an influence at T-type calcium channels.<sup>49</sup> Another mechanism of anticonvulsant activity is glutamate antagonism. Glutamate is an excitatory transmitter, as is aspartate. Glutamate excites neurons at the NMDA receptor, the AMPA receptor, and the kainite receptor. A number of antiepileptic drugs have antiglutamic acid activity; they include primidone, valproate, carbamazepine, oxcarbazepine, felbamate, gabapentin, lamotrigine, and topiramate.<sup>54</sup> Thus, the beneficial actions of antiepileptic drugs seem principally related to inactivation of voltage-dependent sodium channels, inhibiting T-type calcium channels, activating GABA<sub>A</sub> receptors, and antagonizing glutamate at NMDA, AMPA, and kainite receptors. There is some evidence that the effects of AEDs on voltage-gated calcium signals may prevent the fast-developing cellular damage resulting from ischemic and traumatic brain injury<sup>55</sup> (see [Chapter 1](#)).

Regardless of their mode of action, there is a paucity of Class I evidence-based medical studies confirming the beneficial effects of AEDs in traumatic brain injury. Valproate and carbamazepine appear the most often within studies of AEDs in traumatic brain injury. Both have been advocated for the treatment of organic manic syndromes and aggression.<sup>50</sup> Obviously, agitation and aggression are difficult behaviors to manage following traumatic brain injury, and their treatments are myriad.<sup>56</sup> Recent PET scan research reveals that unlike normal subjects, patients with impulsive aggression do not show activation in the left anteromedial orbital cortex in response to a challenge from meta-chlorophenylpiperazine (m-CPP). The anterior cingulate in nonaggressive humans is normally activated by m-CPP. In contrast, patients with impulsive aggression show a deactivation of the anterior cingulate, and those who are aggressive have an activation of the posterior cingulate. It is thought that the decreased activation of inhibitory regions in patients with impulsive aggression in response to a serotonergic stimulus may contribute to their difficulties in modulating aggressive impulses.<sup>65</sup>

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**TABLE 8.6**  
**Use of Antiepileptic Drugs Following TBI**

- These are generally used for: (1) posttraumatic seizures, (2) behavioral dyscontrol syndromes, and (3) neuropathic pain.<sup>34</sup>
  - For psychiatric use, AEDs may assist to manage mania or disinhibition.<sup>34,49</sup>
  - Valproate will not prevent posttraumatic seizures.<sup>58</sup>
  - Carbamazepine and lamotrigine may have specific usefulness in the brain-injured population.<sup>60-62</sup>
  - Secondary effects of brain injury may alter by enzyme induction the pharmacokinetics of valproate and phenytoin in adults and children, respectively.<sup>59,63</sup>
  - Appropriate laboratory monitoring should accompany the use of valproate, phenytoin, carbamazepine, and lamotrigine.
  - Lamotrigine may modify metabolism of multiple other AEDs.
- 

Wroblewski and others have reviewed the effectiveness of valproic acid in aggressive behaviors following brain injury.<sup>57</sup> A recent randomized, double-masked, parallel group clinical trial compared valproate to phenytoin for seizure prevention and neuropsychological effects following traumatic brain injury in 279 adult subjects. These individuals were randomized within 24 h of injury and examined with a battery of neuropsychological measures at 1, 6, and 12 months postinjury. Drug effects were evaluated cross-sectionally at 1, 6, and 12 months and longitudinally by examining differential changes from 1 to 6 months and from 6 to 12 months as a function of protocol-dictated changes in treatment. No significant adverse or beneficial neuropsychological effects of valproate were detected. Thus, it was determined that valproate has a benign neuropsychological side effect profile, making it a cognitively safe AED to be used for controlling established seizures or stabilizing mood. However, based on this study, valproate should not be used for prophylaxis of posttraumatic seizures because it will not prevent such seizures. There was a trend toward more deaths in the valproate groups, and it did not have positive effects on cognition. It did have positive effects upon unstable mood.<sup>58</sup> Another study has noted that brain injury itself may cause a nonspecific enzyme induction in response to head injury, which could have importance when other drugs are used in conjunction with valproate.<sup>59</sup> Carbamazepine and, more recently, oxcarbazepine are frequently used for treatment of aggressive behaviors and mood instability following traumatic brain injury. However, studies are primarily clinical in nature and with poor randomization. Carbamazepine has been used safely within brain injury acute care units<sup>60</sup> and over long-term treatment of 3 years or more.<sup>61</sup> A recent study suggests that lamotrigine may stimulate improvement of patients with impaired consciousness (Rancho Scale I to III).<sup>62</sup>

As previously discussed, there is little evidence-based medical literature on antiepileptic drugs in brain-injured adults, and even less is found for their uses chronically in brain-injured children. Phenytoin used in prepubescent children with severe brain trauma demonstrates markedly altered protein binding and metabolism.<sup>63</sup> Thus, there may be a similar brain injury-induced pharmacokinetic mechanism at work, as noted previously with valproate in adults.<sup>59</sup> AEDs have been used and studied in an open-label fashion in both children and adolescents following mild to severe traumatic brain injury. Antiepileptic drugs were found to be of assistance to children and adolescents who demonstrated staring spells, memory gaps, and temper outbursts.<sup>64</sup> AED usage is summarized in [Table 8.6](#).

When using AEDs, the clinician must be aware of the potential risks associated. Carbamazepine has been reported rarely to induce bone marrow suppression and to be hepatotoxic. Complete blood counts and liver functions should be monitored appropriately during administration of this compound. With valproate, liver functioning and pancreatic enzyme assay should be performed routinely, initially every 2 weeks, and over the chronic term, every 3 to 6 months. Lamotrigine may produce significant alterations in the blood levels of valproate if administered concurrently, and caution should be exercised when using this agent with other AEDs. The clinician should obtain routine blood level monitoring when using phenytoin, carbamazepine, and valproate. Currently,

there is no clinical indication for measuring blood levels of oxcarbazepine, gabapentin, lamotrigine, or topiramate. In those patients who have both posttraumatic epilepsy and behavioral dyscontrol following traumatic brain injury, routine consultation with a neurologist for management of the posttraumatic epilepsy should be sought.

## Lithium Salts

Cade<sup>66</sup> first discussed the use of lithium salts for restless impulsiveness and ungovernable temper. In fact, he argued more than 50 years ago that the use of lithium salts for these types of behaviors would be preferable to prefrontal leucotomy. While lithium also has been used for post-brain injury mania, many brain injury experts are now more likely to use atypical antipsychotic agents to manage this disorder than to use lithium.<sup>49</sup> Lithium has been used in patients following traumatic brain injury,<sup>67</sup> but these individuals have increased sensitivity to the neurotoxic effects of lithium salts<sup>68</sup>; therefore, the use of lithium following traumatic brain injury is recommended for those whose aggression is related to manic effects or recurrent irritability related to cyclic mood disorders.<sup>50</sup> More recently, lithium has been touted for the treatment of aggressive behaviors in brain-injured patients many years following the original injury.<sup>69</sup>

Obviously, if lithium is used for treatment of aggression following traumatic brain injury, or treatment of manic episodes following traumatic brain injury, appropriate laboratory testing must be performed to maintain the blood level within the therapeutic range, as lithium has a low therapeutic index. Also, it is wise to periodically evaluate serum creatinine, free T-4 and thyroid-stimulating hormone (TSH), and electrolytes. McAllister and Flashman at Dartmouth Medical Center recommend keeping serum levels of lithium in the 0.6 to 1.0 meq/l range when prescribed to a brain-injured patient.<sup>12</sup>

## Neuroleptic Drugs

The psychotic disorders following traumatic brain injury were discussed in some detail in [Chapter 2](#). A recent meta-review of the characteristics of psychotic disorder due to traumatic brain injury suggests that this syndrome can be discriminated routinely from the psychosis of schizophrenia.<sup>70</sup> The psychosis of traumatic brain injury is less likely to demonstrate negative symptoms than are the classical schizophrenic disorders. On MRI and CT (see [Chapter 5](#)), a higher percentage of brain trauma patients than schizophrenic patients demonstrate positive imaging lesions, and the types of findings are qualitatively different. Focal lesions are more commonly found following brain trauma, particularly in the frontal and temporal regions. On the other hand, atrophy and volume loss are the most common MRI and CT finding in schizophrenia patients, whereas focal findings are uncommon, unless the schizophrenic patient has a prior traumatic brain injury. Of more interesting importance from the Fujii and Ahmed study<sup>70</sup> is the long duration following traumatic brain injury before psychosis appears. The mean time of onset was  $4.1 \pm 6.6$  years. However, half of the subjects demonstrated psychotic symptoms before the second year following traumatic brain injury, and 72% reported an onset of psychosis before 4 years. These findings are similar to those of other studies, indicating a wide range of delay in the onset of psychotic symptoms following traumatic brain injury.<sup>71-74</sup>

There has been a significant prejudice against using antipsychotic or neuroleptic medication in neuropsychiatric disorders, including traumatic brain injury. This prejudice is an outcome of the overuse of these agents in residential facilities for the mentally retarded, nursing homes for the elderly, and other facilities for chronically ill persons. Moreover, risk factors for tardive dyskinesia list traumatic brain injury as a prominent clinical issue. Diaphragmatic dyskinesia has been reported and may impair ventilation and increase fatigue.<sup>75,76</sup> Neuroleptics are used following traumatic brain injury primarily for control of agitation, treating psychosis, and in the treatment and control of chronic aggression. Be that as it may, Neppe preferentially uses the typical neuroleptic, perphenazine, in posttraumatic psychosis.<sup>77</sup>

In fact, some emergency medicine and rehabilitation departments argue that the use of multiple neuropharmacologic agents early in the treatment of posttraumatic brain injury agitation may be an effective therapeutic intervention. However, these results are not based on controlled studies.<sup>78</sup> Droperidol, a more potent haloperidol-like neuroleptic, has been used in acutely agitated patients within brain injury units. It can reduce acute agitation in persons with brain injury<sup>79</sup> in a fashion similar to its usefulness in acute agitation in psychotic disorders and emergency room situations.<sup>80</sup> The neuropsychiatrist is unlikely to be treating long-term agitation with neuroleptics, but when the examiner reviews medical records within the acute care facility or rehabilitation unit that treated the patient, it is important to see if there have been prior usages of neuroleptic medications, particularly if the patient now demonstrates an abnormal involuntary movement disorder.

There is some recent anecdotal reporting of the use of risperidone (an atypical neuroleptic) in patients following traumatic brain injury. A beneficial effect has been noted upon both sleep and psychosis following traumatic brain injury by using risperidone. Risperidone has also been used in patients who have refused to eat after traumatic brain injury.<sup>81,82</sup> The question of using atypical neuroleptics in traumatically brain-injured patients is unanswered at this time, however. Theoretically, it would appear that these agents are safer cognitively and carry a lower risk of inducing tardive dyskinesia. However, there is no evidence-based data to support this conclusion when they are used in brain-injured patients, and, currently, any such conclusions are based on clinical anecdotes and theoretical grounds. However, the author uses risperidone, olanzapine, quetiapine, and ziprasidone routinely in patients with appropriate symptomatology as a result of traumatic brain injury. To date, no evidence of tardive dyskinesia has arisen and no significant adverse cognitive effects have been seen. However, these data were not obtained under Class I medical evidence paradigms.

Stanislav has studied typical antipsychotics before, during, and after discontinuation in patients undergoing rehabilitation for traumatic brain injury.<sup>83</sup> Preliminary results support the hypothesis of cholinergic involvement in regulating cognitive processes. His data do reveal that select areas of cognition improve after typical antipsychotic discontinuation in subjects with traumatic brain injury, suggesting a deleterious effect upon cognition during the rehabilitation phase. In current clinical trials, intramuscular olanzapine and ziprasidone have been found superior for treating acute agitation, in contrast to haloperidol and lorazepam. No significant extrapyramidal syndromes have been seen in these early clinical trials. However, none of these patients had sustained a traumatic brain injury.<sup>56</sup> The problem in neuropsychiatry is that the short-term benefits of neuroleptic medication treatment segue into long-term treatment with little evidence-based data to support the usage. For instance, a patient is treated with intramuscular and then oral haloperidol to control agitation and assaultiveness during emergence from a coma. The drug is continued for months after the patient is discharged. Then, the patient appears for evaluation at a rehabilitation facility and is anergic, depressed, and apathetic with fine and gross motor coordination problems and deficits in attention, memory, and emotional control. The neuroleptic drug is withdrawn, and there is immediate improvement. This is the general sequence of events for typical neuroleptics.<sup>49</sup> We know that neuroleptic effects on brain plasticity are unequivocally negative and probably related to ultrastructural and synaptic changes in the caudate nucleus.<sup>84</sup> However, we currently do not understand these effects with the atypical neuroleptic drugs (e.g., clozapine, olanzapine, risperidone, quetiapine, and ziprasidone). Thus, at this time the best advice for the clinician treating behavioral disorders or psychosis with neuroleptics following traumatic brain injury is probably to avoid long-term use of the typical neuroleptics and judiciously apply use of the atypical neuroleptics with routine clinical monitoring. [Table 8.7](#) reviews neuroleptic usage in TBI.

## **Anxiolytic Medications**

Anxiety disorders are frequent following traumatic brain injury, and they have been described with an incidence rate as high as 70% (see [Chapter 2](#)). Moreover, posttraumatic stress disorder (PTSD) is common in adults and even more common in children who sustain severe injuries. It has been

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**TABLE 8.7**  
**Use of Neuroleptics Following TBI**

- There is a prejudice against using neuroleptics in the brain-injured population.<sup>84</sup>
  - Brain injury is a risk factor for tardive dyskinesia.<sup>75,76</sup>
  - If psychosis occurs after brain injury, neuroleptics generally must be prescribed.<sup>77</sup>
  - Withdrawal of typical neuroleptics, if appropriate, may lead to an improvement in cognition or reduce abulia.<sup>83</sup>
  - Atypical neuroleptics may confer less tardive dyskinesia risk than typical compounds, theoretically.
  - Neuroleptics are routinely used to control acute agitation following TBI.<sup>78,79</sup>
  - However, routine use of neuroleptics acutely may segue into long-term treatment with little evidence-based data to support the usage.<sup>49</sup>
- 

recommended that psychopharmacologic agents should not be used to treat anxiety in traumatic brain injury patients until behavioral and psychotherapeutic methods have been tried first.<sup>85</sup> The Dartmouth group<sup>12</sup> finds little confirmation for the use of benzodiazepines in traumatic brain injury. In particular, they have noted that clonazepam may not be particularly useful in the brain-injured population, as it can sedate without affecting the core symptoms of posttraumatic mania and may lead to disinhibition. Fava<sup>86</sup> has underscored that benzodiazepines can reduce agitation and irritability in the elderly and demented populations, but they can also induce behavioral disinhibition, and therefore, one should be careful in using this class of drugs in patients with pathologic aggression, a frequent finding following traumatic brain injury. Gualtieri<sup>49</sup> notes that benzodiazepines are a troublesome class of drugs, and while an occasional patient with traumatic brain injury will do well on one of the benzodiazepines, this is rare. Not only do benzodiazepines disinhibit, like alcohol and the barbiturates, but they also tend to impair memory in this population.<sup>87</sup> There does seem to be a small role for benzodiazepines in patients with traumatic brain injury who have associated cervical injuries or those patients with spasticity. Diazepam may be a particularly good muscle relaxant for these patients, and in some, it assists with the management of posttraumatic headaches with a muscle contraction component.<sup>49</sup> The author has found that benzodiazepines, particularly clonazepam, add to memory impairment in those patients who demonstrate trauma-induced memory decrements following brain injury.

There is some evidence that buspirone, an azaspirodecanedione, not a benzodiazepine, may have some particular usefulness in anxious or aggressive traumatic brain-injured patients. Buspirone seems to exert its primary effect on 5-HT<sub>1A</sub> receptors, and it appears to reduce serotonin turnover by inhibiting the firing rate of serotonin neurons in the dorsal raphe of the brain stem.<sup>88</sup> It also has effects on the norepinephrine and dopamine systems, but those pharmacological properties are poorly understood and do not seem to necessarily correlate with its clinical effects.<sup>89</sup> Neppe<sup>77</sup> suggests initiating dosage at 5 mg three times daily and to build up gradually by 5 mg every 3 days to a target dose of 60 mg daily. If dizziness intervenes, drop the dose by 5 mg daily until the dizziness disappears and then reinstate the dosage or taper the dosage away if the patient is intolerant. The author has found high-dose buspirone to be effective in the reduction of hostility and mild aggression following traumatic brain injury, especially in the elderly. However, no randomized controlled studies have been conducted to evaluate buspirone's effectiveness in traumatic brain injury.

### **Cholinergic Cognitive Enhancers**

Cognitive impairment after traumatic brain injury is correlated with decreased cholinergic markers of neuronal viability<sup>90</sup> (see [Chapter 1](#)). Disruption of the cholinergic system in the hippocampus may account for disturbances in sensory gating and divided attention following traumatic brain injury.<sup>91</sup> It is well known that cholinergic blockade can produce an anticholinergic syndrome or delirium.<sup>92</sup> On the other hand, medications that enhance acetylcholine neurotransmission may be

depressants and may aggravate Parkinsonian symptoms. However, one of the earliest uses of cholinergic agents was by Luria et al., the esteemed Russian physician and neuropsychologist who used the acetylcholinesterase inhibitor galanthamine to enhance cognitive function in some of his patients who had sustained brain injury.<sup>93</sup> Recently, the psychiatric cholinomimetic armamentarium has added donepezil, galantamine, and rivastigmine. Tacrine has little clinical usefulness today because of substantial risk of hepatotoxicity.

As with most psychotropic agents, the cholinergic enhancers are used, at this time, off-label in the treatment of cognitive and memory dysfunction following traumatic brain injury. They are Food and Drug Administration (FDA) approved for Alzheimer's disease syndromes only. However, they are becoming widely used in clinical practice, and the author has used them successfully in many patients demonstrating memory dysfunction and other cognitive dysfunctions following traumatic brain injury. A recent open-label study examined donepezil in the treatment of cognitive dysfunction associated with traumatic brain injury.<sup>94</sup> Fifty-three ambulatory psychiatric patients who were receiving care for psychiatric sequelae of brain injury were studied. Cognitive assessment was made using the *Wechsler Adult Intelligence Scale-Revised* (WAIS-R) and the *Hooper Visual Organization Test*. While this study reported significant improvement using this cholinesterase inhibitor, to date, no randomized controlled studies of cholinesterase inhibitors in traumatic brain injury patients have been published.

## Psychostimulants

Psychostimulants are the oldest psychotropic drugs in continuous use. They are the only drugs clearly demonstrated to have positive effects on cognitive performance in patients with known cognitive impairment and also in normal persons.<sup>49</sup> They are perhaps the best-studied class of drugs among brain injury patients, and there is a significant body of medical literature to suggest they may augment the neurological recovery process.<sup>37</sup> The psychostimulants exert their therapeutic effect primarily by augmenting the release of catecholamines into the synaptic space, but serotonergic actions occur at higher concentrations. Dextroamphetamine is the prototype agent in this class; methylphenidate is a more potent releaser of dopamine from storage vesicles, and while pemoline has a longer half-life than these two agents, it is seldom used because of the need to rapidly clear medication effects in the event of an adverse response.<sup>95</sup> The dosages of stimulants in traumatic brain injury generally are methylphenidate, 5 to 15 mg daily; dextroamphetamine, 15 to 20 mg daily; and pemoline, 18.75 to 75 mg daily. In general, these drugs appear to be a reasonable treatment choice for certain types of mood, behavior, and cognitive symptoms following brain injury; however, larger-scale controlled studies are needed to adequately assess the clinical usefulness.<sup>96</sup> Psychostimulants appear to improve symptoms of inattention, distractibility, disorganization, hyperactivity, disinhibition, impulsiveness, and emotional lability in many patients with traumatic brain injury. This has been established in short-term and longer-term studies.<sup>97,98</sup>

Two recent double-blind, placebo-controlled trials have examined methylphenidate for treatment of attentional and other cognitive dysfunction following traumatic brain injury.<sup>99,100</sup> The first study indicated that subacute administration of methylphenidate after moderately severe head injury appeared to enhance the rate, but not the ultimate level of recovery as measured by the *Disability Rating Scale* and other tests of vigilance. Follow-up evaluations were conducted at 30 and 90 days after baseline was determined. Methylphenidate was administered twice daily at a dose of 0.30 mg/kg, and placebo was administered according to the same schedule in identical pill form. The second study focused more intensely on attentional function in individuals with traumatic brain injury, and the results suggested that methylphenidate may be a useful treatment in traumatic brain injury, but it is primarily useful for symptoms that can be attributed to slowed mental processing. However, the second study<sup>100</sup> did not specifically measure the rate of improvement, as was performed in the first study.<sup>99</sup> Further uses of methylphenidate for cognitive and behavioral dysfunction after traumatic brain injury can be found in the publication by Glenn.<sup>101</sup> With regard to children



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**TABLE 8.8**  
**Psychostimulant Use Following TBI**

- These may augment the neurological recovery process.<sup>37</sup>
  - Psychostimulants improve symptoms of inattention, distractibility, disorganization, hyperactivity, disinhibition, impulsiveness, and emotional lability in properly selected TBI patients.<sup>97,98</sup>
  - Methylphenidate is useful for slowed mental processing, and it may enhance the rate of recovery.<sup>99,100</sup>
  - Stimulants may be combined with amantadine, levodopa, and antidepressants.<sup>49</sup>
- 

and the use of methylphenidate following brain injury, unlike the extensive pediatric literature for ADD, there are no significant controlled studies of methylphenidate use in pediatric traumatic brain injury. There has been a recent review of the topic, however.<sup>102</sup> Amphetamine has received less attention than methylphenidate in childhood traumatic brain injury, and no significant controlled studies are available. A recent chart review suggests that amphetamine treatment enhances the recovery and functional status of patients recovering from brain injury.<sup>103</sup> The medical literature contains no significant evaluations of pemoline in traumatic brain injury. [Table 8.8](#) reviews psychostimulant usage following TBI.

The psychostimulants, especially methylphenidate and amphetamine, in low to moderate doses are safe and effective for a variety of clinical symptoms after traumatic brain injury. They may enhance recovery by attenuating negative processes such as kindling or enhancing new learning. The stimulant effect may be long-lived despite the short duration of drug action. Stimulation effect on norepinephrine is probably central to both methylphenidate and amphetamine, although dopamine and serotonin may also be involved. Long-term treatment with psychostimulants seems to be perfectly safe but it is not always necessary. Treatment usually continues for a few weeks or a couple of years. Stimulants are better for patients who have had milder injuries or those who have made good recovery from severe injuries. Stimulants may be successfully combined with amantadine, L-dopa, and antidepressants.<sup>49</sup>

### **Dopamine Agonists and Amantadine**

At present, the most frequently used dopamine agonists in traumatically brain-injured patients are L-dopa, bromocriptine, and pergolide. Others have recently been released for use in the treatment of idiopathic Parkinsonism, but their usefulness in traumatic brain injury has not been significantly reported. Several clinical reports suggest that dopamine agonists improve the state of patients with various types of brain injuries. It has been claimed that L-dopa promotes coma recovery or may advance the recovery process in traumatic brain-injured patients who have reached a plateau in their recovery.<sup>104,105</sup> Amantadine, on the other hand, is not a true dopamine agonist. It appears to enhance presynaptic dopamine release and inhibit dopamine reuptake,<sup>106,107</sup> but its most likely action is as an NMDA (N-methyl-D-aspartate) antagonist with actions on dopamine that are indirect.

Bromocriptine seems to offer dopamine stimulation as well as dopamine-blocking effects, depending on the dosage used. At a low dose, it inhibits dopamine transmission, and at high doses, dopamine action is increased. Bromocriptine may help with memory, motivation, and executive functions of the brain. It is most likely to be used in the acute or subacute period following traumatic brain injury and much less likely to be prescribed for long-term effects. Therefore, the clinician providing a neuropsychiatric assessment is unlikely to see patients who are receiving bromocriptine. Its strength is that it has few side effects other than occasional nausea, and it does not require any blood monitoring and does not preclude participation in any specific type of employment.<sup>34</sup> Bromocriptine is thought to enhance functional recovery in the acute phase following traumatic brain injury.<sup>108</sup> It also seems to be of benefit to those patients who have dysexecutive syndromes following frontal brain injury.<sup>109</sup> It seems to have significant enhancing and improving effects upon those

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**TABLE 8.9**  
**Dopamine Agonists and Amantadine Usage in TBI**

- Dopamine agonists may enhance functional recovery and improve dysexecutive syndromes.<sup>108–110</sup>
  - These agonists may advance neurobehavioral recovery if it plateaus.<sup>104,105</sup>
  - Amantadine seems to improve tremors, visual inattention, concentration, and speed of mental operations.<sup>34,112</sup>
  - Amantadine may be combined with levodopa to decrease impulsivity–perseveration and to improve executive function.<sup>113,114</sup>
- 

with frontal lobe injury.<sup>110</sup> It has also been found useful in children with autonomic dysfunction after severe traumatic brain injury.<sup>111</sup>

Amantadine can be used to reduce the severity of tremors. Its benefits in traumatic brain injury include improved visual attention, increased speed of information processing, and improved concentration. It does not require blood monitoring, and it does not interfere with occupational activities.<sup>34</sup> It is sometimes used for problems of fatigue and depression, and it has found usefulness in multiple sclerosis for that reason.<sup>112</sup> Table 8.9 describes uses of dopamine agonists and amantadine.

Amantadine has been combined with L-dopa/carbidopa for those with persistent frontal dysfunction chronically present following brain injury. It has been noted to decrease impulsivity and perseveration in these persons as well as improve executive function.<sup>113</sup> Moreover, it has been noted to potentiate motor recovery many years following a traumatic brain injury. Its effects seem to be primarily upon motor speed rather than motor ability.<sup>114</sup> While the neuropsychiatrist or other physician examiner performing a neuropsychiatric examination is not likely to see this, amantadine use shows some benefit in improving the minimally conscious state in vegetative patients following traumatic brain injury.<sup>115</sup>

### Other Categories of Drugs

The Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation has reviewed the types of medications most likely to be prescribed by rehabilitation physicians to brain-injured patients. The five most frequently prescribed drugs by expert physicians were carbamazepine, tricyclic antidepressants, trazadone, amantadine, and beta-blockers. Physicians nonexpert in the treatment of traumatic brain injury most often report prescribing carbamazepine, beta-blockers, haloperidol, tricyclic antidepressants, and benzodiazepines. Trazadone and amantadine are significantly more likely to be chosen by experts than nonexperts.<sup>116</sup> However, in this study, the majority of physiatrists surveyed did not formally measure agitation. Adrenergic beta-blockers have selective affinity for various adrenergic receptors. Atenolol, for example, is cardioselective and primarily blocks B<sub>1</sub> receptors. Propranolol and nadolol have mixed selectivity and block both B<sub>1</sub> and B<sub>2</sub> receptors. Propranolol crosses the blood–brain barrier quite readily, whereas nadolol crosses slowly, if at all. Pindolol appears to cause less bradycardia and hypotension than the other beta-blockers. However, it has behavioral toxicity and may cause excitement and agitation; therefore, it is not recommended for use in traumatically brain-injured patients. Propranolol has a very short half-life unless the long-acting form is used. Propranolol has been touted for a number of years as an effective treatment for chronic aggression, particularly following traumatic brain injury.<sup>117</sup>

The guidelines for using high-dose propranolol for the control of chronic aggression following traumatic brain injury are fairly simple and straightforward.<sup>117</sup> A thorough medical evaluation should be completed, and patients should be excluded who are contraindicated for using beta-blockers, particularly those with asthma, chronic obstructive pulmonary disease, insulin-dependent diabetes, congestive heart failure, persistent angina, peripheral vascular disease, or hyperthyroidism. Propranolol and other beta-blockers should never be rapidly discontinued, particularly if the patient has hypertension. The patient is given a test dose of 20 mg daily, and then the

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**TABLE 8.10**  
**Propranolol for Aggression Following TBI<sup>117,118</sup>**

- Complete a thorough medical evaluation and avoid contraindications for beta-blocker use.
  - Give trial dose of 20 mg/day.
  - If no untoward effects, increase dosage 20 mg every 3 days.
  - When 60 mg/day dosage is reached, increase dosage 60 mg every 3 days.
  - Keep resting pulse rate above 50/min and resting systolic blood pressure above 90 mmHg. Monitor for dizziness, ataxia, or wheezing.
  - Target dose is 12 mg/kg of body weight. Dosages above 800 mg daily are not usually required.
  - Give at least an 8-week trial at appropriate dosage. Depression is a rare side effect.
- 

dosage may be increased by 20 mg every 3 days. After a 60-mg dosage schedule is obtained, the dosage can be increased by 60 mg daily every 3 days. The medication may be increased unless the pulse rate drops below 50 beats per minute or systolic blood pressure falls to less than 90 mmHg. If the patient has dizziness, ataxia, or wheezing, the medication should not be administered. The target dose is 12 mg/kg of body weight, or until aggressive behavior is under control. Dosages greater than 800 mg daily are usually not required. The patient should be maintained at the highest dose of propranolol for at least 8 weeks before concluding that the patient is unresponsive to medication. Some patients may respond rapidly, whereas others may not. Concurrent use of medication requires caution, particularly for those who require antipsychotic or anticonvulsive medication. The major side effects of this protocol are reduced blood pressure and pulse rate. Beta-adrenergic receptors are fully inhibited after doses of approximately 300 to 400 mg daily. Further decreases in cardiovascular markers do not occur even when dosages are increased to much higher levels. Depression is rarely seen, even at high doses.<sup>50</sup> A propranolol dosing regimen is found in [Table 8.10](#).

Since Yudofsky et al.<sup>117</sup> suggested the use of propranolol for chronic aggressive states, other reviews have assessed the effectiveness of beta-blockers in the treatment of chronic aggression. Beta-blockers appear to be effective in decreasing the frequency and intensity of aggressive outbursts associated with a wide variety of conditions, including dementias, attention deficit disorders, personality disorders, Korsakoff's psychosis, posttraumatic stress disorder, schizophrenia, profound mental retardation, autism, and brain injury.<sup>118</sup> A recent review of the literature of the past 20 years found that the most likely drugs used for posttraumatic agitation and aggression are carbamazepine, antidepressants, and propranolol, in that order.<sup>119</sup>

A number of other recent reviews of novel approaches to treating behavioral difficulties following traumatic brain injury include the use of naltrexone. This drug has been used in children with autism and developmentally disordered children who engage in self-injurious behavior.<sup>49</sup> However, it was reported that doses of 50 to 100 mg daily were successful in reversing cognitive impairment following postconcussion disorder.<sup>120</sup> Naltrexone was reported recently to improve accelerated recovery in an 18-year-old male with prolonged coma following traumatic brain injury.<sup>121</sup> This is an interesting response since there is very recent evidence that cyclo-oxygenase-2 (Cox-2) activation contributes to motor and cognitive dysfunction following diffuse traumatic brain injury in rats. If this also occurs in humans, one might consider use of the recently introduced Cox-2 anti-inflammatory agents.<sup>122</sup> Since patients seem willing to try anything when they are desperate for relief, the physician treating traumatic brain injury must remember that oftentimes patients will seek herbal or over-the-counter remedies. There is a recent report of St. John's wort and *Ginkgo biloba* being added to fluoxetine and buspirone thus causing hypomania.<sup>123</sup> Thus, clinicians treating traumatic brain-injured patients with pharmaceutical agents should ask if non-prescription medicines or herbal remedies are being used at the time the physician prescribes psychotropic agents.

What does the future hold for the pharmacologic treatment of traumatic brain injury? It seems exciting for the most part. Even though successful treatment strategies remain elusive, the recent understanding of mechanisms of cellular response to trauma and injury is enlarging our knowledge of effective treatments for brain-injured patients. Pharmacological strategies under investigation are targeting sites involved in the secondary neurochemical cascade that contributes to overall poor outcome following the primary brain injury. These treatments include ion channel antagonists, calcium channel antagonists, growth factors, antioxidants, stem cells, apoptosis inhibitors, and inhibitors of other signal modulators. This research should be enlarged; as can be seen from the data presented in this chapter, the likelihood of improving outcomes with a single approach is extremely small. Collaborative efforts are needed to investigate the logical sequence or combination of treatments that will ultimately lead to improved cognitive and behavioral outcomes in the brain-injured population.<sup>124</sup>

### **INDIVIDUAL PSYCHOTHERAPY FOLLOWING TRAUMATIC BRAIN INJURY**

Interestingly, only a small portion of people with mild traumatic brain injury will seek or accept referral for counseling immediately after the injury. Those with moderate or severe injuries are generally too impaired to enter into individual psychotherapy until late in their recovery phases. However, it is the persistence of symptoms that typically escalates the patient into severe emotional distress and leads to the family, physician, or patient referral for an assessment of cognitive and emotional status.<sup>12</sup> It has generally been only in the decade of the 1990s that psychiatrists as individual practitioners, or psychiatry as a profession, demonstrated interest in working with people who had experienced significant brain trauma. Prior training in psychiatry and consultation from colleagues in neurology and neurosurgery produce the belief that improvement, following the initial 6 months or so of recovery, could not be brought about by any type of therapeutic intervention. The myth was that after about 6 months, what you see is what you will have.<sup>125</sup> Traumatic brain injury often causes a “loss of sense of self,” and loneliness is quite disturbing to most adults following a traumatic brain injury.<sup>126,127</sup> Therapy must develop within the relationship until the therapist and the patient come to share an understanding of the nature of the problem as it is experienced by the patient. Therapists working with traumatic brain injury patients need symbols, concepts, or analogies that adequately represent, for both the therapist and the patient, what it is like to have a damaged brain.<sup>128</sup>

Pollack<sup>125</sup> recommends that therapy should begin, assuming the patient is competent enough, with reassurance to the patient that a brain injury is causing the behavioral or emotional disturbances, and that it is not due to a neurotic or psychotic process, of course assuming the patient has neither. The patient’s complaints should be carefully heard by the therapist, and then the injury and its causation of emotional difficulties should be explained to the patient in nontechnical language. Positive language should be used with persons following brain injury, and while the therapist cannot predict the outcome, it is clear that in most instances patients will improve, even though positive changes will be slow in coming. As patients are labile over time with their injuries, therapists must be very flexible when working with the traumatically brain-injured.

As would be expected with any skilled therapist, he or she must acknowledge transference and counter-transference issues. Earlier interpersonal experiences may be magnified by the brain injury, and patients may misinterpret transference issues as a result of their brain injury. Moreover, the therapist must be in touch with personal counter-transference issues that may be raised. These forces can lead a therapist to underestimate the severity of the patient’s disabilities or overestimate the degree of recovery that can be reasonably expected following treatment. As discussed previously in this text, denial may be a core issue. Whereas in traditional psychotherapy denial may be due to intrinsic psychological processes or transference issues, in most instances, denial following substantial traumatic brain injury may in fact be due to organic factors or interference with right hemisphere control of prosody and the emotional aspects of language.

Other substantial issues may be raised during individual psychotherapy with the traumatically brain-injured patient. These include guilt and shame from either the impact of the injury upon the patient's family or, in many instances, someone having been killed or maimed during the accident. Other issues are the stigmatization and marginalization that occur by society and citizens toward the disabled. This, in turn, may produce substantial loneliness within the brain-injured person.

If the therapist chooses to treat a brain-injured person, it is wise to understand the operation of anxiety after brain injury, as it can interfere with these individuals in attaining and maintaining interpersonal relationships.<sup>129</sup> Moreover, the therapist should be aware that individual psychotherapy may be neither indicated nor effective unless patients have a realistic perception of their present skill level following injury.<sup>130</sup> The frequency of therapy may play a role in recovery as well. Weekly feedback may result in a greater reduction in maladaptive behaviors than more interrupted therapy.<sup>131</sup> In some instances, the therapist may wish to refer more intact individuals for group therapy. It is considered advantageous to conduct anger management in a group format if the patient is capable of receiving feedback.<sup>132</sup> Group therapy may also assist with retraining individuals in social confidence.<sup>133</sup>

Some therapists experienced in individual psychotherapy, who choose to work with brain-injured patients, follow a rehabilitation model of psychotherapy. This model focuses on the patient's present interpersonal, social, and cognitive strengths and weaknesses. Goals are established mutually by the therapist and patient, and the strengths and weaknesses of the patient are identified and goals are then established. The therapist explores and reinforces methods of using the patient's strengths in problem situations and provides ways for discontinuing or modifying problem behaviors. Of course, as with any therapy, the strength of the doctor-patient relationship is the most powerful modulator of therapy effectiveness, even in the rehabilitation model.<sup>134</sup>

Therapy with children, of course, requires a different set of skills than therapy with adults. Thus, therapists treating brain-injured children should possess fundamental child psychiatric or psychological therapy skills. Studies of children following traumatic brain injury have demonstrated significantly lower levels of self-esteem and adaptive behavior, and higher levels of loneliness, maladaptive behavior, and aggressive or antisocial behavior.<sup>135</sup> A Finnish study<sup>136</sup> followed children with severe brain injuries at preschool age between January 1959 and December 1969. Final evaluations were then performed in adulthood. They noted that the long-term outcome after severe brain injury in preschoolers is worse than expected, and this may provide substantial challenges to child therapists. In adulthood, only 23% of injured children were able to work full-time as adults. Twenty-six percent were employed at sheltered workshops, and 36% lived independently but at their parents' home. Fifteen percent needed continued physical and psychotherapeutic support into adulthood. They noted that the sense of identity was the best indicator of final outcome for brain-injured preschoolers. The study results recommended that the necessity for the child to develop a firm identity was essential for good social outcome.<sup>136</sup>

A cohort study from the University of Washington in Seattle noted that brain-injured children tend to plateau within a year and that they do not achieve parity with peers. They noted that the moderate to severely brain-injured child shows a strong improvement rate during the first year, but a negligible rate of change in the following 2 years postinjury in most domains. The greatest slowing of recovery occurred in performance IQ, adaptive problem solving, memory, and motor skills.<sup>137</sup>

Unfortunately for therapists, behavioral interventions to brain-injured children have not been empirically validated.<sup>138</sup> There also is limited information and research that pertains to internalizing features of maladaptive behavior in children following brain injury. There are published behavioral interventions to address externalizing behaviors, and these have received the greatest focus.<sup>139</sup> However, there is scientific evidence that early psychosocial assessment and interventions aimed at increasing a child's coping may attenuate the emotional consequences of pediatric brain injury. Thus, while there is no empiric evidence for focusing upon internalizing behaviors, studies note that where therapists focus upon mood and anxiety disorders following pediatric traumatic brain injury, children, in turn, perform better.<sup>140</sup>

## FAMILY INTERVENTIONS AND THERAPY

Kay and Cavallo have conceptualized the impact of traumatic brain injury on the family in three broad phases: (1) the acute phase, with primary issues of survival, medical stabilization, and minimization of permanent damage; (2) in the subacute phase, family roles are reorganized and the goal is the restoration of physical and cognitive function; and (3) during the reintegration phase, the brain-injured person attempts to return as much as possible to a level of maximum engagement in the family and productivity in the community, while the family settles into a pattern in hopes of achieving equilibrium and resuming the family life cycle, even though with an altered identity.<sup>141</sup> Regardless of what stage the family finds itself following a brain injury in one of their loved ones, significant care needs, stress, and burdens are experienced. All focus of treatment and therapy directed toward the family is generally directed toward establishing life cycle trajectories for the family as well as assisting in the reintegration of the injured party within their family and larger social system.<sup>142</sup> There has been some argument in the last few years that there is a divergence of perspective between brain-injured persons and their families. A recent study found that brain-injured patients and family members have similar perspectives on reintegration of the patient into the community.<sup>143</sup> Moreover, recent research demonstrates that family needs and support systems change over time. Unfortunately, with many brain-injured persons, caregiver life quality diminishes over time as well.<sup>144</sup>

Recent research has helped define the changes in stress and burdens in families seeking therapy following traumatic brain injury. One recent Australian study noted that counseling extended for 24 months improved reported levels of anger. However, they noted in the duration of their study that the anger actually increased back to its original level at 24 months follow-up. Marital adjustment also worsened in the latter half of this study, with couples reporting a similar level of marital adjustment to what they had experienced prior to counseling. As a result of this study, it has been recommended that marital couples receive longer-term counseling to address their specific needs and 2 years may be insufficient in light of the huge transitions required within the family.<sup>145</sup> Another Australian study noted that the best measures and indicators of family functioning were the severity of the injury in the patient, the patient's residual neurobehavioral function, and the adequacy of social support available for caregivers.<sup>146</sup> In the U.S., a recent Columbia University Study noted that frequent telephone calls to caregivers provided a relatively low-cost, nonintensive intervention that offered substantial benefit to the families.<sup>147</sup> A Michigan study reconfirmed what has been known in the area of traumatic brain injury for a number of years. That is, the level of social support to the family showed a direct and linear relationship to the family's ability to function. In fact, it was the strongest predictor of family functioning, and it is recommended that rehabilitation and other treating professionals should stress the importance to caregivers and families that adequate social supports be obtained for them.<sup>148</sup> When individual members within a family are assessed for adverse effects following traumatic brain injury in their loved one, it is noted that caregivers who provide direct care for a person with brain injury in the home experience a larger number of role changes than those other family members who do not provide direct care to the person.<sup>149</sup> When levels of depression are assessed within the family unit following brain injury, the primary stressor significantly related to caregiver depression is the number of adverse effects on family members as a result of the brain injury.<sup>150</sup> After a brain injury, there is a significant impact on family structure, which, in turn, produces role changes. The spouse, usually the wife, since more men suffer brain injuries than women, bears the greatest burden when her partner sustains a brain injury. When a child is injured, special burdens and pressures accrue to the parents. The mother is the usual primary caregiver, and this often creates a tension within the marital relationship. If the marriage is slightly unstable, a major crack may appear in the veneer of the marriage and produce a complete breakdown or rupturing of the marital relationship. Children may suddenly find that they have lost the nurturance of a parent who was previously competent. Thus, the child suddenly has a father who can no longer read or use a computer at the level he

did prior to his injury. Young children may not understand this, whereas older children may separate themselves and produce a distance between themselves and the injured parent. This may surface as depression, anger, or school problems.

With regard to siblings, they may develop anger toward the injured child. Parents may redirect their energies toward the injured child, which, in turn, removes levels of nurturance from the siblings. The siblings may then react adversely toward the injured youngster. In families who have strong networks with grandparents, aunts and uncles, and other significant others, even the extended family network may suffer substantial role changes as a result of the injury. These dynamic changes have been expressed by some authors as stages of family adjustment not unlike the information found in the grief literature, that is, initial shock, denial, and unrealistic expectations, then an acknowledgment of permanent deficits, which produces emotional turmoil. At this point, the family may establish a bargaining ritual until they eventually begin to mourn and work through their grief. Lastly, if the grief work and adjustment proceed according to plan, there will come acceptance and restructuring of the family unit.<sup>151</sup>

Therapy for spouses is particularly problematic. When the injured party is a husband and primary breadwinner, the wife may sense an immediate threat to her well-being as a result. This may go unrecognized, and in fact, many health care professionals fail to recognize the spouse's need for individual psychotherapy independent of the injured spouse.<sup>152</sup> The grief experienced by spouses can be quite profound, basically because they often suffer the death of a relationship, but continue to be married. The author routinely hears spouses say, "This is not the man I married." The changes can be subtle but profound enough to fracture the marital bond. This may produce a significant impact upon the caregiving spouse. If the marital relationship fractures, the injured spouse is aggressive or impulsive and unkind, then the caregiving spouse may lack the emotional will to provide essential caregiving needs. Moreover, dealing with the injured spouse may produce significant emotional and physical exhaustion by assuming all of the adult roles within the family that were previously shared. This results in a sense of isolation. To overcome these obstacles, the therapist must provide a comforting relationship by listening and validating the concerns of the spouse, translating the concerns to language the spouse can understand, and educate the spouse about the difficulty of grieving when the brain-injured spouse remains in the home. Further focuses of therapy should include reducing isolation and expanding networking so that the surviving spouse can develop the needed social supports.

When it is a child who has sustained the traumatic brain injury, the effects can also be very substantial. Six months after an initial postinjury assessment, parents of children with traumatic brain injuries report more family disagreements than do parents of children who have sustained orthopedic injuries. Moreover, with brain-injured children, there is a trend for parents to report higher levels of psychological distress than when the child is orthopedically injured. Surprisingly, few studies of traumatic brain injury in children have examined the family impact of childhood brain injury or the relevance of the family environment to the child's long-term recovery.<sup>153</sup> The impact upon the family following pediatric traumatic brain injury is major. It can include psychological, financial, role change, and relationship risks. When treating the family, even more so than when treating the adult, the child is only one component of a comprehensive recovery program and family intervention is a must.<sup>154</sup>

## **COGNITIVE REHABILITATION**

The neuropsychiatric examiner or psychiatric treater generally will not be concerned about cognitive rehabilitation. That occurs primarily in the rehabilitation phase following traumatic brain injury, and those skills are usually practiced by physiatrists, speech pathologists, occupational therapists, physical therapists, and social workers. However, there is a recent controversy over the cost-effectiveness of cognitive rehabilitation for brain injury. There is also a controversy about whether cognitive rehabilitation meets the evidence-based standards now required for

medical treatments. A comprehensive single-center, parallel-group, randomized trial conducted from January 1992 through February 1997 by the U.S. Military Medical Referral Center examined the efficacy of inpatient cognitive rehabilitation for patients with traumatic brain injury.<sup>155</sup> At 1-year follow-up, there was no significant difference between patients who had received the intensive hospital-based cognitive rehabilitation program and those who had a more limited home rehabilitation program. The primary measure of success was the ability to return the patient to employment. There also were no significant differences in cognitive, behavioral, or quality-of-life measures. The only significant difference found between the two study groups was within a *post hoc* subset analysis of patients who were unconscious for more than 1 h. The patients treated within the hospital had a greater rate of return to duty (80 vs. 58%,  $p = .05$ ). An editorial in the *Journal of the American Medical Association* (J.A.M.A.) after this study emphasized the importance of conducting randomized controlled trials in traumatic brain injury research. The authors argued that the Salazar et al. study<sup>155</sup> will serve as a stimulus for building a solid base of evidence for clinicians to use when making decisions regarding the role of cognitive rehabilitation for patients with brain injury.<sup>156</sup>

Another study is a contraposition to the J.A.M.A. study. A less powerful method of study, using meta-analysis, was performed at the J.F.K.–Johnson Rehabilitation Institute in New Jersey. The authors of this study, based entirely on a literature review of 655 published articles, concluded that support exists for the effectiveness of several forms of cognitive rehabilitation for persons with stroke and traumatic brain injury. In their opinion, sufficient data existed that specific recommendations could be made for remediation of attention, memory, functional communication, and executive functioning after traumatic brain injury.<sup>157</sup> Unfortunately, this study was not randomized and does not meet Class I levels of research proof within an evidence-based medical analysis.

The question of the importance of long-term, hospital-based cognitive rehabilitation, and its effectiveness for rehabilitation of the brain-injured patient, remains in evolution. The psychiatric treater or physician performing neuropsychiatric brain injury evaluations should keep in mind that there is a question as to the effectiveness of cognitive rehabilitation programs following traumatic brain injury. Unfortunately, there is no ethical way to provide a conclusive research study of the question. It is probably not ethical to withhold treatment from brain-injured persons while others are placed into cognitive remediation. The data of Salazar and others force us to rethink the issue. It may be that appropriate and intensive interventions of the brain-injured patient within the family structure may be as effective as hospital-based programs, with the exception of the most severely injured persons who have been unconscious for prolonged periods of time. Therefore, the psychiatrist, family physician, internist, or other person treating the aftereffects of traumatic brain injury should become an important part of the team providing services to those who may rehabilitate at home following traumatic brain injury.

## CLINICAL NEUROBEHAVIORAL ANALYSIS OF CASE DATA

### CASE 1: TRAUMATIC BRAIN INJURY DISCOVERED 2<sup>1</sup>/<sub>2</sub> YEARS LATE

#### Introduction

M.E., a 41-year-old male, was neuropsychiatrically examined 2<sup>1</sup>/<sub>2</sub> years after a motor vehicle accident. Since his injury, he had proceeded poorly within physical therapy. He had been unable to return to work. He was being treated for a nonunion of the distal left leg, and he had had numerous surgical interventions. His case manager asked for a neuropsychiatric examination, as he was not proceeding in the expected fashion during orthopedic rehabilitation; he was having substantial difficulty with healing, and it was questioned whether he had sustained a brain injury that had been overlooked.



## **History of the Accident**

He was traveling to attend a conference with his supervisor on a four-lane circle highway. On the way to the appointment, he was the victim of a severe motor vehicle accident. He was operating the vehicle and was restrained by a seat belt. His vehicle was struck head on, and the driver of the vehicle that struck his died in the accident. M.E. sustained crush injuries to both legs. He was hospitalized at a teaching hospital and underwent open reduction on both legs on several occasions. He sustained a laceration of the left forehead that required closure with sutures, bilateral superior and inferior rami pelvic fractures, bilateral femoral fractures, a right navicular fracture, first and second metatarsal fractures, a right scapular fracture, a left adrenal contusion, a lung contusion, and a liver contusion. He underwent a CT scan of the head, which was reported to be within normal limits.

He underwent a second CT examination of the head 21 months following his injury, and it again was interpreted as being unremarkable. Neurological evaluation 27 months postaccident revealed a history of “memory trouble” since the accident. He reported impairment of recent memory, and he also reported verbal perseveration. He had no history of posttraumatic epilepsy. At the neurological examination, he used a rolling walker. His gait was antalgic, favoring the left leg. Tinel’s sign was positive over the right median nerve at the wrist and negative on the left. Cranial nerve testing was normal. Muscle examination revealed no focal weakness. No abnormal movements were present. Reflexes were symmetric and normal. The Babinski sign was absent bilaterally. He demonstrated normal amplitude and velocity in the hands and feet. His coordination was intact to finger–nose testing and heel–shin examination. There was no drift of the outstretched arms. Sensory examination revealed reduction in pinprick sensation in the left arm and left leg. Vibratory sense was normal in the feet.

## **History from the Patient**

M.E. related that his last memory prior to impact was leaving his home that morning. He had no memory of impact. He could not remember how he was extracted from his vehicle. He did not remember being transported by ambulance. He did not remember treatment in the hospital emergency department that received him. He had no memory of his initial orthopedic surgery. His first memory came to him at least 2 days posthospitalization, when he could recall being asked in his room, “Who is the president of the U.S.?”

A few months after his injury, he developed a depressive syndrome. He was treated with sertraline 100 mg daily, which he was taking at the time of his neuropsychiatric examination. His wife reported that it had been helpful to his mood and motivation, but he did not report that it had been helpful to him. In his review of neuropsychiatric symptoms, he subjectively admitted to depression, sadness, nervousness, and feelings of panic. He admitted asking his wife questions over and over, as he could not remember having asked her. He complained of inability to remember what he read. He would forget what his children and wife told him. He became easily agitated with perceived stress. He reported word-finding difficulty. His thinking was muddled and confused.

## **Past Medical and Psychiatric History**

While he did not know his birth weight or his developmental milestones, he reported no significant childhood illnesses and no birth injury. He was not born prematurely. He was able to sit still in school, and he could keep his mind on tasks and learned to read without difficulty. He had no prior injuries in motor vehicle accidents. He had never been in a coma and had never had a head or brain injury. He had no surgeries prior to the subject accident. At the time of his neuropsychiatric examination, he was prescribed atenolol, rofecoxib, prazosin, sertraline, sulfa, hydrocodone, and amitriptyline. He used Benadryl™ to assist with sleep. He was not using herbs or natural products. He did not use tobacco products, alcoholic substances, or illicit drugs.

With regard to his psychiatric history, he had never been hospitalized for psychiatric, drug abuse, alcohol, or mental problems. He had never been prescribed previously any form of antide-

pressant, tranquilizer, or other psychiatric medicine. He had no history of receiving counseling or psychotherapy. He had never intentionally overdosed himself on drugs or medicine, and he had never made an attempt to take his life. He had never intentionally cut, burned, or disfigured himself.

### **Family and Social History**

His father died of heart disease at age 79, and a brother died of bone cancer at age 32. A sister had recently been found with a thyroid nodule. His mother was alive at age 79, and she was afflicted with hypertension and had had a cerebral stroke. There was no family history of mental illness or depression. There was no alcoholism or drug addiction in the family. There was no family history of suicides, homicides, violence toward others, child abuse, or spouse abuse. No one in the family had epilepsy, other neurological diseases, or Alzheimer's disease.

He was born outside the U.S. and immigrated to this country as a youngster. Both parents were present in the home when he was young, and his father did not abuse his mother. He had no history of sexual or physical abuse, and he had no history of violence toward others. He had never been in legal or personal difficulty due to his sexual behavior. He attended high school but dropped out prior to graduation. He was employed at the time of his injury, he maintained a quality marriage, and he and his wife had five children. His legal history was negative. He was employed as a midlevel executive at the time of his injury. He had no history of military service.

### **Review of Systems and Activities of Daily Living**

He had gained at least 40 lb since his accident. He had significant evidence of excessive sweating intermittently. He would change his shirt two or three times daily because of sweating. He reported substantial fatigue and headaches several times weekly. Due to his leg fractures, he reported bilateral leg swelling. He was deconditioned and had shortness of breath on walking. He reported gastroesophageal reflux, poor balance, and pain and weakness in his legs. He reported difficulty walking, sitting, bending, and lifting, and an inability to ambulate without a four-point walker.

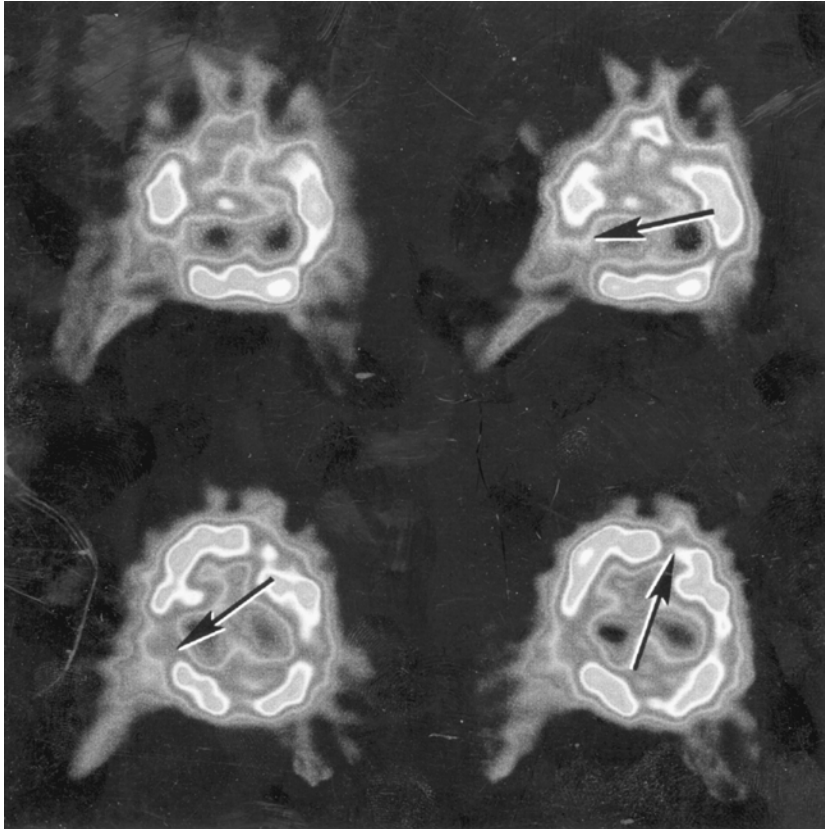
He had not returned to employment since his injury, and he lived with his wife and five children. He had variable times of going to bed and arising. He was unable to drive a vehicle. He reported no hobbies, but he was able to read a newspaper, write, and watch television. He ate outside the home socially every 2 or 3 months. He needed help dressing his lower body, but he was able to bathe himself.

### **Mental Status Examination**

He was a pleasant, cooperative man, but he ambulated slowly using a four-point walker while wearing an external knee brace around the left knee. He was oriented to person, place, and time. He was very cooperative and polite. His mood was subjectively saddened and depressed, and objectively, his affective range was constricted and anxious, and he demonstrated excessive perspiration in an air-conditioned room. He was able to make appropriate eye contact. He denied suicidal ideas or plans. He was logical and coherent in his thinking and without evidence of loose associations or circumstantiality. No delusions or hallucinations were present. Articulatory agility was reasonably good, and the melodic line and phrase length were both normal. There was no evidence of paraphasia. He did complain of word-finding difficulty, but it was not detectable on face-to-face examination.

### **Neurological Examination**

His body mass index was 31. Blood pressure was 142/94 in the left arm, sitting position. There were no bruits about the head or neck. He ambulated with a four-point rolling walker and demonstrated an antalgic gait with an articulating external knee brace supporting the left leg. He followed commands appropriately.



**FIGURE 8.1** SPECT scan of brain using technetium 99-Neurolite. The arrows indicate a left frontal hypoperfusion defect as well as bilateral posterior temporal-occipital hypoperfusion defects. The right temporal-occipital defect is much larger than the left.

Cranial nerve function was intact. Strength was symmetric and there was no drift of outstretched arms. Cerebellar examination revealed no evidence of dystaxia, dysmetria, or dysdiadochokinesia. No nystagmus was present. Deep tendon reflexes were symmetric and normal in amplitude. Sensory examination revealed a nondermatomal loss of pinprick sensation over the left arm and leg. The Romberg sign was not present. The Babinski sign was absent bilaterally. No clonus was present.

### Brain Imaging

Single photon emission computed tomography (SPECT) scanning of the brain was obtained using 32.9 mCi of technetium 99-labeled Neurolite intravenously. Scanning was performed using appropriate external standards. [Figure 8.1](#) reveals axial views that demonstrate a significant left frontal hypoperfusion defect and bilateral posterior temporal-occipital hypoperfusion defects. These defects were confirmed on sagittal and coronal imaging.

### Standardized Mental Assessment

On the Test of Memory Malingering (TOMM), he produced a perfect score of 50 on Trial 2. On the Victoria Symptom Validity Test (VSVT), he provided a score of 43 of a total 48, with a score of 19 of 24 on the difficult items section. On the Letter Memory Test, he produced a percentage score of 96. All three scores were within normal limits and indicated good cognitive effort.

On the Minnesota Multiphasic Personality Inventory (MMPI), he produced the following validity profile:

	Cannot Say	VRIN	TRIN	F Scale	Fb	Fp	L Scale	K Scale	S	F-K Index
Raw	0	7	8	8	8	0	4	12	26	-4
T-score		57	57	61	75	41	52	43	51	

Results of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) validity measures were consistent with a valid psychological profile. The elevation of the Fb scale did not preclude interpretation of the supplementary, content, and personality psychopathology scales.

#### *Measures Providing Estimates of Preinjury Function*

The Wechsler Test of Adult Reading (WTAR) was administered to M.E. He produced the following demographic predicted Wechsler Adult Intelligence Scale-III (WAIS-III) and Wechsler Memory Scale-III (WMS-III) indices:

#### **WTAR Demographic Predicted WAIS-III and WMS-III Indices**

	Standard Score	Percentile	Classification
WAIS-III VIQ	96	39	Average
WAIS-III PIQ	95	37	Average
WAIS-III FSIQ	95	37	Average
WAIS-III VCI	95	37	Average
WAIS-III POI	97	42	Average
WAIS-III WMI	98	45	Average
WAIS-III PSI	93	32	Average
WMS-III Immediate Memory Index	89	23	Below average
WMS-III General Memory Index	91	27	Average
WMS-III Working Memory Index	99	47	Average

#### *Attention and Concentration*

The Ruff 2 and 7 Selective Attention Test was administered to assess visual attention, whereas the Brief Test of Attention (BTA) was administered to determine auditory attention. Conners' Continuous Performance Test-II was administered to determine whether lapses of attention were present. The following scores were determined:

#### **Ruff 2 and 7 Selective Attention Test**

Measure	Sum of T-Scores	T-Score	Percentile	Classification
Total speed	99	51	55	Average
Total accuracy	71	35	7	Mildly impaired

#### **Brief Test of Attention**

	Raw Score	Percentile	Interpretation
Form N (numbers)	7		
Form L (letters)	9		
<b>BTA total score</b>	<b>16</b>	<b>25-74</b>	<b>Average</b>

## Conners' Continuous Performance Test-II

Measure	T-Score	Percentile	Guideline
# Omissions	42	79	Average
# Commissions	46	65	Average
Hit RT	52	41	Average
Hit RT standard error	65	5	Markedly atypical
Variability	60	16	Mildly atypical
Detectability (d')	54	33	Average
Response style (B)	47	62	Average
Perseverations	46	63	Average
Hit RT block change	66	5	Markedly atypical
Hit SE block change	64	8	Mildly atypical
Hit RT ISI change	61	14	Mildly atypical
Hit SE ISI change	67	4	Markedly atypical

*Note:* RT = reaction time; SE = standard error; ISI = inter-stimulus interval.

### *Language and Language-Related Skills*

M.E. was administered the Boston Naming Test and the Controlled Oral Word Association Test (COWA). He produced the following results:

#### **Boston Naming Test**

Raw score	52
T-score	34
Classification	Mildly to moderately impaired
Percentile	6

#### **Controlled Oral Word Association Test**

Raw score	31
T-score	41
Percentile	19
Classification	Low normal

### *Visuospatial Skills*

To test this modality, M.E. was administered the Judgment of Line Orientation Test. He produced the following mean corrected score:

#### **Judgment of Line Orientation**

Raw score	18
Age-corrected raw score	18
Percentile	4
Classification	Moderately defective

### *Memory*

Memory was measured by administering the WMS-III. The following memory scores and classifications were obtained:

#### **Wechsler Memory Scale-III**

	Scale Score Sums	Index Scores	Percentiles	Classification
Auditory immediate	18	94	34	Average

### Wechsler Memory Scale-III (Continued)

	Scale Score Sums	Index Scores	Percentiles	Classification
Visual immediate	14	81	10	Low average
Immediate memory	32	86	18	Low average
Auditory delayed	18	94	34	Average
Visual delayed	15	84	14	Low average
Auditory recognition delayed	8	90	25	Average
General memory	41	87	19	Low average
Working memory	19	96	39	Average

#### Sensory Perceptual Skills

He was administered the Reitan-Kløve Sensory Perceptual Examination. This test instrument consists of three measures: bilateral simultaneous sensory stimulation, fingertip writing, and tactile finger recognition. His T-scores are noted next:

#### Sensory Perceptual Examination

	Raw Score	T-Score	Classification	Percentile
Total right errors	4	42	Below average	23
Total left errors	2	47	Average	39
Sensory perceptual total	6	44	Below average	27

#### Motor and Visual Motor Skills

To make these measurements, M.E. was administered the Grooved Pegboard Test, Grip Strength Test, and Finger Tapping Test. He produced the following T-scores and classifications:

#### Grooved Pegboard Test

	Raw Score	T-Score	Classification	Percentile
Dominant right hand	82	40	Below average	16
Nondominant left hand	78	42	Below average	23

#### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant right hand strength	34	35	Mildly impaired	7
Nondominant left hand strength	41	41	Below average	19

#### Finger Tapping Test

	Mean Raw Score	T-Score	Classification	Percentile
Dominant right hand	46	39	Mildly impaired	14
Nondominant left hand	42	42	Below average	23

#### Executive Functions

For these measurements, M.E. was administered the Wisconsin Card Sorting Test (WCST) and Trail-Making Tests A and B. He produced the following scores and classifications:

## Wisconsin Card Sorting Test

	Raw Score	Standard Score	T-Score	Percentile	Classification
Total errors	49	81	37	10	Mildly impaired
Completed categories	3				
Perseverative responses	28	83	39	13	Mildly impaired

## Trail-Making A

Raw Score	T-Score	Classification	Percentile
53	32	Mildly to moderately impaired	4

## Trail-Making B

Raw Score	T-Score	Classification	Percentile
64	55	Above average	70

## Test Intelligence

This was assessed using the WAIS-III. Scaled scores were converted to standard scores. M.E. produced the following scaled scores and standard scores:

## Wechsler Adult Intelligence Scale-III

Subtests	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Picture Completion		5		5		
Vocabulary	10		10			
Digit Symbol-Coding		4				4
Similarities	9		9			
Block Design		5		5		
Arithmetic	7				7	
Matrix Reasoning		7		7		
Digit Span	9				9	
Information	10		10			
Picture Arrangement		7				
Comprehension	9					
Symbol Search		8				8
Letter-Number Sequencing	13				13	
<b>Sum of scaled scores</b>	<b>54</b>	<b>28</b>	<b>29</b>	<b>17</b>	<b>29</b>	<b>12</b>

## Deviation IQs

	Standard Score	Classification	Percentile	Range
Verbal IQ	93	Average	32	88-98
Performance IQ	73	Borderline	4	68-81
Full-scale IQ	83	Low average	13	79-87

## WAIS-III Index Scores

	Standard Score	Classification	Percentile	Range
Verbal comprehension	98	Average	45	92-104

### WAIS-III Index Scores (Continued)

	Standard Score	Classification	Percentile	Range
Perceptual organization	74	Borderline	4	69–83
Working memory	97	Average	42	90–104
Processing speed	79	Borderline	8	73–90

#### Psychopathology

For this measure, M.E. was administered the MMPI-2. The validity indicators and the clinical scaled scores represented as T-scores are presented next.

#### Minnesota Multiphasic Personality Inventory-2

Scale	VRIN	TRIN	F	Fb	Fp	L	K	S
T-score	57	57	61	75	41	52	43	51

Scale	1	2	3	4	5	6	7	8	9	0
T-score	92	93	96	77	44	64	85	70	38	77

#### Records Reviewed

The records reviewed included those of a university hospital trauma center, home health agency, rehabilitation hospital, community hospital, sports medicine clinic, neurologist, internal medicine physician, and orthopedic surgeon. The ambulance report was not available for review.

#### Diagnoses

The diagnostic considerations are dementia due to traumatic brain injury and neurocognitive disorder due to traumatic brain injury. His overall neurobehavioral analysis qualifies him for the diagnosis of dementia. As noted previously in this text, the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) diagnostic system works poorly in traumatic brain injury cases, and some diagnostic latitude by the examiner is required. Specifically, his diagnostic category is dementia due to head trauma 294.1x (ICD-9-CM). In association, a secondary most likely diagnosis is cognitive disorder not otherwise specified 294.9 (ICD-9-CM).

#### Neurobehavioral Analysis

This case is included so that the reader may understand that, sometimes, there will be incomplete data available for review. For instance, the original ambulance report was not available, and therefore, the GCS score at the scene was not known. At the university hospital, the initial GCS score was 14. The reader may find it helpful at this point to follow the schema noted in [Table 8.1](#) for a data analysis. For instance, the records of the university hospital noted severe bodily trauma. This, of course, should raise suspicion that this gentleman could have sustained a brain injury. As noted in [Chapter 1](#), multiple orthopedic fractures and contusions of internal bodily organs often are associated with traumatic brain injury. Within the university hospital records, it was difficult, if not impossible, to determine his mental state. The patient had such severe body trauma, and so many medical complications, that his mental state was either unobtainable at times or influenced by the administration of multiple medications required for his management. Therefore, an accurate assessment of his mental state could not be made during the acute hospital phase. This, in turn, led to overlooking his mental disorder in the succeeding years during which he was treated until the case manager nurse noted alterations in behavior and memory.



The rehabilitation hospital records primarily focused upon his severe orthopedic injuries, the nonunion of the tibial fracture, and his inability to ambulate. Little data existed in that record to determine his mental state. While it was noteworthy that he was progressing poorly during the rehabilitation phase, there was insufficient data for the author to determine his cognitive state during that admission. In fact, at discharge, his Rancho Scale was reported to be VIII.

A substantial neurological record existed on this gentleman following discharge from rehabilitation. However, it was 1½ years after his original injury before he first came to the attention of the neurologist. The neurologist reviewed a prior CT scan of the head, which was interpreted as normal. However, the neurologist did not order an MRI or functional imaging studies at the time of examination. The neurologist did point out that the history was consistent with a memory disorder and that neuropsychological testing should be undertaken. However, due to the significant difficulties with repeated orthopedic surgeries, M.E.'s complete neuropsychiatric examination was not undertaken until 2½ years postaccident.

If the reader will refer to M.E.'s history, there is raised a suspicion of both cognitive and mood changes following his injury. He admits to depression, sadness, nervousness, and panic feelings. It could be argued that these are the result of problems coping with his accident. However, the complaints of memory disorder, perseveration, agitation, and confusion point to cognitive impairments. His past medical history and past psychiatric history reveal no evidence of medical problems prior to his accident that would account for his present mental state, nor is there evidence of a preinjury psychiatric disorder. There is no family history of genetic mental disorders that possibly could play a role in his mental state. While this gentleman was not highly educated, he was a very functional person and operating as a middle manager. This, in and of itself, would suggest at least average intelligence prior to injury. As is sometimes the case, academic records are reviewed to assist in determination of preinjury cognitive capacity, but they were not available in this instance. His social functioning was within normal limits and he was a devoted family man and father.

His face-to-face mental status examination revealed evidence of autonomic instability. Not only was he anxious, but also he excessively perspired, which was consistent with the history he reported to the author. His mood was subjectively reported to be saddened and depressed. Objectively, he demonstrated a constricted affective range. Constricted affective range could point to either mood disturbance or right hemisphere damage affecting language prosody. He showed no evidence of brain stem dysfunction, as the articulatory agility was good and the melodic line and phrase length were normal. Normal melodic line and phrase length reduce the likelihood of dysprosodic language dysfunction.

Upon review of the SPECT scan, it is obvious that there are multiple perfusion deficits. The most troublesome perfusion deficits lie in the posterior temporal-occipital areas, and they are present bilaterally, as noted in [Figure 8.1](#). Moreover, there also is a left frontal defect present. Recall from [Chapter 6](#) that diffuse functional imaging deficits are more likely to result in cognitive dysfunction than are single focal perfusion deficits. Since the temporal-occipital perfusion deficits are so extensive, the examiner of cognition at this point should make a mental note to review for impairment of visuospatial ability. The neurological examination does not provide much assistance to the examiner of cognition. While obvious orthopedic impairments are present, there is no evidence of significant focal neurological deficits to assist with the neurobehavioral analysis.

At this point in the analysis, after the examiner has reviewed the history, mental status examination, and neurological examination data and coordinated that with the brain imaging data, focus should be placed upon the results of the neuropsychological assessment. First, the examiner should review cognitive effort testing. In this case, the examiner can see that this gentleman produced valid results on both the Test of Memory Malinger and the VSVT. This allows the examiner, using the psychologist's data, to conclude that at the time of examination, the patient provided optimal cognitive effort. Therefore, if cognitive deficits are determined to be present, they are probably validly reported deficits. The examiner should also review for evidence of psychological distortion. The reader may want to review [Chapter 6](#) at this point regarding problems of psycho-

logical distortion. In the case of this patient, the MMPI-2 validity measures were consistent with a valid psychological profile. Therefore, the neuropsychiatric examiner felt confident that the patient provided good cognitive effort and valid psychological effort.

In order to determine injury, it is necessary to have some baseline. The measures providing estimates of preinjury function will assist the examiner in this regard. If one reviews the data from the WTAR, it should be clear that the data predict average ability on the Wechsler-III IQ testing and the Wechsler-III memory testing. The predicted standard scores predicting IQ all lie within the 90s and range from the 37th to the 42nd percentile. The predicted standard scores for memory range from 89 to 99, with respective percentile ranges from a low of 23 to a high of 47. Only the WMS-III immediate memory index is predicted to be below average. All other memory and IQ functions are predicted to be within the average range. These scores then become the internal standards for reviewing other neuropsychological data on this patient.

It is noted that he is mildly impaired on visual attention based upon the Ruff 2 and 7 Selective Attention Test. On the Conners' Continuous Performance Test-II, his performance was characterized by inconsistent response speeds and his rate of response increasingly became slower and less consistent as the test progressed. This would indicate fatigue factors associated with maintaining vigilance. However, with regard to auditory attention as measured by the Brief Test of Attention (BTA), he is within the average range. Therefore, his attentional deficits seem primarily to be in the visual domains, and this is consistent with the SPECT scan findings of perfusion deficits in the temporal-occipital regions bilaterally.

On the Boston Naming Test, he is showing an anomia; this is consistent with the left frontal damage noted on the SPECT scan. As noted in [Chapters 2 and 6](#), anomias are frequent following traumatic brain injury. His word generator seems intact and fluent, as the Controlled Oral Word Association Test, while in the low normal range, probably remains sufficiently intact.

The reader was cautioned above to pay particular attention to visuospatial skills because of the temporal-occipital findings on SPECT scan. On the Judgment of Line Orientation, the patient is moderately defective and only at the fourth percentile in performance. These findings are consistent with the findings on the SPECT scan. On the other hand, memory is probably mildly reduced relative to preinjury predictions as noted on the WMS-III. However, it is not severely impaired. This in turn is a good prognostic factor for treatment planning for the patient. Patients who cannot remember are very difficult to assist with psychotherapy or social improvements.

As might be expected, the patient's sensory perceptual skills are slightly below average in some areas. This is not inconsistent with a person who has sustained severe orthopedic trauma. Executive function, measured by the Wisconsin Card Sorting Test, is mildly impaired and not consistent with what is predicted by preinjury factors. On measures of test intelligence, the reader should note the WAIS-III. The deviation IQs show a 20-point verbal-performance difference with a substantial reduction in performance IQ. If the reader reviews the performance scaled scores, it is noteworthy that the patient's performance is subpar on Digit Symbol-Coding, Picture Completion, and Block Design. These are visuospatially loaded tests, and this finding is not unexpected and probably accounts in part for the drop in performance IQ. Moreover, the index scores indicate impaired perceptual organization and mental processing speed skills, respectively. Reduced mental speed will adversely affect performance IQ ([Chapter 6](#)).

With regard to psychopathology, the pattern on the MMPI-2 indicates substantial elevations on scales 1, 2, and 3, and a high elevation on scale 7. The very low score on scale 9 is noted. The elevations on scales 1, 2, and 3 are consistent with a "somatic" profile on the MMPI. This is not an unusual finding in a patient who has sustained severe bodily trauma and CNS injury. The elevation on scale 7 is consistent with a substantial anxiety component, and that was observable during the mental status examination. The very low score on scale 9 is consistent with loss of energy and fatigue. The mild elevation on scale 4 is consistent with irritability and impulsiveness. The mildly elevated scale 8 is a frequent finding in traumatic brain injury and often represents confusion or addled thinking (see [Chapters 2 and 6](#)).

## Treatment Planning

It was recommended to the case manager that a substantial neuropsychiatric treatment plan be established. The patient clearly has a mood impairment. Therefore, a revision of the sertraline treatment was required. He was switched to venlafaxine, which subsequently improved his mood. He was prescribed donepezil, which improved cognitive performance. A psychotherapeutic program was established to provide not only individual psychotherapy, but also family therapy for his burdened wife. The children were included where appropriate due to the substantial stress they experienced as a result of their ill father.

## CASE 2: AIRBORNE EJECTION FROM VEHICLE

### Introduction

This case is a more classical traumatic brain injury case as a result of a motor vehicle accident. It represents many of the issues most likely to confront physicians performing neuropsychiatric examinations following traumatic brain injury. The patient developed a secondary hypoxic injury due to hypovolemia or respiratory arrest. The patient was 20 years old at the time of this neuropsychiatric evaluation.

### History of the Accident

S.K. was operating an automobile when he apparently struck a tree. This caused his vehicle to spin, and in turn he was ejected from the rear hatch of the vehicle. He was airborne until his body struck a guardrail on the opposite side of the highway. When attended by the ambulance squad, his GCS score was 3 (E = 1, V = 1, M = 1). His Revised Trauma Score (RTS) was 8 at the scene. When the ambulance attendants examined him, he was unresponsive. He was intubated at the accident site and transported to a university medical center. GCS during transport was 3T (intubated) and 3T, respectively, at 1-h intervals. His clinical condition deteriorated, and the trauma scores taken at the same time as the GCS were 4 and 4, respectively.

At reception in the emergency ward, a CT scan of the head revealed a large right frontal epidural hematoma with a small amount of intraventricular hemorrhage. There was noted to be a fracture of the greater wing of the right sphenoid bone with pneumocephalus present. He was admitted to the hospital. A second CT was obtained the third hospital day following neurosurgical evacuation of the epidural hematoma. This CT scan revealed a new and acute infarction in the right middle cerebral artery-anterior cerebral artery watershed vascular territory. The radiologist interpreted this to be consistent with infarction following respiratory arrest or hypovolemia. A repeat CT scan of the head on the eighth day of hospitalization revealed a small right subdural hematoma and a persistent intraventricular hemorrhage in association with a left temporal epidural hematoma. The watershed infarct remained. A CT scan of the head was obtained on the eleventh day of hospitalization. The subdural hematoma was now resolved, but bifrontal subdural hygromas were present (see [Chapter 5](#)). On the 18th hospital day, a CT scan was obtained and compared to prior studies. The watershed infarct in the right frontal region remained. Bilateral subdural hygromas were again noted, but there was an interval decrease in the intraventricular hemorrhage noted on prior examinations. A low density was now present within the splenium of the corpus callosum, which was interpreted to represent diffuse axonal injury.

During the acute hospital phase, S.K. was noted to have a closed-head injury, a right frontal epidural hematoma, right anterior and posterior lateral maxillary sinus fractures, a complex fracture of the right zygoma, bilateral nasal fractures, right anterior-posterior table frontal sinus fractures, and a nondisplaced lateral mass fracture in the right C1 vertebra. He incidentally developed *Staphylococcus aureus* pneumonia.

He spent 23 days in the acute care hospital and was discharged subsequently to a rehabilitation brain injury unit. He required a further 25 days at that facility. At admission to the brain injury

unit, he was at Rancho level III-IV. He was nonverbal, agitated, and unable to follow commands for motor examination or sensory testing. He was hyperreflexic over the left upper extremity. He had an upgoing toe on the left and a downgoing toe on the right. Further examination and review of the laboratory data established the presence of a cardiac contusion and pulmonary contusion.

### **History from the Patient**

At the time of his neuropsychiatric examination, he was 15 months postinjury. Due to behavioral abnormalities, he was under treatment with trazadone, paroxetine, valproate, and captopril. He could not give the examiner any factual information about the accident. He had no memory of being ejected from the vehicle. He had no memory of being attended at the accident scene or of transport to the emergency department. He had no memory whatsoever during his 23-day acute hospitalization. His first memories came to him during the posthospital brain injury rehabilitation treatment. He reported sadness, loss of memory, confusion, irritability, excessive anger, arguing, flashbacks of the accident, geographic disorientation, difficulty falling asleep, and inability to maintain sleep. He often awakened during the night and paced the floor before returning to sleep. He noticed a significant dysfunction in his left arm, and he was unable to coordinate that arm for fine motor control. While walking, he reported he felt as if he were walking at an angle. He denied a specific suicidal plan or suicidal ideation, but he complained of having thoughts of cutting off his left fifth finger or even cutting off his hand.

### **Past Medical and Psychiatric History**

He was a 6-lb, 7-oz baby who was not born prematurely. He sustained no birth injury and his developmental tasks and milestones were normal. He reported no difficulty sitting in school or keeping his mind on tasks, and he learned to read without difficulty. Teachers did not complain that he was hyperactive.

Following his brain trauma, he became hypertensive and was thus treated with captopril. He had no prior history of head injury or bone fractures. He had no prior history of surgery. He had never been treated for any form of psychiatric disturbance prior to the injury. However, following his injury, he was referred to a mental health center near his home and was treated both with the psychiatric medicines noted above and psychotherapy. He had no history of intentional overdose of drugs or medications, and he had never made an attempt to take his life. He had never intentionally cut, burned, or disfigured himself.

### **Family and Social History**

Both parents were alive. His mother had been treated for depression and possibly anorexia nervosa. He was an only child, and his parents divorced when he was 2 years of age; he was subsequently raised by his mother. There was evidence of physical abuse by his father toward his mother. His family reported that following the accident, he was excessively angry and developed rages and would then tear up property. There were no guns in his home, and he denied any statements to his family to harm himself. While in school, his grades were erratic, but he did graduate from high school. He was attending a community college at the time of his injury, and he never had been married or had children. His legal history was negative. He had held no significant public employment.

### **Review of Systems and Activities of Daily Living**

He reported shaking in his left arm, chronic headaches, and blurred vision in the right eye. He reported a reduction in visual acuity in the right eye. He complained of chronic upper airway and sinus drainage producing a wet cough. His remaining review of systems was negative. With regard to activities of daily living, he was unable to attend college following the accident, was not working,

and resided with his mother. He was able to eat outside the home socially, and he could use the telephone. He had difficulty dressing as a result of left arm dysfunction, but he could bathe himself. He had no significant social relationships.

### Mental Status Examination

He was a pleasant, cooperative young man. He was extremely hypergarrulous, and he was unable to properly monitor language. He demonstrated significant logorrhea. During escort throughout the examination offices, he demonstrated geographical difficulty and he could not find his way back to an examination area after he had left. He was judged to be a marginal historian.

Articulatory agility was impaired, and the melodic line was not normal. He demonstrated word-finding difficulty, but no paraphasias were noted. His mood was pseudoeuphoric, and he was impulsive and socially forward. He denied suicidal ideas or plans. No delusions or hallucinations were present.

### Neurological Examination

He was confused and poorly oriented to date. He also demonstrated probable impaired orientation to time. His face was asymmetric in the right zygomatic maxillary area. Cranial nerve examination was within normal limits. Deep tendon reflexes were pathological and revealed hyperreflexia in the left upper extremity and right upper extremity. There was a nondermatomal pattern loss of sensation over the left arm and left leg. Fine motor activity was impaired in the left hand and dysdiadochokinesia was present. The Babinski sign was positive in the left foot. The Romberg sign was positive. There was noted to be a 2-in. drift of the outstretched left arm with pronator rotation. He could not squat and rise due to left leg weakness.

### Brain Imaging

Figure 8.2 reveals extensive areas of encephalomalacia noted in the right frontal and right occipital areas. Other MRI views reveal lesions in the right subfrontal and right parieto-occipital areas as well. Hemosiderin deposits were present on some MRI slices. The hemosiderin was noted to be present in several of the encephalomalacic areas consistent with a resolved hemorrhagic contusion. There was striking evidence of atrophy of the hippocampal structures bilaterally (Figure 8.3).

Figure 8.4 represents portions of the PET scan obtained on S.K. Arrows indicate reduced metabolic activity in the right frontal and right temporal-parietal areas. The right thalamus is hypometabolic. The remainder of the PET examination, not represented on Figure 8.4, indicated a crossed diaschisis with hyperperfusion of the left cerebellum and hypoperfusion of the right cerebellum (see Chapter 5). While Figure 8.4 represents axial images, the coronal images confirmed the axial findings, as did the sagittal views.

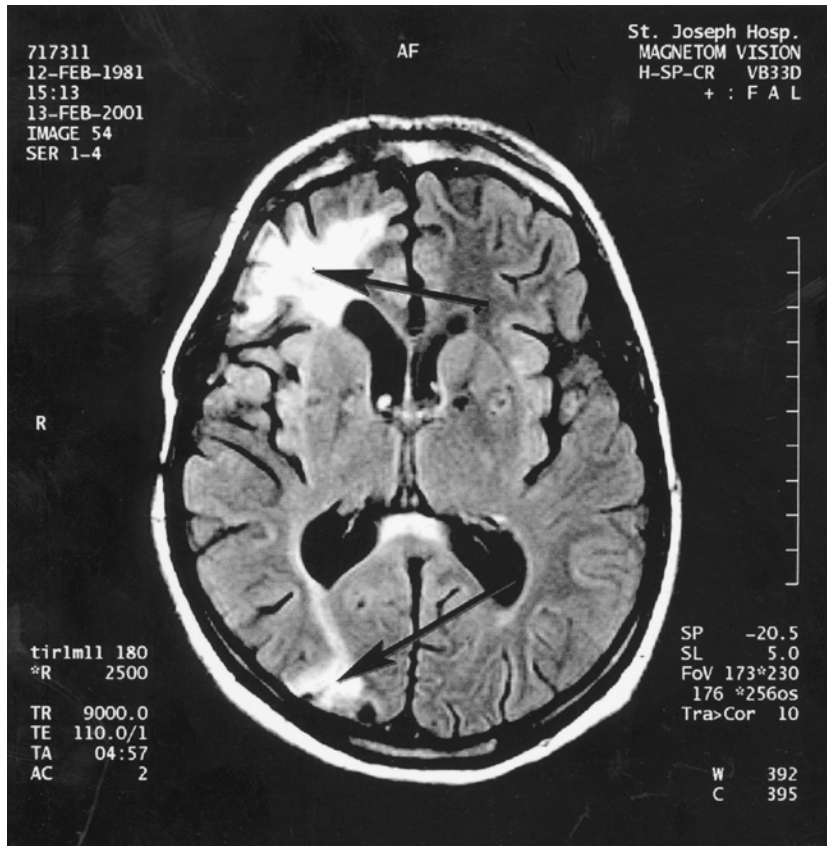
### Standardized Mental Assessment

#### *Measures of Cognitive and Psychological Effort*

His results on the Test of Memory Malingerer revealed a score of 50 on Trial 2. On the Portland Digit Recognition Test, he scored 78%, and the VSVT revealed the scores noted in the table below:

#### Victoria Symptom Validity Test

	Raw Score	Classification
Easy items correct	22/24	Valid
Difficult items correct	16/24	Valid
<b>Total items correct</b>	<b>38/48</b>	<b>Valid</b>



**FIGURE 8.2** T1-weighted MRI of brain. Significant signal change is noted in the right frontal and right occipital areas consistent with encephalomalacia.

Three of three cognitive validity indicators were within normal limits and indicated valid cognitive effort. However, with regard to psychological effort, he had a tendency to acquiesce to item content (TRIN = 72). He also portrayed himself in a negative fashion, which created an impression of significant psychopathology (F = 104, Fb = 112, and VRIN = 61). His validity indicators on the MMPI are noted next:

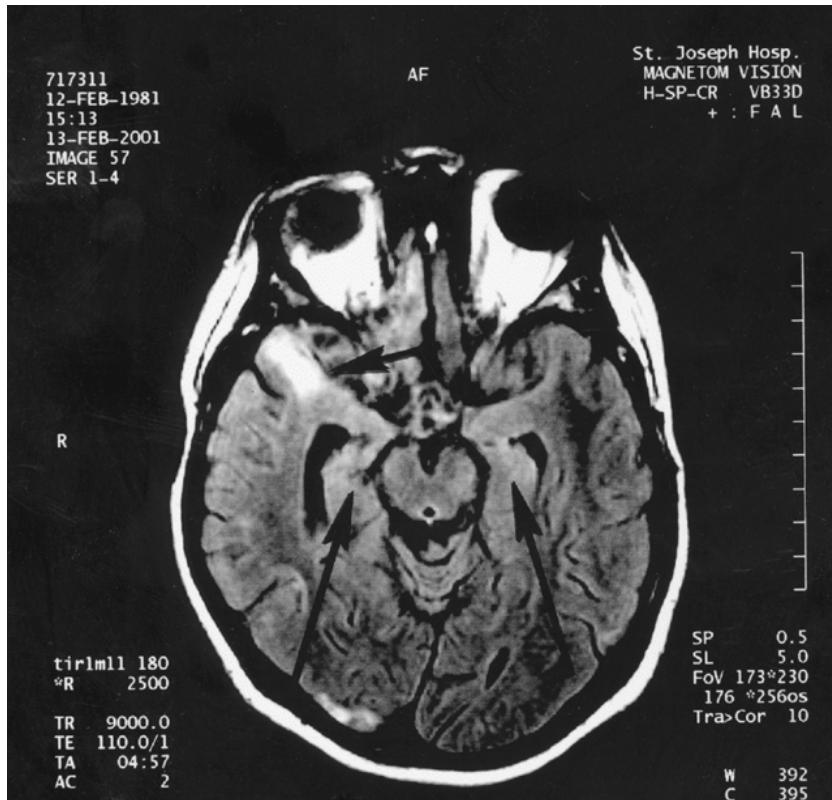
	Cannot Say	L Scale	F Scale	K Scale	F-K Index	Fb	TRIN	VRIN	Fp	S
Raw	0	1	22	7	15	17	12	8	3	5
T-score		39	104	33		112	72	61	63	30

#### *Measures Providing Estimates of Preinjury Function*

S.K. was administered the Wide Range Achievement Test-III, the National Adult Reading Test, and the Vocabulary subtest of the WAIS-III. He produced standard scores of 97, 93, and 100, respectively. Thus, based on these verbal measures, his preinjury verbal functioning was probably within the average range.

#### *Attention and Concentration*

Visual attention was measured using the Ruff 2 and 7 Selective Attention Test. He produced the following results:



**FIGURE 8.3** This is a more inferior slice of the MRI noted in Figure 8.2. The superior arrow indicates continuing evidence of encephalomalacia. The lower two arrows indicate bilateral atrophy of the hippocampi, greater on the right than the left. Note that the right hippocampal cistern is enlarged relative to the left cistern.

### Ruff 2 and 7 Selective Attention Test

Measure	Sum of T-Scores	T-Score	Percentile	Classification
Total speed	< 40	< 20	< 1	Severely impaired
Total accuracy	88	44	27	Below average

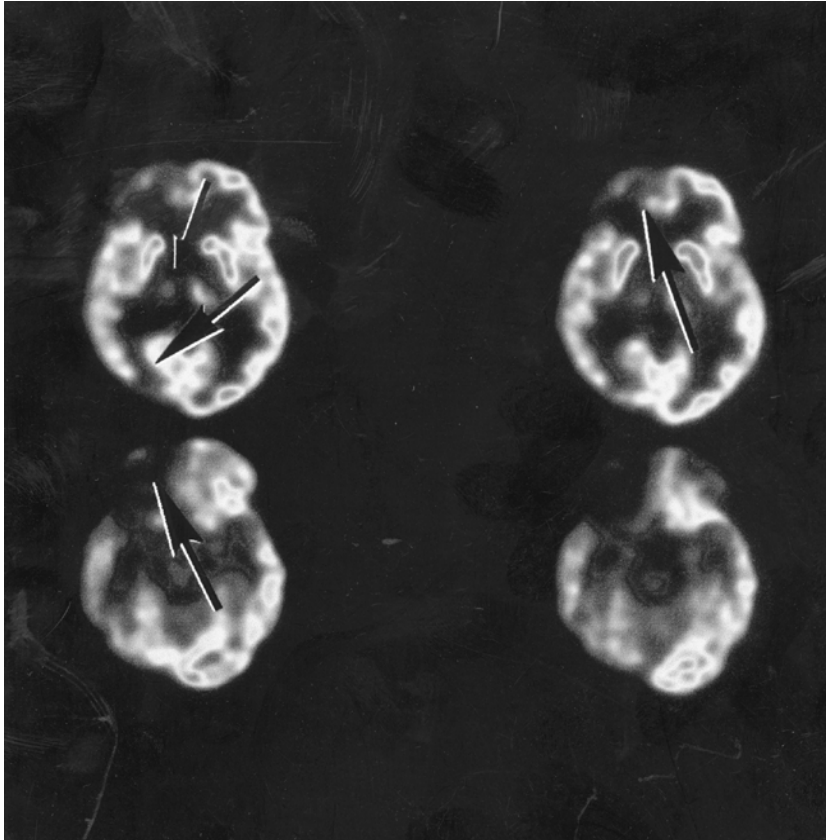
His auditory attention was measured by the BTA. He produced the following profile:

### Brief Test of Attention

	Raw Score	Percentile	Interpretation
Form N (numbers)	9		
Form L (letters)	6		
<b>BTA total score</b>	<b>15</b>	<b>10-24</b>	<b>Low average</b>

### Language and Language-Related Skills

S.K. was administered the Boston Naming Test as an indicator for naming ability. The COWA was administered to determine fluency, and the Token Test was administered to determine receptive language ability. On the Boston Naming Test, he produced a T-score of 29, which was interpreted



**FIGURE 8.4** PET scan images of the person in Figures 8.2 and 8.3. Note that the hypometabolism in the right cerebral hemisphere corresponds to the anterior and posterior right hemisphere lesions noted on MRI. Also note the arrow in the left superior PET image indicating hypometabolism of the right thalamus.

to be moderately impaired, as it was only at the second percentile. On the COWA, he produced a T-score of 26, which placed him at the first percentile. On the Token Test, he produced a score at the 12th percentile, which is within normal limits.

#### *Visuospatial Skills*

He was administered the Judgment of Line Orientation and the Ruff–Light Trail Learning Test. On the Judgment of Line Orientation, he produced a score at the 40th percentile, which was within the average range. However, on the Ruff–Light Trail Learning Test, he was in the severely deficient range for learning, borderline range for immediate memory skill, and deficient range on a measure of delayed memory.

#### *Memory*

Memory was assessed using the WMS-III. He produced the following scores:

#### **Wechsler Memory Scale-III**

	Scale Score			
	Sum	Index Score	Percentile	Classification
Auditory immediate	13	80	9	Low average



## Wechsler Memory Scale-III (Continued)

	Scale Score			Classification
	Sum	Index Score	Percentile	
Visual immediate	6	53	< 1	Extremely low
Immediate memory	19	61	< 1	Extremely low
Auditory delayed	10	71	3	Borderline
Visual delayed	7	59	< 1	Extremely low
Auditory recognition delayed	7	85	16	Low average
General memory	24	64	1	Extremely low
Working memory	13	81	10	Low average

### Sensory Perceptual Skills

On the Reitan–Kløve Sensory Perceptual Examination, he produced the following profile:

#### Sensory Perceptual Examination

	Raw Score	T-Score	Classification	Percentile
Total right errors	10	33	Mildly to moderately impaired	5
Total left errors	6	29	Moderately impaired	2
<b>Sensory perceptual total</b>	<b>16</b>	<b>31</b>	<b>Mildly to moderately impaired</b>	<b>3</b>

### Motor and Visual Motor Skills

These were assessed by measurements using the Grooved Pegboard, Grip Strength, and Finger Tapping Tests. S.K. was impaired in both hands, but as expected from his neurological examination, his level of impairment was much greater in the left hand than the right hand.

#### Grooved Pegboard Test

	Raw Score	T-Score	Classification	Percentile
Dominant right hand	109	19	Severely impaired	0.2
Nondominant left hand	> 300	3	Severely impaired	< 0.01

#### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant right hand strength	40	38	Mildly impaired	13
Nondominant left hand strength	35	34	Mildly to moderately impaired	6

#### Finger Tapping Test

	Mean Raw Score	T-Score	Classification	Percentile
Dominant right hand	43	32	Mildly to moderately impaired	4
Nondominant left hand	21	7	Severely impaired	< 0.01

### Executive Functions

The Wisconsin Card Sorting Test was administered. He produced the following scores:

## Wisconsin Card Sorting Test

	Raw Score	Standard Score	T-Score	Percentile	Classification
Total errors	63	70	30	2	Mildly to moderately impaired
Completed categories	2				
Perseverative responses	45	61	24	< 1	Moderately to severely impaired

He was further administered Trail-Making Tests A and B, and he produced the following profiles:

### Trail-Making A

Raw Score	T-Score	Classification	Percentile
45	30	Mildly to moderately impaired	2

### Trail-Making B

Raw Score	T-Score	Classification	Percentile
121	31	Mildly to moderately impaired	3

### Test Intelligence

This modality was measured by use of the WAIS-III. S.K. produced the following profiles:

### WAIS-III

Subtests	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Picture Completion		4		4		
Vocabulary	10		10			
Digit Symbol-Coding		3				3
Similarities	9		9			
Block Design		5		5		
Arithmetic	10				10	
Matrix Reasoning		8		8		
Digit Span	7				7	
Information	7		7			
Picture Arrangement		3				
Comprehension	8					
Symbol Search		(1)				1
Letter-Number Sequencing	(7)				7	
<b>Sum of scaled scores</b>	<b>51</b>	<b>23</b>	<b>26</b>	<b>17</b>	<b>24</b>	<b>4</b>

### Deviation IQs

	Standard Score	Classification	Percentile	Range
Verbal IQ	91	Average	27	86-96
Performance IQ	67	Extremely low	1	62-76
Full-Scale IQ	78	Borderline	7	74-83

## WAIS-III Index Scores

	Standard Score	Classification	Percentile	Range
Verbal comprehension	93	Average	32	88–99
Perceptual organization	74	Borderline	4	69–83
Working memory	88	Low average	21	82–95
Processing speed	60	Extremely low	< 1	56–74

### Psychopathology

Psychopathology was measured using the MMPI-2. However, as noted, his psychological validity indicators do not allow accurate interpretation of the clinical scales. He produced the following profile:

#### MMPI-2

Scale	L	F	K	Fb	TRIN	VRIN	Fp	S
T-score	39	104	33	112	72	61	63	30

Scale	1	2	3	4	5	6	7	8	9	0
T-score	92	72	81	74	54	75	94	108	85	57

### Records Reviewed

During the neuropsychiatric examination, a large number of records were examined. These included the ambulance report, the community hospital records where S.K. was triaged, the university hospital records where he was treated, the rehabilitation brain injury unit records, and the treatments provided to him by psychiatrists, plastic surgeons, ophthalmologists, and neurologists.

As a further attempt to determine preinjury cognitive capacity, his high school and college transcripts were obtained. He had completed an ACT test, producing a composite score of 20, which placed him at the 49th percentile nationally. He had a grade point average of 2.5 at graduation from high school.

### Diagnoses

He was diagnosed with dementia due to traumatic brain injury. The logic for this diagnosis is very similar to that discussed in Case 1. He was also diagnosed with a personality change due to a general medical condition (310.1). The general medical condition was, of course, the brain trauma. Lastly, he received a diagnosis of cognitive disorder, not otherwise specified (294.9).

### Neurobehavioral Analysis

Again, using the schema from [Table 8.1](#), it is clear that at the accident scene, S.K. demonstrated evidence of severe brain trauma (see [Chapter 1](#)). The GCS was scored 3. The trauma score deteriorated during ambulance transport to the receiving hospital. Thus, there is substantial evidence, before reception at the hospital, that S.K. had a severe brain injury. This, of course, was documented when received at the university hospital. The initial CT scan and subsequent CT scans documented severe brain trauma. He required neurosurgical intervention to evacuate an epidural hematoma. The presence of the epidural hematoma most likely accounted for the deteriorating trauma score during transport to the hospital ([Chapter 1](#)). His hospitalization was prolonged. This case demonstrates the impact of secondary factors upon traumatic brain injury ([Chapter 1](#)). As noted, he had sustained a watershed infarct due to hypovolemia or hypoxemia.

The rehabilitation record provided evidence that he had a left hemiparesis due to right cerebral hemisphere trauma. This did not resolve during his hospitalization. Following his hospitalization, deteriorating behavior required psychiatric intervention. At the time of the neuropsychiatric examination, he was probably undertreated, as his paroxetine dosage was 20 mg daily and his valproate dosage was only 500 mg daily, with a subtherapeutic blood level (37 mg/l).

The neuropsychiatric examination confirms substantial postinjury cognitive and behavioral difficulties. His neuropsychiatric history provided evidence of memory disorder, confusion, irritability, arguing, and excessive anger. He reported substantial geographic disorientation, and he was unable to drive a vehicle due to this dysfunction. Not only did he complain of hemiparetic dysfunction in the left arm, but he was behaviorally quite disturbed and he considered self-amputation of the left hand. His mental status examination was very abnormal, revealing inability to self-monitor language, pseudoeuphoria consistent with infraorbital frontal lobe damage, and impaired prosody (the melodic line was not normal). Thus, while he appeared to have a primary injury to the right cerebral hemisphere, there was also evidence of substantial left cerebral dysfunction in that language usage was impaired.

The neuropsychiatric brain imaging confirmed 15 months after his injury that permanent brain damage was present. The encephalomalacia was documented on MRI. The MRI also revealed bilateral hippocampal atrophy, which in part explains the significant deterioration of memory noted on the WMS. However, the MRI noted primarily deficits in the right cerebral hemisphere. When the PET scan was obtained, diffuse hypometabolism of the cerebral cortex was noted, including the left hemisphere, even though it was clearly better metabolically than the right hemisphere.

When the standardized mental assessment was reviewed, confidence could be placed in the cognitive portion of the examination, but the psychological portion of the examination could not easily be quantified. Since S.K. passed three of three cognitive tests, the examiner can be reasonably certain that the neurocognitive portion of the examination is valid. The invalidity in the psychological examination most likely represents the severe dyscontrol he experiences as a result of frontal brain injury. Recall that on face-to-face examination, he was pseudoeuphoric and disinhibited. The best estimate of his mental function placed him in the average range of cognitive capacity prior to his injury. Therefore, average is the standard against which his other neuropsychological tests are compared to determine if there is an internal consistency. In this case, there was not, and many tests clearly are well below average.

S.K.'s visual attention in particular is impaired for speed. While he was below average in accuracy, he was below the first percentile on the speed portion of the test. On the other hand, auditory attention is probably reduced from preinjury levels, but not as dramatically so as the visual attention. Formal testing did document language impairment in this man, even though the vast majority of his injury is in the right cerebral hemisphere. He produced a Boston Naming Test score at the second percentile, and his COWA score was at the first percentile. These both point to anterior language impairment of the frontal language systems. On the Ruff-Light Trail Learning Test, S.K. showed substantial impairment of visuospatial learning and memory. Since his injury is preferentially to the right cerebral hemisphere, this finding is consistent with the anatomical locus of injury.

His WMS scores are not consistent with his estimated preinjury mental ability. Many of the memory scores are at or below the first percentile, and this would be unexpected in a person with average preinjury cognitive capacity. Recall that he scored 20 on the ACT test and was attending community college. His memory scores are not consistent with that level of functioning.

Since physical examination revealed impairment of sensory perceptual skill, it is not surprising that he performed very poorly on those portions of the neuropsychological assessment. Moreover, the impaired level of executive function is consistent with his behavior on mental status examination and with the history of anger, irritability, and inability to monitor his own behavior. The Wisconsin Card Sorting perseverative response standard score was 61. This is below the first percentile and

not consistent with his preinjury predictions. Trail-Making Tests A and B were at the second and third percentiles, respectively, again confirming substantial executive dysfunction.

On measures of test intelligence, the reader can see that S.K.'s verbal IQ was 91. This is in the average range and consistent with preinjury estimates. On the other hand, his performance IQ was 67. This is a substantial deterioration of test intelligence and confirms the diagnosis of dementia. When reviewing the age-adjusted scaled scores, it can be seen that S.K. was very impaired on Picture Completion, Digit Symbol-Coding, and Picture Arrangement subtests of the WAIS-III. Moreover, his index scores revealed impaired perceptual organization and very impaired mental processing speed. The MMPI was not factored into this analysis due to the distortion of the validity indicators, and assessments of his behavior were made on clinical grounds.

## **Treatment Planning**

S.K.'s functioning was quite poor at the time of the neuropsychiatric examination. It was recommended that the paroxetine be increased to a more therapeutic range of 30 to 40 mg daily. If this proved inadequate after a 2- to 3-month trial, it was recommended that bupropion or venlafaxine also be considered as treatment modalities for the mood component of his injury. With regard to the behavioral disinhibition, it was recommended that the valproate dosage be increased to bring blood levels into the therapeutic range. Likewise, if after 3 months he was showing insufficient response, it was recommended that a trial of lamotrigine be considered, as there is evidence that this antiepileptic drug enhances mental function in the depressive realm. Very little attention was given to this young man's mother. It was determined that she was quite burdened by her severely impaired son. Therefore, it was recommended that his mother be provided both individual therapy and conjoint therapy with her son.

## **CASE 3: CHILD REAR-SEAT PASSENGER**

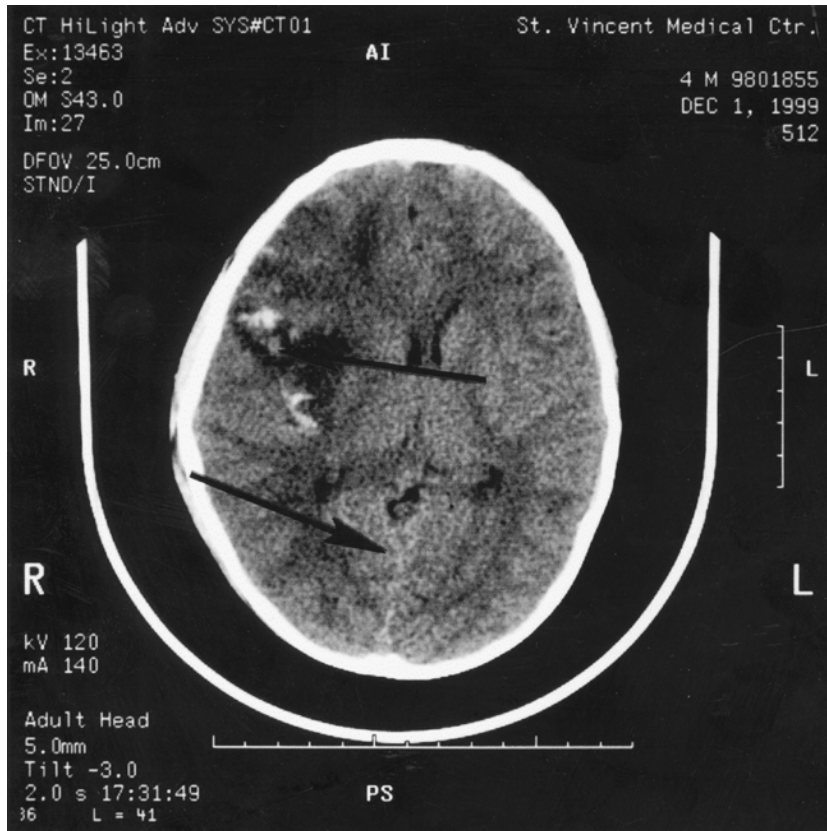
### **Introduction**

This case represents a child. This was a 6-year-old youngster at the time of neuropsychiatric examination. He was injured when he was approximately 4½ years of age while riding in the backseat of a vehicle struck in the rear by a second, larger vehicle. As a result of the impact, a depression was made on the right side of T.R.'s skull. It was thought that the youngster's head hit his sister's car seat upon impact.

### **History of the Accident**

T.R., at age 4½, was riding in the rear seat of the family vehicle. The vehicle was a small four-door sedan struck by a much larger vehicle in the rear. T.R. was attended at the scene by an ambulance service and sent by helicopter to a city hospital near the Great Lakes. When attended by the emergency medical services, he was mentally unresponsive and posturing, and he did not respond appropriately to verbal or painful stimulation. His GCS score was 7. He was combative and had intermittent flexure and extensor posturing at the scene and during helicopter transport.

On reception at the emergency department, a CT scan of the head was obtained. [Figure 8.5](#) reveals that scan and demonstrates blood products about the right Sylvian fissure. Bone windows revealed a comminuted fracture of the calvarium involving the right parietal and frontal bones, which are not noted in [Figure 8.5](#). Review of this figure should demonstrate to the reader a mild right-to-left shift of the midline and mass effect upon the body of the right lateral ventricle. After admission to the hospital, a second CT scan was obtained on the second hospital day. A mild interval improvement was noted with minimal resolution of right hemisphere cerebral edema. T.R. spent 9 days in the hospital and was discharged with diagnoses of a closed-head injury, right temporal lobe intraparenchymal hemorrhage, right cerebral hemisphere edema, right tibial fracture, and left tibial–fibular fracture.



**FIGURE 8.5** CT scan of head taken at the time of injury to a 4½-year-old child. Blood is noted around the area of the right sylvian fissure (temporal lobe). Also note the rightward-to-leftward shift of the falx as a result of distant pressure effects from the hemorrhagic lesion in the right cerebrum.

Neuropsychological evaluation was performed at a large university children's hospital approximately 6 weeks after injury. The primary neuropsychological tests administered were the NEPSY and the Wechsler Preschool and Primary Scale of Intelligence (WPPSI). His overall cognitive ability on this examination was judged to be below normal. He had specific deficits in most nonverbal skills and a general deficit in attention. Fine motor skills were poorer than expected. An occupational therapy evaluation was performed 2½ months postinjury. T.R. was noted to have significant left-sided hypertonicity. He was not functioning at an age-appropriate level. His left hand was fisted, his left elbow was flexed, and he held it in a guarded position close to his chest. He was noted to have left-sided neglect, and he rotated his trunk and pelvis away from the midline. He was poorly cooperative when asked to use his left hand to assist in motor activities. A speech and language evaluation was conducted on or about the same time as the neuropsychological evaluation. T.R. exhibited deficits in attention, reasoning, organization, and short-term memory. He was frequently agitated and required short sessions in order to complete the required tasks. He had specific deficits of expressive language skill.

### History from the Patient

The primary historian was T.R.'s father. When the examiner attempted to interview the parents independent of T.R., he would not tolerate the separation from his mother. Therefore, his father was interviewed independent of the mother. The father reported that T.R. had developed significant

behavioral problems. He perseverated on themes, and once he got involved in a task, he became stimulus-bound and it was difficult to detach him from the task. While engaging in motor behaviors, he could not self-monitor or discontinue the task when appropriate.

The father noted in T.R. some sadness, particularly when T.R. would compare himself to his younger sister. The father also noted poor concentration, memory impairment, word-finding difficulty, and confusion. T.R. was irritable and would cry for no reason. In peer relations, he was overly immature and very shy. He would hit or grab his younger sister when he recognized she was performing certain tasks better than he. He slurred his words when speaking and had difficulty finding words.

Physically, the father reported that T.R. had poor coordination with partial paralysis of the left arm and leg and spasticity in the two extremities. He had developed significant contractures in the left hand and had to wear a bracing splint to reduce the contractures. He was undergoing botulinum injections for spasticity and was treated with 0.75 mg of baclofen three times daily to reduce spasticity.

### **Past Medical and Psychiatric History**

T.R. weighed 8 lb, 14 oz when he was born. He had normal developmental milestones and no significant childhood illnesses. He was a happy child prior to the accident with no obvious cognitive deficits. He had yet to attend school at the time of his accident. Prior to his injury, he had no evidence of a psychiatric disturbance. For the first month following his traumatic brain injury, he was treated with methylphenidate for cognitive stimulation. He had never displayed any form of suicidal or impulsive behavior prior to his injury. He had never intentionally cut, burned, or disfigured himself. He did not hurt or abuse animals prior to his injury.

### **Family and Social History**

Both parents were alive and well. Neither parent had any significant health problems. T.R.'s siblings were in good health. There was no family history of mental or nervous disorders and no history of any neuropsychiatric or neurological conditions. Specifically, there was no family history of ADD or learning disorders. The father was employed as a professional, and the mother was a homemaker with 2 years of college education. There was no evidence of abuse or neglect in the home.

### **Review of Systems and Activities of Daily Living**

T.R.'s parents reported that he had difficulty swallowing and poor ability to maneuver food in his mouth. He would lose liquids from his mouth and had to eat slowly. His sleep review was within normal limits. He reportedly had difficulties with walking, using the left hand, and bending and lifting, all related to the obvious left hemiparesis.

At the time of his neuropsychiatric examination, he had been enrolled in kindergarten in a Montessori school. This school had specific experience teaching handicapped children. After school, he attended physical therapy. He did display a hobby of playing with small cars. He enjoyed television shows that were age appropriate.

### **Mental Status Examination**

He was a pleasant, cooperative youngster with an obvious left hemiparesis. However, he demonstrated significant separation anxiety when an attempt was made to remove him from his mother. He was poorly oriented and not a competent historian. He frequently ran to his mother for assistance during examination. If he perceived he was doing poorly on tasks, he became distressed and would ask for his mother's assistance. He demonstrated an obvious articulation disturbance. Thought and

motor speed were both reduced, and emotions were labile and he was easily agitated. He was not age appropriate and his behaviors were immature for age. Expressive language abilities were poor.

## Neurological Examination

T.R.'s weight was at the 15th percentile for his age and height. He could actively supinate his left forearm. He could throw a large ball, but he required two hands to do so. He could use his right hand for most motor activities. He would attempt to run but did so in a clumsy fashion. He could jump slightly more than 2 ft. He had a drift of the left arm when both arms were stretched before him. Deep tendon reflexes were hyperreflexic in the left upper and lower extremities. His fist was noted to no longer be clenched, but he was receiving active botulinum treatment at the time of the neuropsychiatric examination.

## Brain Imaging

In [Figure 8.6](#), the MRI images reveal an irregular region of encephalomalacia and a cystic loss of brain tissue involving the superior portion of the anterior right temporal lobe and the midportion of the right parietal lobe. Adjacent, there are several small, well-defined foci of encephalomalacia within the deep white matter. Mild *ex vacuo* dilatation of the right lateral ventricle is present. Contrast media were used for images not displayed in [Figure 8.6](#); no abnormally enhancing foci were seen. Other images not available in this text indicated abnormal increased signal on inversion recovery consistent with reactive gliosis.

In [Figure 8.7](#), some of the PET images are displayed. A large, metabolically inactive area is seen in the right parietal region. When all PET images are viewed, this region extends from the level of the Sylvian fissure to high in the cortex and anterior into the frontal lobe. The right thalamus is hypometabolic relative to the left thalamus. The left occipital pole reveals greater activity than the right, probably related to visual activation.

## Standardized Mental Assessment

### *Measures of Cognitive and Psychological Effort*

Due to T.R.'s young age, no specific measures of cognitive or psychological effort were obtained.

### *Measures Providing Estimate of Preinjury Functioning*

As T.R. had attained no formal education prior to his injury, there were no measures that could be provided from his environment to be used for estimation of preinjury cognitive functioning. Generally, that is the case with small children who have been brain-injured prior to formal education.

### *NEPSY Measurements*

#### NEPSY Core Domain Scores

	Core Domain Score	Percentile	Classification
Attention/executive functions	56	0.2	Well below expected level
Language	68	2.0	Well below expected level
Sensorimotor functions	58	0.3	Well below expected level
Visuospatial processing	73	2.0	Well below expected level
Memory and learning	81	1.0	Below expected level

### *Measure of Receptive Language*

T.R. was administered the Token Test. He produced a raw score of 57, which placed him below the first percentile and classified him as low functioning in receptive language ability and verbal





**FIGURE 8.6** MRI of the same child represented in [Figure 8.5](#). This is a T1-weighted image revealing cystic loss of brain tissue and deep white matter encephalomalacia. This MRI was obtained approximately 1½ years following the CT scan in [Figure 8.5](#). This is consistent with the distant neurodegeneration often seen following intraparenchymal hemorrhage.

comprehension of commands. The Token Test is sensitive to even minor impairments of receptive language.

#### *Spatial and Constructional Skills*

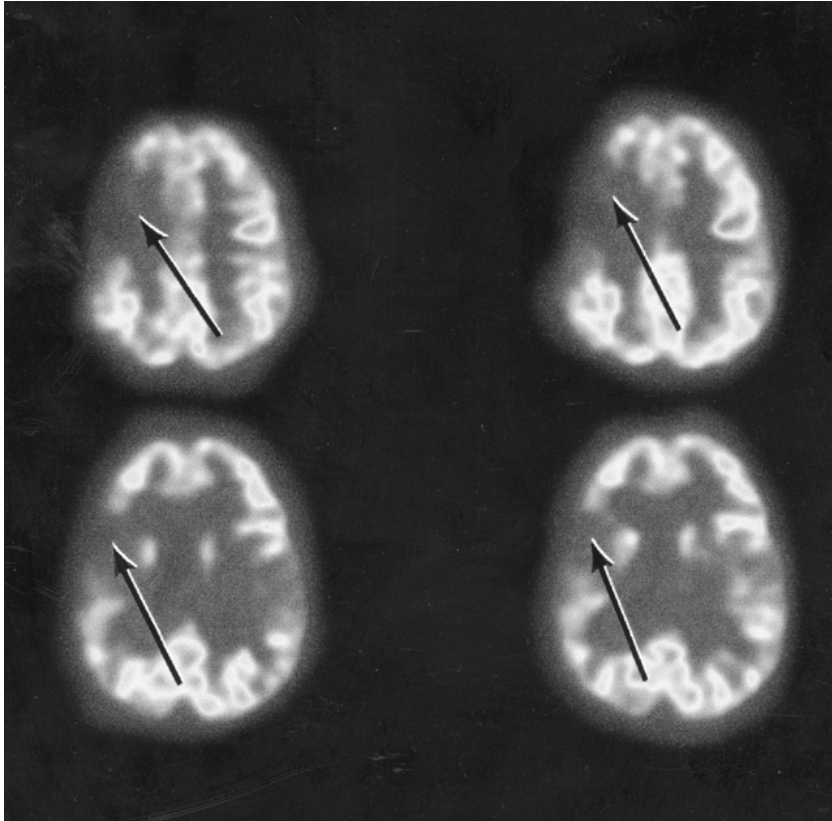
The Hooper Visual Organization Test was administered. Thirty pictures of more or less readily recognized cut-up objects comprise the test, and T.R. was asked to name each object verbally. Cognitively intact persons generally fail no more than six items. T.R. produced 15 failures and the following profile:

#### **Hooper Visual Organization Test**

Total raw score	14.5
T-score	78
Probability of impairment	Very high probability of impairment
Percentile	< 1

#### *Motor Skills*

T.R. was tested on the Grip Strength and Finger Tapping Tests. He produced the following scores:



**FIGURE 8.7** PET scan of child represented in [Figures 8.5](#) and [8.6](#). The area of hypometabolism in the right cerebral hemisphere is quite large and is present on these four tomograms representing a vertical height of at least 4 cm.

### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant right hand strength	9	43	Below average	25
Nondominant left hand strength	n/a	n/a	Severely impaired	< 1

*Note:* n/a = not applicable.

### Finger Tapping Test

	Kilograms	T-Score	Classification	Percentile
Dominant right hand	30	37	Mildly impaired	10
Nondominant left hand	n/a	n/a	Severely impaired	< 1

*Note:* n/a = not applicable.

### Executive Functions

Even though there is an executive domain on the NEPSY, T.R. was also administered the Wisconsin Card Sorting Test. He produced the following profile:

	<b>Raw Score</b>	<b>Standard Score</b>	<b>T-Score</b>	<b>Percentile</b>	<b>Classification</b>
Total errors	86	74	33	4	Mildly to moderately impaired
Completed categories	1				
Perseverative responses	103	58	22	< 1	Moderately to severely impaired

### *Test Intelligence*

T.R. produced the following profile for the Wechsler Intelligence Scale for Children-III (WISC-III):

### **Wechsler Intelligence Scale for Children-III**

<b>Verbal Subtest</b>	<b>Scaled Score</b>	<b>Performance Subtest</b>	<b>Scaled Score</b>
Information	5	Picture Completion	5
Similarities	7	Coding	1
Arithmetic	1	Picture Arrangement	5
Vocabulary	6	Block Design	6
Comprehension	4	Object Assembly	2
Digit Span	(5)	Symbol Search	—
		Mazes	(7)
<b>Verbal score</b>	<b>23</b>	<b>Performance score</b>	<b>19</b>

T.R.'s IQ scores were:

	<b>Standard Score</b>	<b>Classification</b>	<b>Percentile</b>	<b>Range</b>
Verbal IQ	70	Borderline	2	66–77
Performance IQ	63	Extremely low	1	60–73
Full-Scale IQ	64	Extremely low	1	61–70

T.R.'s index scores were:

	<b>Standard Score</b>	<b>Classification</b>	<b>Percentile</b>	<b>Range</b>
Verbal comprehension	76	Borderline	5.0	72–83
Perceptual organization	69	Extremely low	2.0	65–79
Freedom from distractibility	61	Extremely low	0.5	58–74
Processing speed	—	—	—	—

### *Adaptive Behavior*

The Vineland Adaptive Behavior Scale is useful for assessing personal and social sufficiency of individuals from birth to adulthood, and it is applicable to handicapped and nonhandicapped individuals. It is a very useful categorization test, particularly for children. T.R. produced the following profile:

### **Vineland Adaptive Behavior Scale Results**

	<b>Raw Score</b>	<b>Standard Score</b>	<b>Percentile</b>	<b>Stanine</b>	<b>Adaptive Level</b>
Communication domain	186	70	2	1	Moderately low
Daily living skills domain	185	66	1	1	Low
Socialization domain	167	82	12	3	Moderately low

## Vineland Adaptive Behavior Scale Results (*Continued*)

	Raw Score	Standard Score	Percentile	Stanine	Adaptive Level
Motor skills domain	112	50	< 0.1	1	Low
Adaptive behavior domain	218	67	1	1	Low

### *Psychopathology*

The behavior assessment for children was administered using the Parent Rating Scale. The father completed the scale for T.R. and produced the following results:

Scale	Raw Score	T-Score
Hyperactivity	11	54
Aggression	10	53
Conduct problems	2	45
<b>Externalizing problems composite</b>	<b>152</b>	<b>51</b>
Anxiety	13	57
Depression	10	58
Somatization	2	41
<b>Internalizing problems composite</b>	<b>156</b>	<b>53</b>
Atypicality	4	53
Withdrawal	13	76
Attention problems	12	61
<b>Behavioral Symptoms Index</b>	<b>336</b>	<b>59</b>

Adaptive Scale	Raw Score	T-Score
Adaptability	9	36
Social skills	23	50
Leadership	10	37
<b>Adaptive skills composite</b>	<b>123</b>	<b>40</b>

Validity Scale	Raw Score	Classification
F-index	0	Acceptable
Response pattern	91	Acceptable
Consistency	6	Acceptable

The validity profile was within normal limits. Results of the Behavior Assessment System for Children–Parent Rating Scale clinical scales indicated an elevation of the Withdrawal and Attention Problems Scales. In addition, the Adaptability and Leadership Scales were significantly low.

### Records Reviewed

Substantial records were available, including those of the emergency medical service, the receiving hospital, and a children’s hospital medical center, as well as pediatric records, neuropsychology and physical medicine rehabilitation records, and records from an early childhood center.

### Diagnoses

Following the neuropsychiatric examination, T.R. was diagnosed with dementia due to traumatic brain injury (294.1), mixed receptive–expressive language disorder (315.31), amnesic disorder due to traumatic brain injury (294.0), and personality change due to a traumatic brain injury (310.1).

## Neurobehavioral Analysis

Using the schema of [Table 8.1](#), the emergency medical squad records and the helicopter records were reviewed. It was clear that T.R. sustained a severe head trauma at the scene with a GCS score of 7. The emergency department who received T.R. noted a comminuted fracture of the calvarium involving the right parietal and frontal bones. As can be seen in [Figure 8.4](#), there was evidence of right hemispheric blood, cerebral edema, and a right-to-left shift to the midline. A mass effect was evident to the body of the right lateral ventricle.

Following hospitalization, the neuropsychological examination 6 weeks postinjury indicated substantial deficits in nonverbal skills (preferentially right hemisphere) and a general deficit in attention. Little was said about his language function at that time, but the neuropsychiatric examination demonstrates substantial language deficits. As pointed out in [Chapters 2 and 6](#), children often can demonstrate language dysfunction, and in this case, T.R. demonstrates substantial language dysfunction that was not apparent shortly after his injury. The outpatient occupational therapy records did indicate a substantial presence of left hemiparesis with spasticity requiring botulinum injections. The speech and pathology evaluation performed about 6 weeks following his injury did document receptive and expressive language deficits, which persisted to the time of the neuropsychiatric examination.

At this neuropsychiatric examination, T.R. had a rather abnormal mental status examination. His neurological examination was also abnormal and consistent with right cerebral dysfunction. He was not age appropriate in his behavior, nor was he physically developing appropriate to his age. The MRI revealed permanent encephalomalacia occupying much of the anterior right temporal lobe and portions of the middle right parietal lobe. These findings were confirmed on the PET scan, and an even larger area of hypometabolism in the right parietal region was noted. Moreover, the right thalamus appeared smaller and less metabolically active on the PET scan than the left thalamus.

With regard to specific neuropsychological function, upon review of the NEPSY Core Domain scores, it can be seen that T.R. functions between the 0.2 and 2nd percentile on all five domains. When the Token Test was administered to detect receptive language ability, T.R. scored below the first percentile. Spatial and constructional skills, which often are preferentially governed by the right cerebral hemisphere, were notably impaired in this youngster, and he was below the first percentile for that particular domain.

His motor impairment was confirmed neuropsychologically using the Grip Strength and Finger Tapping Tests. A second executive function test was administered, the gold standard Wisconsin Card Sorting Test, which indicated a perseverative response standard score of 58, placing T.R. below the first percentile. On test intelligence, his dementia was documented with a full-scale IQ of 64, placing him in the mildly mentally retarded range of function. The reader is referred to the scaled scores. As discussed previously in this text, arithmetic is often significantly impacted by a brain injury in children, and it is noteworthy that T.R. produced a scaled score of 1 on the Arithmetic Scale. He again shows visuospatial impairment, as his Coding scaled score was also 1. Object Assembly, a test often preferentially controlled by the right cerebral hemisphere, was at a scaled score of 2. T.R. showed significant lack of adaptive behavior, and he also demonstrated impaired behavior on the Behavior Assessment System for Children.

## Treatment Planning

It was recommended that T.R. receive speech and language therapy throughout his educational process. It was also recommended that he have made for him an individualized educational plan so that his educational progress could be monitored throughout his educational life. It was further recommended that his parents receive therapy for the substantial caregiver's stress T.R.'s injury was placing upon them, and it was recommended that the siblings be examined for stress as well. Further recommendation was given that ongoing child psychiatric monitoring during his educational

process would be required in order to determine if stimulant medications would be of assistance with attentional deficits and problematic behaviors. Since T.R. was only 6 years old at the time of his neuropsychiatric examination, he will require continuous monitoring throughout the educational years, and further alterations in his behavioral and cognitive treatment most likely will be required as he matures.

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# 9 Special Properties of Traumatic Brain Injury Forensic Examinations and the Detection of Deception

## INTRODUCTION

The physician performing a neuropsychiatric examination of a traumatically brain-injured patient who is involved in litigation must clearly understand the differences between an examination for treatment or clinical use and an examination for forensic purposes.<sup>1</sup> In the first instance, a clinical psychiatrist or other physician is unconcerned about issues of causation, potential malingering, damages, or other legal constructs that may have importance in the forensic medical examination. The clinical psychiatrist should first be focused upon an accurate assessment of his or her patient and provide the most comprehensive assessment possible to determine the cognitive and behavioral impact of a traumatic brain injury. This level of clinical attention will provide to the patient an optimal setting for developing a treatment plan and for providing therapeutic and psychopharmacologic assistance.

If, on the other hand, the physician is examining a brain-injured patient for forensic purposes, the rules, standards, and ethics are substantially different than those of a clinical examination. It must be understood first that a treating psychiatrist is a health advocate for the patient. As a result, following the Ethical Principles of the American Academy of Psychiatry and the Law,<sup>2</sup> in most instances a treating psychiatrist should not be an expert witness in a forensic matter. It is perfectly appropriate for the treating psychiatrist to testify as a fact witness on the behalf of the patient, but this testimony should provide to the trier of fact how the patient came to be treated by the physician, the nature and scope of the diagnostic examination, the treatment plan, the treatment provided, the diagnosis, and the prognosis. In the same vein, a physician providing a forensic examination should not be a health advocate for the examinee. The physician acting in a forensic matter is the agent for whomever hired the physician. The forensic physician is not an agent for the patient. This difference is critical. Thus, the physician providing a forensic neuropsychiatric examination has a duty to provide a comprehensive evaluation at such a level of expertise that she does not participate in providing to the court erroneous or misleading information. This, of course, requires the physician providing a forensic evaluation to determine whether there has been a brain injury, measure the patient for cognitive effort and the possibility of psychological malingering, and explore issues of causation and damages related to the brain trauma. It also may be necessary for the physician providing a forensic neuropsychiatric evaluation to determine a level of impairment that can be presented to a trier of fact (judge or jury) for a legal determination of the level of injury. If physicians, while performing brain injury evaluations, will maintain a clear distinction between their role as either a treater or an evaluator, they will serve either their patients or the legal system admirably.

## CRITICAL DIFFERENCES BETWEEN TREATMENT AND FORENSIC ASSESSMENT OF TRAUMATIC BRAIN INJURY

The physician performing a treatment examination of a brain-injured patient has a doctor–patient relationship with that person. Therefore, the physician is a health advocate for the patient. Issues of confidentiality must be maintained, and thus, in general, a treating psychiatrist should not be an expert witness, as this may breach confidentiality.<sup>2</sup> For instance, in order to assess malingering, the physician must have doubt and skepticism about the patient’s veracity. While this is appropriate in a forensic examination, it is not appropriate when treating one’s patient. This may interfere with the doctor–patient relationship, which should be held sacrosanct. In contrast, the physician performing a forensic neuropsychiatric examination of a brain-injured patient should function entirely as an independent medical evaluator and not have a doctor–patient relationship with the examinee.

### ARE YOU EXAMINING A PATIENT OR AN EXAMINEE?

The patient issues during brain injury evaluation have been discussed at length in [Chapters 1](#) through [8](#). The remainder of this text will focus upon examinees rather than patients, and the thrust of [Chapters 9](#) to [12](#) concerns the provision of a forensic neuropsychiatric examination of a brain-injured litigant. Therefore, the physician providing a neuropsychiatric examination in this context is functioning as a potential expert, if called to testify, and is not functioning as a treatment physician. The expert is always functioning as a consultant, but this role occurs primarily early when retained by an attorney. The expert generally offers consultation on neuropsychiatric examination of brain injury to the attorney who will not be as knowledgeable about that specialty in most instances. Thus, the role of consultant almost always antedates the role of being a witness.<sup>3</sup> As an expert, it is clear that the forensic physician is entering into a business arrangement with the attorney or other person and is hired within the context of a medical–legal evaluation. The expert is selling time, skill, and consultation. Testimony is never for sale. Only the physician’s expertise, clinical and forensic skills, knowledge, and time are sold contractually within the context of a forensic examination. The best way to think of oneself in this role is that of a teacher–consultant. The physician consults in order to teach the attorney about vagaries of the neuropsychiatric brain injury examination and lastly, if asked to testify, will teach the jury. Further aspects of jury teaching are described in [Chapter 12](#). Unlike treating patients and acting as a health advocate for one’s patient, advocacy is not allowed for the physician employed in a forensic matter. The physician is never to be an advocate for the person being examined or an advocate for the person who hired the physician. On the other hand, the physician should be an advocate for his or her opinions. This is clearly distinguished from being an advocate for the person who hired the physician. It is perfectly ethical for physicians to advocate for their opinions. In fact, a jury will place little credibility in a physician expert who does not respect the opinion she has given.

When employed by an attorney or other person for whom the physician is acting as an agent, the fee relationship should be discussed openly and immediately. Whereas a physician should never take a retainer for treating a patient, as this would be unethical, it is perfectly ethical and even reasonable from a business standpoint to accept the fee before the time of a forensic examination. Although we do not like to admit it, on some occasions, physicians have been taken advantage of by lawyers who did not like the physician’s opinions or testimony and refused to pay the bill. Thus, it is recommended that the fee relationships be discussed immediately at the time the contractual relationship is made between the attorney and the physician and that the fee be paid in advance. Then, the physician can testify with impunity knowing fully that forceful opinions can be given, even if they may not be in the best interest of the person who hired the physician. The physician performing forensic neuropsychiatric examinations must strive always for honesty and objectivity, and this type of fee relationship ensures that this will transpire. It is recommended that the fee never be paid by the person being examined. The physician should avoid this type of fee relationship,

and the fee should always be paid by the person or organization for whom the physician is acting as an agent. Therefore, in an attorney–expert contract, the fee should come from either the attorney or the attorney’s law firm and never from the person being examined, as this might suggest or develop a doctor–patient relationship. If the attorney wishes to charge the examinee the fee, that is between the attorney and client and the physician should not be involved in those matters. This ensures further that the forensic evaluator is definitely not the agent of the subject of the evaluation, even if the examinee is ultimately paying the bill to the attorney.<sup>4</sup>

## **ETHICS AND BOUNDARY ISSUES OF THE FORENSIC NEUROPSYCHIATRIC EXAMINATION**

Remembering that the practice of forensic medicine is a business, one must negotiate the time involved in preparing a case. The expert examiner who intends to perform neuropsychiatric examinations on a regular basis should determine how fast he reads and how many materials he can review in a given hour or other time quantification. For instance, if it requires an hour to read an inch of paper or 100 pages, the expert examiner can roughly predict the time required by the number of deposition inches or pages. This in turn allows the examining physician to estimate the time required in order to set a fee for the attorney.<sup>4</sup> However, if more time is required, additional charges can be supplemented ethically to the original retainer fee. Preparation for a deposition is clearly a billable time and should be charged on an hourly basis. Preparation and reviewing of documents for court testimony likewise should be charged on an hourly basis. On the other hand, it may be more practical for the expert examiner to set a base rate for the type of neuropsychiatric examination. For instance, if most brain injury examinations contain the same components and require the same amount of time and the same amount of radiology, nuclear medicine, and psychological consultation, then it is appropriate for the examining physician to set a base rate fee for a particular type of neuropsychiatric examination and supplement the fee with hourly charges as required. Obviously, some cases may require the review of multiple depositions and 15 or 20 in. of medical records, and this, of course, would require an hourly charge at a fee different than a case that had only a single medical record of 150 pages.

The expert physician should recognize that examinees do not distinguish clearly the difference between a clinical and a forensic examination. Examinees generally believe that, when they see a doctor for evaluation and diagnosis, the physician is functioning in a manner no different than their personal physician or the physicians who treated them at the time of their original brain injury. Even with clear explanations to examinees, supplemented in writing if necessary, examinees still may not understand the nature of the examination. Thus, it is not unusual during the course of the examination for the examinee to ask the physician what she thinks about the tests or what she thinks about the neurological examination or what her opinion is regarding the medical records. Recall that no doctor–patient relationship exists. At this point, it is wise to politely remind the person being examined that your report will be made available to the party who hired you and that the examinee should consult with his lawyer for further information regarding your examination and opinions.

In order to be clear about such issues and to insure that examinees are not unduly confused by the forensic neuropsychiatric examination, it is recommended that at the top of the intake history questionnaire a warning statement be given that the examination is not for purposes of treatment, no doctor–patient relationship will exist, and the examination may be audio- or video-recorded (see [Chapter 10](#)). If recording does take place, the examinee may request a copy of the tape through his attorney. During the history process, orally remind the patient of these factors again, if necessary. If the examinee is illiterate, of low intelligence, or otherwise cognitively compromised, it may be necessary to provide cautionary statements to the examinee’s advocates or guardian. With a child examinee, obviously the parent(s) would be told this information.

After the examinee has left the physician's office, it is not uncommon then to receive telephone calls requesting information about the examination. The same caveat is recommended. Thus, the physician should direct the examinee to consult with his attorney. At all times, the examining physician should maintain a distance between herself and the examinee such that boundaries are not crossed. It is very easy for a compassionate physician to have some difficulty switching modes from the clinical realm to the forensic realm, and one must maintain high levels of cognizance in order to ensure that there is no inappropriate blurring of boundaries during forensic examinations.<sup>5</sup>

Within the context of a forensic neuropsychiatric examination for brain injury, the examinee or his guardian should sign a waiver for the physician to examine the patient, a waiver for psychological testing to be performed, and a waiver to send the information to the party for whom the physician is acting as an agent, and lastly, the examinee should be asked to sign a statement and certify that he is telling the physician the truth regarding the history. Waivers for neuroimaging and laboratory testing will be obtained by the facility providing the services. Moreover, it is wise to make it a practice to have each examinee complete an extensive medical questionnaire that asks a comprehensive medical, neuropsychiatric, social, and family history of the patient and also provides a review of systems for the patient (see [Chapter 10](#) for suggested questionnaire). It is this documentation that is then signed by the patient for truthfulness. Should issues of the credibility of the examination arise later in the litigation, this document can prove invaluable within the context of the examiner's overall assessment.

## **THE ADMISSIBILITY OF SCIENTIFIC EVIDENCE**

Rules that govern the admissibility of scientific evidence at trial have undergone a profound transformation within the last 50 years. In the early part of the 20th century, U.S. law relied upon common law concepts for admissibility of scientific evidence, and there were no standardized rules for the use of scientific evidence as testimony. The utilization of expert testimony at court dates back more than 2000 years. In Roman times, the judge had discretion to bring experts to inform the court about unknown "scientific" phenomena. In 1606, Henry IV of France appointed coroners in all cities and important towns, and entrusted to them the duty of examining all murdered persons and reporting the findings to the court. The famous criminal code framed by Emperor Charles V at Ratisbon in 1532 required that the opinion of medical experts be heard in essentially all murder cases. The basic common law test regarding the admissibility of expert testimony was a simple one. If the person offered as a witness was "qualified" as an expert in his or her field, that person was competent to render expert testimony.<sup>6</sup> More recently, many legal scholars have expressed concern about the introduction of so-called "junk science" into the courtroom.<sup>7</sup> In 1923, the *Frye* test became the standard of general acceptance in a particular scientific field.<sup>8</sup> *Frye* was the law of the land until the famous *Bendectin* case.<sup>17</sup> *Bendectin* was a commonly used antiemetic prescribed by physicians for hyperemesis during pregnancy. Merrell Dow Pharmaceutical Company was sued with the plaintiff alleging a birth defect in her child as a result of consuming *Bendectin* during pregnancy. During the litigation, the lower court had excluded the plaintiff's medical experts from testifying and a judgment was entered for Merrell Dow. The legal appeals and subsequent cases generated by this lawsuit have led to current modern standards for the admission of scientific evidence at court.

### ***Frye v. United States: General Acceptance Standard***

The issue in this case was the defendant's offer of an expert witness to testify that the defendant was truthful when he denied he had committed the charged crime. The expert reached a conclusion by using a systolic blood pressure "deception test." This was a crude precursor of the current polygraph or "lie detector" machine. The case was heard in the District of Columbia Circuit Court in 1923. The opinion of the court stated: "Just when a scientific principle or discovery crosses the



line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while the courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs.”<sup>8</sup> The court concluded that the systolic blood pressure test had not yet gained such general acceptance. *Frye* received little attention initially. However, after World War II, courts were flooded with newer forms of expert testimony and *Frye* was suddenly rediscovered by lawyers. Initially, *Frye* was applied only in criminal cases, and it was used to determine the admissibility of opinions derived from voice prints, neutron activation analyses, gunshot residue tests, bite mark comparisons, use of sodium pentothal for interview, scanning electron microscope analysis, and many other forms of scientific inquiry.<sup>9-14</sup> It was not until 1984 that *Frye* first was applied in a civil case.<sup>15</sup> *Frye* became more and more established as a standard in civil cases, and in the 1980s and early 1990s, it was thought that the general acceptance test under *Frye* would eliminate the need for hearings on the validity of innovative techniques.<sup>16</sup>

*Frye* endured from 1923 until 1993, but it was strongly criticized throughout that time period. It was argued that it worked too well and resulted in not only the exclusion of unreliable evidence but also potentially reliable evidence.<sup>16</sup> Courts also faced a daunting problem of identifying which scientific field would “generally accept” a new test. Since new scientific tests and evidence approach old problems, many scientific techniques thus overlap and involve two or more academic disciplines or professional fields. A professional in one field might well develop a new test that lies on the fringes of an existing discipline and spawn an entirely new profession. This has been seen clearly in medicine with the advent of computed tomography (CT) scanning, which is in the realm of radiology, whereas magnetic resonance imaging (MRI) scanning is not only in the realm of radiology but, since it does not use radiation, overlaps into the imaging field in general. Single photon emission computed tomography (SPECT) scans and positron emission tomography (PET) scans are not “x-rays,” but rather, they rely upon radioactive tracers, thereby belonging in the nuclear medicine field, which is distinct and apart from that of diagnostic radiology. Yet, as we have seen in [Chapter 5](#), CT, MRI, SPECT, PET, and the newer techniques of functional magnetic resonance imaging (fMRI) and magnetic resonance spectroscopy (MRS) have evolved and continue to evolve. These rapid changes in scientific techniques applied to brain injury pose challenges to the admission of scientific evidence at trial.

## THE *DAUBERT* RULE

In *Daubert*, the plaintiffs were two infants who sued Merrell Dow Pharmaceuticals, alleging that they had suffered phocomelia (limb reduction) birth defects as a result of their mother’s ingestion of Bendectin, a product manufactured by the defendant, Merrell Dow. At trial, Merrell Dow moved for summary judgment based on the testimony of Stephen Lamm, M.D. He had served as a birth defect epidemiology consultant for the National Center for Health Statistics and was well published in the scientific area of teratology risk from exposure to chemicals and biological substances.<sup>17</sup> The plaintiffs did not dispute Dr. Lamm’s characterization that the relevant medical literature produced no studies concluding that Bendectin caused human birth defects. They responded to the defendant’s position by presenting eight experts who concluded that Bendectin can cause birth defects. However, the plaintiff’s experts based their conclusions on animal cell studies, live animal studies, and chemical structure analyses, as well as recalculations of prior data presented in the medical literature that found no causal connection between Bendectin and birth defects. Even in light of this evidence, the trial court granted to the defendant a motion for summary judgment. The trial court concluded that Bendectin did not cause birth defects and that the plaintiff’s expert opinion was not admissible, as it was not sufficiently established to have a general acceptance in the field to which it belonged. The recalculations offered by the plaintiff’s experts were judged to be inadmissible since they had

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**TABLE 9.1**  
**The *Daubert* Rule**

- The witness must be qualified by training, study, or experience to express an expert opinion.
  - The witness must have personal experience in dealing with the specific technical and scientific application that is the subject of the court's inquiry.
  - Can the expert's basis for opinion be tested or has it been tested?
  - Have the expert's techniques, theories, or scientific concepts been published in peer-reviewed journals?
  - What are the known potential rates of error or standards controlling the expert's examination techniques?
- 

never been published in a peer-reviewed medical journal. The U.S. Court of Appeals for the 9th Circuit affirmed the District Court ruling based on the standard of general acceptance, and the U.S. Supreme Court granted certiorari. The Supreme Court went on to list factors that could be considered to establish scientific credibility, for example, testing, peer review, publication, and known or potential rate of error of the test or data. Thus, general acceptance could be considered as only one factor in the admission of scientific evidence.

As Gutheil and Simon point out,<sup>4</sup> an expert must perform an adequate forensic examination. This methodology requires considering readily available collateral sources of information, especially corroborating ones. The expert should employ psychological testing, where appropriate, and present reliable and objective data of effort, malingering, and mental state. These can reveal attempts at feigning, minimizing, or exaggerating mental conditions. A face-to-face examination for brain injury, with only a mental status examination, history, and review of records, probably would be inadequate and incomplete. [Table 9.1](#) lists the basic requirements to satisfy the *Daubert* rule.

**CASE LAW SINCE *DAUBERT***

The use of expert testimony at court was expanded with the *Joiner* case.<sup>18</sup> *Joiner* alleged that his exposure to certain chemicals led to lung cancer. The trial judge excluded testimony offered by *Joiner's* experts, basically stating that it was no better than speculation. The case was appealed and the appellate court, applying a stricter standard of review, reversed the trial court. The case was appealed to the U.S. Supreme Court, which reversed the appellate court's decision. It restated the trial judge's exclusion of plaintiff's experts. The Supreme Court ruled that the trial judge did not exceed his level of discretion in the case. The Supreme Court noted that a trial court may conclude that there is simply too great an analytic gap between the data and the opinion proffered. In the *Joiner* case, the *New England Journal of Medicine* (N.E.J.M.) filed an *amici* brief noting that judges should be encouraged strongly to make greater use of their inherent authority to appoint experts. Reputable experts could be recommended to courts by established scientific organizations such as the National Academy of Sciences or the American Association for the Advancement of Science. The *N.E.J.M.* went on to opine that, given the offer of cooperative effort from the scientific to the legal community, the court's task in implementing *Daubert* would not prove inordinately difficult to implement.

There were still concerns in the legal community after *Joiner* that *Daubert* was not strong enough to raise the threshold for junk science to be presented to juries. *Kumho Tire v. Carmichael* advanced the issues in *Daubert* to a new level.<sup>19</sup> The plaintiff's automobile tire blew out. The plaintiff used as an expert a person holding himself out as a tire failure analyst. The trial court excluded this expert's testimony and entered a judgment in favor of Kumho Tire Company. The judge argued against the reliability of the tire analyst's methods. When appealed, the trial court's decision was reversed. The court of appeals concluded that the tire failure analyst based his conclusions on experience rather than science, and as a result, the district court was in error as a matter of law by applying *Daubert* rules in this particular case. The U.S. Supreme Court heard the case and, in part, argued that an expert's testimony can be tested for reliability using *Daubert*, even

when that testimony is experience-based. The Supreme Court concluded that it is appropriate for a trial judge to ask whether such a method is generally accepted in the relevant scientific community, and furthermore, the expert can be asked whether his preparation is of a kind that others in the field would recognize as acceptable. *Joiner* and *Kumho* both endorsed the idea that the legal standard for permitting expert testimony to be heard by the jury is the very same standard that the relevant professional community employs.<sup>4</sup>

In light of *Daubert*, *Joiner*, and *Kumho*, Gutheil and Simon<sup>4</sup> have offered suggestions wherein an expert examiner may address questions of scientific validity regarding the expert's own opinions. They note that expert opinion is strengthened by drawing upon recognized clinical entities (such as the diagnostic and clinical categories noted in [Chapter 2](#) that are associated with traumatic brain injury). They further point out that literature review and the use of citations that are "on point" are extremely useful techniques for meeting the requirements of both a general acceptance standard and a scientific reliability standard. A forensic expert functioning in a traumatic brain injury case should be able to provide empirical, scientific, or consensus bases for opinions. Data useful for fulfilling these functions might be taken, for example, from clinical studies of traumatic brain injury, task force reports from the neurosurgical, neurological, neuropsychiatric, and psychiatric literature, official practice guidelines from medical disciplines relevant to the evaluation and treatment of traumatic brain injury, and other relevant sources. The question to be asked by the neuropsychiatric examiner is: Do my medical discipline and examination techniques have anything to say about this case at all? If the answer is yes, the examiner must then consider whether her particular expertise can assist the fact finder to understand some relevance to the legal issues regarding brain injury at hand. In complex cases, Gutheil and Simon suggest peer consultation. However, they caution that it is unclear whether such consultation might be legally discoverable.

## **DETECTION OF DECEPTION DURING NEUROPSYCHIATRIC EXAMINATION OF TRAUMATIC BRAIN INJURY**

The forensic examiner is obligated to consider the possibility of malingering in a forensic assessment done for any purpose.<sup>4</sup> This, of course, includes examination for cognitive and behavioral effects of traumatic brain injury. Failure to consider malingering constitutes substandard practice for a forensic neuropsychiatric examination.<sup>20,21</sup> Malingering and factitious disorder are legitimate psychiatric diagnoses and are found in the current *Diagnostic and Statistical Manual of Mental Disorders* (DSM).<sup>22</sup> The detection of malingering is becoming increasingly scientific and reliable, and malingering should never be an exclusion diagnosis arrived at merely because certain symptoms are or are not present.<sup>4</sup> Even with children, distortion and effort factors must be considered when psychic trauma is an issue in civil litigation.<sup>23</sup>

### **MALINGERING**

Malingering is a condition not attributable to a mental or physical disorder. It is defined as the intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives, such as financial compensation. In contrast, factitious disorders involve the intentional production of symptoms in order to assume a patient or sick role. Both disorders require a deceitful state of mind.<sup>24</sup> Resnick describes subcategories of malingering. Pure malingering is the feigning of disease when it does not exist at all in the particular person. Partial malingering is the conscious exaggeration of existing symptoms or the fraudulent allegation that prior genuine symptoms are still present. In addition, false imputation refers to the ascribing of actual symptoms to a cause consciously recognized to have no relationship to the symptoms. For instance, cognitive or behavioral symptomatology due to marital or family stress may be falsely attributed to a traumatic event at work in order to gain compensation.<sup>25</sup> Malingering a mental disorder usually occurs for one of five purposes:

1. Criminals may seek to avoid punishment by pretending to be incompetent to stand trial or insane at the time of the crime.
2. Malingerers may seek to avoid induction into military service, avoid combat, or be relieved from undesirable military assignments.
3. Malingerers may seek financial gain from Social Security Disability, Veterans Administration benefits, workers' compensation, or legal damages.
4. Prisoners may malingering to obtain drugs or to be transferred to a psychiatric hospital.
5. Malingerers may seek admission to a psychiatric hospital to avoid arrest, to obtain free room and board, or to seek medication.<sup>24</sup>

The assessment of a potential malingered traumatic brain injury is difficult. There is little, if any, research support for a “malingering profile” within a brain injury evaluation.<sup>26,27</sup> The examiner should not expect an actual malingerer to acknowledge deception. As a result of lack of admission, it is difficult to identify real-life malingerers for an empirical comparison.<sup>28</sup> Many evaluations of traumatic brain injury occur within civil or criminal litigation. Therefore, the examiner must rely on both direct and indirect measures of malingered brain injury. Apparent inconsistencies in the examination, misrepresentations in the examination, and poor performance on neuropsychological instruments beyond the level of apparent brain injury should always raise the suspicion of malingering.<sup>29,30</sup>

Basically, there are two ways to malingering in a brain injury evaluation: (1) cognitive or neuropsychological, and (2) psychological. Fakers, attempting to feign believable neuropsychological or cognitive deficits, often present with a particular strategy. The following is a summary of the types of response styles that fakers may present to the examiner in their attempt to produce false cognitive and neuropsychological symptoms:<sup>31</sup>

1. *Present realistic symptoms:* The faker may employ a common sense or popularly understood schema of what brain-damaged persons are thought to be like. They will present symptoms with that naïve view. It is hoped that the expert evaluator will have a more objective and detailed view of realistic neurological and neuropsychological symptoms in order to see through the charade.
2. *Distribute errors:* Fakers tend to make a deliberate number of mistakes throughout their evaluation rather than miss only difficult items. They attempt to seek a balance between missing too few items and appearing too impaired by missing too many items on the tests. From a practical standpoint, fakers are unable to maintain a realistic percentage of errors, and they can be detected in this fashion.
3. *Protest that tasks are too difficult or feign confusion and frustration:* The faker may appear confused or angry or display other emotions that are superimposed upon reasonably adequate cooperation and task compliance.
4. *Perform in a crudely estimated fraction of their actual ability:* Speed may be deliberately decreased. Since many neuropsychological assessments have a time component, this is an excellent way to fake the examination. The evaluator should search for failures on easy test items in the neuropsychological assessment. The physician forensic examiner will need to rely upon the neuropsychologist or psychologist for assistance in this regard.
5. *Errant affective style:* Many traumatic brain injuries will produce changes in the expressed affect (see [Chapter 2](#)). Fakers may employ changes in affect as part of their malingering strategy. This can be a difficult response style to detect, but the psychological validity measures described next will assist the examiner in determining whether this has occurred. The *Minnesota Multiphasic Personality Inventory-2* (MMPI-2) in particular will provide the examiner with valuable information regarding behavioral, emotional, and psychiatric issues. The scales determining response bias on the MMPI-2 are partic-

ularly useful if affective coloring is being used as a deception technique during the cognitive portion of the testing.<sup>32</sup>

From a psychological malingering standpoint, evaluating psychological credibility of persons who have sustained a brain injury is one of the most fundamental and yet often difficult forensic tasks. However, it must be done, and as noted previously, in a forensic neuropsychiatric evaluation, failure to evaluate the cognitive and psychological credibility of a traumatic brain injury litigant is substandard or negligent on the part of the forensic neuropsychiatric examiner.<sup>20,21</sup> Persons being evaluated within the context of litigation often tend to respond to personality test items in an effort to create a particular impression for the examiner. This “impression management” can be detected by many psychological tests. Moreover, the forensic neuropsychiatric examiner must assume that the person being examined has been coached by his attorney. The extent to which attorneys brief their clients prior to their being assessed in a forensic evaluation is likely to be considerable. Wetter and Corrigan conducted a survey of 70 attorneys and 150 law students with respect to whether they briefed their clients before they were administered psychological tests. Attorneys and law students considered it their responsibility to consult with their clients on testing beforehand to prepare them for the evaluation.<sup>33</sup> Moreover, the forensic neuropsychiatric examiner should expect in some instances to be asked which tests will be administered to a person’s client before the attorney will allow the client to appear for examination. An important question derives from this coaching issue: Can individuals successfully fake the results of their psychological tests if they are informed in advance about the validity scales? Rogers and others<sup>34</sup> found that criminal legal clients can be instructed in strategies that will allow them to present a faked clinical pattern on the MMPI-2 and avoid detection by the MMPI-2 validity indicators such as the F scale. The MMPI-2 F scale was ineffective at detecting coached simulators from genuine patients with schizophrenia. However, Storm and Graham<sup>35</sup> were unable to replicate the findings of Rogers and others. They did find that the Fp scale was effective in detecting both uncoached and coached malingerers. Pope and others<sup>36</sup> believed that the coaching of symptoms does not appear to influence the detection of malingering. However, they cautioned that the distortion of test results by coaching can be difficult to determine. Obviously, if a criminal defendant is managing the impression produced on an MMPI test, this could have serious implications for the detection of psychopathology or antisocial personality disorder. On the other hand, a brain injury litigant who manages the psychological test profile in an effort to produce a normal symptomatology shoots himself in the foot with regard to litigation, as obviously there would be no detection of a significant mental disorder that might assist him in his claim of brain injury. Yet, it may be rather easy to fake pathology during a brain injury examination.

With regard to the detection of malingered psychological symptoms by mental status examination only, Resnick offers one of the best descriptions of simulated psychiatric symptomatology that exists in the medical literature.<sup>24</sup> He points out that persons suspected of feigning auditory hallucinations should be asked what they do to make the voices go away. Persons having actual auditory hallucinations will usually report that the voices tend to diminish if they are involved in activities. With brain-injured examinees, this information may not be forthcoming and the person is more likely to report that no activity or influence diminishes the extent of the voices. With regard to the report of visual hallucinations, dramatic, atypical visual hallucinations should arouse suspicions of malingering. In true psychotic disorders, visual hallucinations appear suddenly without a prodrome. Psychotic hallucinations do not change if the eyes are closed or open. Organically induced hallucinations more readily occur under low light conditions, with the eyes closed or in darkened surroundings (conditions of reduced sensory input).<sup>37</sup> The distinction of malingering from conversion disorder can be extremely difficult. However, the MMPI-2 should help in this regard, as one would expect the conversion pattern on scales 1, 2, and 3 to be present in the absence of abnormal validity indicators. Also, the critical element that distinguishes conversion disorder from malingering is that conversion symptoms are not under voluntary control. Resnick<sup>24</sup> offers

clinical characteristics that may assist in a differential diagnosis between malingering and conversion disorder:

1. The malingerer often presents as sullen, ill at ease, suspicious, uncooperative, resentful, aloof, secretive, and unfriendly. Persons with conversion disorder are more likely to be cooperative or appealing and dependent.
2. The malingerer may try to avoid examination, unless it is required as a condition for receiving some financial benefit. While the malingerer may decline to cooperate with recommended diagnostic or therapeutic procedures, patients with conversion disorder are typically eager for an organic explanation for their symptoms.
3. The malingerer is more likely than the person with conversion disorder to refuse employment if offered.
4. The malingerer is likely to give every detail of the accident and its sequelae; the person with conversion disorder is more likely to give an account that contains gaps and inaccuracies, and it may be vague and generalized.

The neuropsychiatric examiner must be particularly suspicious of posttraumatic stress disorder (PTSD) claims in association with a brain injury claim where the evidence for injury is slight or nonexistent. It is extremely easy to be coached on the details of posttraumatic stress disorder, and most skilled plaintiff attorneys have a copy of the DSM. On the other hand, as noted in [Chapter 2](#), PTSD is a frequent outcome and often associated with a traumatic brain injury. However, defense attorneys often assume an attitude of disbelief when PTSD is raised as an issue, and they imply that the individual is not suffering from any genuine psychiatric symptoms. The examiner also should consider the issue of partial malingering, that is, a person with an actual traumatic brain injury who is exaggerating the psychological component of the injury in order to enlarge the claim by including false elements of PTSD.

With regard to children, the examiner must consider, within the evaluation context, either malingering by proxy or Munchausen syndrome by proxy. Young children are extremely coachable and suggestible.<sup>38</sup> With the Munchausen by proxy disorder, suggestibility also plays a role. Munchausen syndrome is a recognized mental disorder and is a factitious disorder. The parent or guardian, through the child, intentionally produces or feigns physical or psychological signs or symptoms. The motivation is to assume the sick role. However, in some instances, assuming the sick role becomes the genesis for litigation, the Munchausen disorder moves from a factitious illness to a malingered disorder. Thus, if the child is being seen repeatedly in emergency rooms or doctors' offices with a claim of brain injury and there is no evidence for financial gain or litigation, the child and his or her parent or guardian would properly be placed in the category of factitious disorder. If, on the other hand, there is obvious economic gain, litigation, or pursuit of insurance monies, then the disorder may be transformed to malingering by proxy.<sup>39</sup> [Table 9.2](#) categorizes types of false mental symptoms.

## **DETECTION OF COGNITIVE MALINGERING**

It is expected in a complete forensic neuropsychiatric examination of traumatic brain injury that the examining physician will utilize psychological and neuropsychological testing to determine the presence or absence of cognitive malingering. Physician examiners are cautioned that they must determine the adequacy of cognitive effort detection on the part of the psychologist or neuropsychologist used to assist in the neuropsychiatric examination. Most psychologists and neuropsychologists come from a clinical background. Therefore, they routinely avoid extensive measurement of cognitive effort or fail to utilize extensive tools to detect cognitive malingering. It is extraordinary how many forensic brain injury examinations by psychologists and neuropsychologists in the U.S. fail to include effort controls or methods to detect cognitive malingering. The neuropsychiatric

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**TABLE 9.2****Syndromes of False Mental Symptoms Common to Forensic Neuropsychiatric Assessment**

Conversion disorder:	Symptoms or deficits that mimic a motor or sensory neurologic disorder or other medical condition; the disorder is not voluntary and is unconsciously produced <sup>22</sup>
Symptom magnification:	Conscious exaggeration of existing symptoms or the fraudulent allegation that a prior genuine psychological or cognitive disorder is still present <sup>24</sup>
False imputation:	Ascribing actual symptoms to a cause consciously recognized to have no relationship to the symptoms (e.g., marital stress falsely attributed to a traumatic event in the workplace) <sup>24</sup>
Factitious disorder:	Intentional production of physical or psychological signs or symptoms; the motivation is to assume the sick role <sup>22</sup>
Malingering:	Intentional production of false or grossly exaggerated physical or psychological symptoms; motivation is to fake for an external incentive <sup>22</sup>
Ganser syndrome:	A form of mental malingering often seen in criminals awaiting trial; it is characterized by approximate answers (e.g., How many legs are on a three-legged stool? Answer: 4; What color is snow? Answer: green.); this has been termed <i>Vorbeireden</i> <sup>102</sup>

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examiner has a duty to ensure that cognitive malingering examinations have been completed within the context of the psychological or neuropsychological testing before presenting this as a part of the evidence in court. Thus, when the physician examiner contracts with a psychologist or neuropsychologist to provide psychological testing services, it is mandatory to ask the psychological examiner to measure for cognitive malingering.

The physician examiner must also make some inquiry as to the type of testing used to detect malingering. The use of techniques that are not based on probability theory are to be avoided, as they are unreliable and their error rates will cause them potentially to be excluded during a *Daubert* hearing. For instance, techniques such as the *Rey 15-Item Memory Test* or dot-counting techniques and other older methods are inappropriate for a modern assessment of cognitive distortion.<sup>40</sup>

In a forensic evaluation of traumatic brain injury, the neuropsychiatric examiner should understand the weaknesses of neuropsychological assessment and potential attacks upon its uses at court. Some of those attacks include the lack of uniformity in neuropsychological testing instruments used by practitioners (see [Chapter 6](#)). The psychologist may have deviated from standardized administration protocols (the physician examiner has a professional duty to insure that the psychologist agent does not deviate in a forensic situation where the physician relies on psychological measurements). Attacks may be directed toward the credentials of the psychologist, and therefore, the physician examiner should ensure that the psychologist providing psychological information to complete the neuropsychiatric assessment is well qualified in the administration and interpretation of neuropsychological tests used for detection of traumatic brain injury. The famous attacks of Faust and others on psychological testing are well known in the legal community.<sup>41</sup> Those attacks are somewhat outdated at this time, as the neuropsychology profession has markedly improved its standards, but the physician should expect attacks purporting to demonstrate that there is a paucity of well-controlled research on the relationship between clinical experience and diagnostic accuracy. Further attacks may be expected upon the ecological validity of neuropsychological assessment (see [Chapter 6](#)). This includes the assertion that there is a lack of data supporting the predictive validity of neuropsychological test results with regard to everyday domestic, educational, and vocational capabilities.<sup>42</sup> Golden has argued that 90% of the tests used in the field of neuropsychology lack validity.<sup>43</sup> That argument is no longer valid since most tests being published at the time of this writing do meet those basic standards of validity. Attorneys have become increasingly sophisticated in their knowledge base of neuropsychological tests. They may seek to exclude the use of neuropsychological testing by attacks upon the validity and reliability of neuropsychological tests, and thereby the conclusions and opinions based on these tests. Thus, the neuropsychiatric examiner should provide a comprehensive evaluation wherein neuropsychological testing is only

one of many components. This has been stressed previously in the clinical sections of this text (see [Chapters 1 to 8](#)).

The use of neuropsychological testing within a neuropsychiatric examination should include at a minimum the following:<sup>44</sup>

1. The full range of neuropsychological functions dependent on brain activity should be evaluated, that is, measurement of the major domains of neuropsychological function as outlined in [Chapter 6](#).
2. Testing that relates to the brain generally, as well as testing that relates to specific areas of cerebral cortical function, should be included.
3. The contemporary standards and methods used in clinical neuropsychology and psychology should be followed closely.
4. Each test used in the evaluation of traumatic brain injury should be sensitive to cerebral damage.
5. The testing used for evaluation should provide a balance so that both cerebral hemispheres are equally represented within the neuropsychological assessment.

The reader is referred to [Chapter 6](#) for the specific tests that may assist in the detection of cognitive malingering. These include instruments such as the *Test of Memory Malingering*, the *Portland Digit Recognition Test*, the *Victoria Symptom Validity Test (VSVT)*, and the *Letter Memory Test*. The forensic examiner should understand that a person with actual traumatic brain injury is expected to perform well within certain domains or upon certain psychological tests, whereas others should demonstrate deficits consistent with the locus and manner of the brain injury. Numerous other symptom validity tests may be utilized to detect test performance that is so poor that it is below the level of chance or random probability. Legal standards for expert testimony in regard to forensic neuropsychological personal injury evaluations have been published recently as well as recent reviews supporting test sensitivity and validity.<sup>45</sup> However, the neuropsychiatric examiner is cautioned to further determine if the symptom validity testing used by the psychologist providing consultation to the physician has been standardized upon a traumatic brain injury population. Not all symptom validity tests have been so standardized. Many examiners fail to understand whether individuals with head injury are capable also of faking their level of disability. In fact, head injury patients are as capable of faking memory deficits on a test such as the Portland Digit Recognition Test as their nonclinical counterparts.<sup>46</sup> Therefore, it is recommended that at a minimum, three tests of cognitive effort be administered in a forensic brain injury evaluation.<sup>48</sup> If the examinee fails one test and passes a second, the third test can be used as a “tiebreaker.” Symptom validity testing during examination for traumatic brain injury is under increasingly rigorous standards for admissibility in court.<sup>47</sup> Thus, to satisfy *Daubert* and other standards, the neuropsychiatric examiner should ensure that the symptom validity testing being used is standardized upon known brain injury populations, includes known error rates, and meets standards of contemporary neuropsychological assessment.

Financial incentives clearly may play a role in the induction of symptomatology in persons presenting for a forensic neuropsychiatric examination of traumatic brain injury. This is more likely to occur if the head trauma is mild rather than moderate or severe. Data have revealed more abnormality and disability in patients with financial incentives, despite less severe head injuries, than in controls. When evaluating patients after closed-head injury, particularly those with mild head trauma, the examiner should carefully include consideration of the effect of financial incentives on symptoms and disability.<sup>49</sup> A very recent study examined demographic, injury-related, and symptom variables at intake, 3 months postinjury, and 12 months postinjury and compared 50 treated adults with traumatic brain injury who were not seeking or receiving financial compensation against 18 litigants who were seeking compensation. The compensation seekers reported symptom incidence and severity at approximately 1 standard deviation (SD) higher at each of the testing intervals. The level of difference between the groups did not significantly differ over the 12-month



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**TABLE 9.3**  
**Principles of Cognitive Malingering Detection**

- At least three probability-based cognitive effort tests should be used during cognitive assessment.
  - Multiple sources of information and data should be gathered in malingering analysis.
  - These include historical indicators (prior history of deception), marked discrepancy between claimed and measurable cognitive deficits, interference with the examination, memory disorder or amnesic claims that deviate from Ribot's law, and failure of performance during cognitive effort measures.
- 

period. No demographic variables distinguished the groups. No injury-related variable, other than more immediate postinjury prescription medication use, was predictive of the greater symptom complaints for the patients seeking or receiving compensation. However, medication effects did not explain away the compensation effect when medication use was covaried in the analysis. Even treatment rated highly by patients is not adequate to wash out the strong relationship between financial compensation status and symptom report after mild traumatic brain injury.<sup>50</sup> Thus, the neuropsychiatric examiner evaluating a person who has been in psychiatric treatment following brain injury for a year should be aware that adequate treatment alone will not remove financial bias. It is probably impossible for persons, where financial reward is a gain, to be entirely unbiased when they are involved in litigation. On the other hand, even people in litigation who have valid traumatic brain injuries should demonstrate true cognitive and psychological deficits if adequate controls are in place within the examination. If the forensic neuropsychiatric examiner follows the *Daubert* rules and provides a comprehensive neuropsychiatric assessment, in most instances, the examination will uncover impression management, symptom magnification, or malingering.<sup>51–56</sup> [Table 9.3](#) outlines the strategy for the detection of cognitive malingering within a neuropsychiatric assessment of traumatic brain injury. Recent symptom validity tests have been validated as accurate in detecting malingered brain injury complaints.<sup>57–59</sup>

### **Detecting False Memory Complaints**

The forensic evaluation of memory complaints has come under increased scrutiny because of contamination from criminal sex abuse cases.<sup>60</sup> Particularly, the alleged ability to “recover” lost memories from traumatic events has been a major issue for the legal and scientific community. While sex abuse cases are not relevant to the issue of traumatic brain injury, neuropsychiatric examiners may, at times, be required to examine victims of head trauma sustained during sex abuse. However, while memory research reveals that clear distinctions exist between traumatically induced and ordinary memory, as people develop a narrative about what has happened to them, these narratives tend to coexist within the sensations of reliving the experience. These sensory components are highly state dependent and cannot be evoked at will. Narrative descriptions are gleaned from explicit or declarative memory stores (see [Chapters 2 and 6](#)) and are semantic and symbolic. They can be adapted to the needs of the narrator and the listener, and they can be expanded or contracted, according to social demands. At the present time, memory research is unable to determine whether sensory perceptions reported by traumatized subjects are accurate representations of the sensory inputs at the time of the trauma. Even with this limitation, the neuropsychiatric examiner can usually determine if there is injury to specific memory systems within the brain as a result of traumatic brain injury. That is an issue very distinct and apart from whether memory reported during a traumatic event is accurate. If the forensic examiner is to provide testimony in a criminal situation regarding issues of the believability of traumatic memories, it must be remembered that while trauma may leave indelible sensory and affective imprints, once these are incorporated into a personal narrative, this semantic memory, like all explicit memory, is likely to be subject to varying degrees of distortion.<sup>60</sup> Cautions with regard to children are even more significant. Virtually no data are available on what predicts

whether a traumatic experience in childhood is stored and manifested primarily as a behavioral memory, a narrative memory, or both. It remains unclear why an explicit, behavioral memory for a childhood trauma may predominate over a verbal memory in an older child or an adult. Selective amnesia for bodily injury does occur in some traumatized children exposed to a single-incident trauma over a relatively short retention interval. The examiner should remember that the specificity of a behavioral reenactment of a trauma in a child is not necessarily an indicator of its accuracy. Children's narrative memories for trauma vary in their degrees of completeness and accuracy. There are exceptions, but most studies demonstrate that a child's verbal memory for a real traumatic experience is generally accurate, although the details may be distorted.<sup>61</sup> However, the same memory principles that apply to the adult also apply to the child. That is, while the content of memories of traumatic events may become distorted, the neuropsychiatric examiner should be able to determine if there is evidence of damage to specific structural and functional memory systems in the brain through a comprehensive neuropsychiatric assessment. The results of that assessment may not allow the examiner to determine the accuracy of memories for the trauma itself.

To detect malingered memory deficits, the examiner first should have performed cognitive effort tests contemporaneous with the neuropsychological examination to determine the level of effort of the examinee. This includes use of tests such as described in [Chapter 6](#), or other appropriate standardized tests validated and found to be reliable in traumatically brain-injured persons. It is also possible within the test instruments used for the clinical detection of traumatic brain injury to determine inconsistencies of response that are biased toward presenting a false memory impairment. As noted previously in this text, violations of Ribot's law are the clearest clinical and historical indicator that the examinee is faking a memory disorder. Memories are lost in reverse order of their acquisition. If an examinee states that she cannot remember the births of her children but yet she can remember how she got to the examiner's office, this should immediately raise suspicion of memory faking. Moreover, if non-brain-injured persons are asked how they would mangle a memory deficit, they report they would show poor cooperation, aggravation, and frustration while slowing their response times and producing frequent hesitations. They also reportedly would demonstrate general confusion during the testing process.<sup>62</sup> Specific clinical neuropsychological tests commonly used for the evaluation of memory can be used within the brain injury evaluation to determine response bias toward faking a memory disorder. The *Colorado Priming Test* may be useful in identifying patients feigning memory impairment.<sup>63</sup> The *Rey Complex Figure and Recognition Trial* may assist in the detection of malingering. Malingerers will produce storage and attention memory error patterns during testing, whereas these patterns do not appear in persons with actual mild brain injury who have adequate motivation.<sup>64</sup> The *California Verbal Learning Test* may add useful data as a symptom validity test of memory during a brain injury assessment.<sup>65</sup> *Sentence Repetition* and the *Rey Auditory Verbal Learning Test-Recognition Task* have shown usefulness in detecting poor effort or conscious faking of memory disorders.<sup>66</sup> During examination of brain-injured litigants, one would expect, even in the brain-injured person, to develop a memory learning curve. The retained elements may be low, but a memory learning curve should be present regardless. The *Sternberg Recognition Memory Test* has been used to demonstrate improvement in lack of recall memory during a 2-year period following traumatic brain injury.<sup>67</sup> Even electrophysiologic studies have been applied to the detection of faked amnesia. Event-related brain potentials have been used in a research paradigm to discriminate simulated autobiographical amnesia related to head injury from control subjects. P300-evoked potentials produced a 92% correct discrimination of simulating individual subjects for birth dates and phone numbers. The same criterion applied to a single birth date yielded a 93% correct discrimination.<sup>68</sup>

## **Detecting False Executive Function Complaints**

We have seen in this text previously that the most common location of traumatic brain injury following a closed-head trauma is the frontal brain systems. This in turn often produces a disorder

of executive function (see [Chapters 2, 4, 5, and 6](#)). In a forensic situation, executive function disorders can present symptomatology that is impossible for the uninitiated to believe. Legal professionals may react with incredulity when confronted with certain executive dysfunctions and malingering may be suspected. In the “alien hand syndrome,” due to a lesion in the isolated lateral premotor system within the damaged contralateral hemisphere, persons may “talk to their hand” to induce compliance, or the hand may have to be peeled away from objects or persons once it has taken a grasp. The Capgras syndrome (reduplication phenomenon) associated sometimes with frontal lobe injuries may produce neurological symptoms that are not believable. The afflicted individual may believe that his spouse has been replaced by another person. The frontal-lobe-injured patient may be aware of what is wrong and yet be unable to utilize knowledge.<sup>69</sup> Therefore, it is extremely important for the forensic neuropsychiatric examiner to determine that cognitive effort was measured extensively within the context of a brain injury examination to ensure that frontal lobe syndromes are not inappropriately perceived as malingering. On the other hand, since executive disorders can be faked easily by coached persons, it is likewise equally important to detect malingering if present. Tests that assess deliberate distortion and deception must be administered to provide an adequate neuropsychiatric assessment.<sup>107</sup>

As noted earlier under the memory section of this chapter, in addition to standardized tests of cognitive effort, tests intrinsic to the measurement of executive function can themselves be used in the detection of malingering. For instance, the number of categories completed and failure to maintain set will usually distinguish malingers from controls who are administered the *Wisconsin Card Sorting Test (WCST)*.<sup>70</sup> High false positive error rates are usually observed in most samples when comparing normal college students asked to fake with neurological patients used as controls.<sup>71</sup> Thus, the Wisconsin Card Sorting Test can be used as an alternative estimator of malingering in addition to the cognitive effort tests such as the Test of Memory Malingering (TOMM), VSVT, and other cognitive effort measures noted in [Chapter 6](#). When administering the *Trail-Making Tests*, performance errors and inflated time scores may be useful in the assessment of malingering.<sup>72</sup>

### **Detecting False Motor Function Complaints**

Faking a motor disorder within the context of a traumatic brain injury generally occurs as a faked hemiparesis, but a conversion disorder must also be considered. Distinguishing the two, of course, is important, and neurological consultation may be required if the neuropsychiatric examiner lacks sufficient neurological skill to complete this part of the assessment.<sup>73</sup> The classic conversion V pattern on MMPI-2 scales 1, 2, and 3, found in the absence of focal neurological deficits, should help the examiner distinguish conversion disorder from faked hemiparesis. However, a V pattern can occur due to actual central nervous system disease or pain.

Use of the common motor tests within a standard neuropsychological profile may not accurately distinguish fakers from nonfakers. The *Grip Strength*, *Finger Tapping*, and the *Grooved Pegboard Tests* are so sensitive to effort that their ability to differentiate malingered from nonmalingered performers is suspect.<sup>74</sup> If the person being examined does, in fact, have a traumatic brain injury associated with an upper motor neuron lesion (see [Chapter 4](#)), then motor measures may separate the central lesion from a nonphysiological pattern in persons faking a motor disorder.<sup>75</sup>

### **Detecting False Visuospatial Function Complaints**

Little exists in the medical or neuropsychological literature regarding visuospatial malingering. Probably the best way to detect this is within the overall context of the neuropsychological evaluation wherein the examiner should evaluate for inconsistencies across tests measuring visuospatial performance. In particular, the *Wechsler Adult Intelligence Scale-III (WAIS-III)* and its visuospatial tests may be compared against the *Judgment of Line Orientation Test*, *Trail-Making Test Part B*, or other tests with visuospatial components. A single test capable of detecting visu-

ospatial malingering is not currently available. The Judgment of Line Orientation Test was evaluated as a single measure of malingering and was compared against the *Computerized Assessment of Response Bias* (CARB) and the *Word Memory Test* (WMT). The Judgment of Line Orientation Test has limited utility as a single screen for biased visuospatial responding.<sup>76</sup>

### **Detecting False Sensory Function Complaints**

The best determination of sensory malingering will come from the physical neurological examination. Sensory function peripherally should follow characteristic dermatome patterns, whereas central sensory function will occupy a nondermatomal pattern and in many instances will demonstrate extinction upon double simultaneous stimulation (see [Chapter 4](#)). There is one sensory examination that offers significant utility in a forensic neuropsychiatric examination of brain injury. The *Smell Identification Test* (SIT) is a forced-choice testing for faked sensory deficit of smell. Anosmia is a frequent component of infraorbital frontal lobe injury (see [Chapters 2](#) and [4](#)). The SIT was developed at the University of Pennsylvania, and it is a 40-item test.<sup>77,78</sup> It provides a quantitative measure of smell function in less than 15 min administration time. It is useful when the examinee is suspected of malingering in regard to sense of smell. In this test, four choices of smells are presented upon release of an odorant, yielding a 25% chance of accuracy in correctly identifying the designated smell, even if the subject guesses while having total anosmia (10 of 40 chances). In studies of this test, most nonfaking patients will correctly identify 35 or more of the 40 odorants. Females usually outscore males at all age levels. In a test of faking, 0 is the modal number of correct guesses for 158 men and women instructed to fake bad. The probability of obtaining a score of 0 by chance is 1 in 100,000.<sup>31</sup> The chance of obtaining 5 or fewer correct responses on the SIT is less than 5 in 100. Those with true anosmia generally demonstrate a guessing response of around 10 correct responses, which is at a chance level due essentially to random responding. Patients who have partial loss of smell will demonstrate an intermediate SIT score. Patients with multiple sclerosis yield scores slightly above average, and patients with Parkinsonism or Alzheimer's disease produce scores that are significantly lower than average, but they are still substantially above the expected range for random responding.

### **Using IQ Tests to Detect Poor Effort**

Numerous tests are available to predict obtained Wechsler Adult Intelligence Scale scores. This includes the *Wide Range Achievement Test*, *National Adult Reading Test*, and the *Wechsler Adult Test of Reading* (WTAR) (see [Chapter 6](#)). Studies have been completed to determine if internal consistency on an IQ test predicts insufficient effort or if a discrepancy between predicted and obtained IQ scores discriminates between traumatic brain injury and insufficient effort. One study<sup>79</sup> measured whether the Vocabulary–Digit Span difference score and a discriminate function based on subtests of the *Wechsler Adult Intelligence Scale-Revised* (WAIS-R) could differentiate patients with moderate and severe traumatic brain injuries from persons with financially compensable mild head injuries who were giving incomplete effort. The discriminate function analysis and Vocabulary–Digit Span difference score accurately classified 90% of moderate traumatic brain injuries and 79% of severe traumatic brain injuries from mild brain injury victims giving poor effort. This reference contains WAIS-R algorithms useful in making those distinctions.<sup>79</sup> Whether these algorithms apply to the current WAIS-III is not clear, and caution in such an application is recommended. Another study examined how well the discrepancy between predicted and obtained WAIS-R scores discriminates between insufficient effort and traumatic brain injury. In this study, 27 patients providing insufficient effort performed significantly poorer on the WAIS-R than 48 traumatically brain-injured patients with moderate to severe injury. Premorbid IQ estimates were calculated using the *Barona Index* or the *Oklahoma Premorbid Intelligence Estimation*. Those patients producing poor effort demonstrated a greater difference between predicted IQ and obtained IQ than those

patients with moderate or severe brain injury. As noted earlier in this text, one does not expect a substantial negative impact upon full-scale IQ by virtue of traumatic brain injury. There are, of course, exceptions to this rule.

## **DETECTION OF PSYCHOLOGICAL MALINGERING**

Virtually any alteration of central nervous system activity can affect a person's personality.<sup>102</sup> When the examinee is a litigant, changes in personality may be a damage issue in the lawsuit. While this text is focusing upon traumatic brain injury due to trauma, the same rules of malingering assessment apply to potential brain injury from solvent exposure, neurodegenerative dementias, or other brain diseases. As discussed previously in this text, personality change can be a direct outcome of traumatic brain injury or can be a reaction to the debilitating effects of traumatic brain injury. Again, in light of potential *Daubert* challenges, the examiner must be able to answer questions of base rates of various neuropsychiatric syndromes following traumatic brain injury and base rates of various neurological deficits following traumatic brain injury. Where those are available, they have been reported previously in this text. As with neuropsychological testing, personality testing should be used to compare obtained data to normative standards, base rates, and the examinee's baseline levels of functioning. Neuropsychiatric examiners may fail to check their own interpretations of data against the available standards from testing they ordered. This can prove problematic and is subject to discovery during cross-examination. Clinical experience, by itself, does not allow the formation of an opinion based on reasonable medical probability. To make such statements at court, the examiner must rely on additional standards of comparison.<sup>80</sup>

The decision-making process of the examiner may be much more accurate when addressing cognitive deficits than psychological deficits. Research on brain-behavioral relationships shows accuracy rates well above 80% for determination of cognitive dysfunction when using such instruments as the *Luria-Nebraska Neuropsychological Battery* or *Halstead-Reitan Battery*, and when comparing one battery against another.<sup>81-83</sup> However, when noncognitive psychological effects are considered, interpretations regarding brain-behavior relationships are much more complicated and more subject to error if based on face-to-face examination alone. Substantial caution on the part of the neuropsychiatric examiner is required to determine whether issues of malingering are present within the context of psychological claims following traumatic brain injury. Within a forensic neuropsychiatric examination, claims of depression, agitation, aggression, anxiety, and other psychiatric disorders cannot go unchallenged without concurrent assessment of psychological validity at the time of the neuropsychiatric examination. However, Berry and others argue that it is vital that examiners generate an estimate in their practice of the base rate of malingering.<sup>106</sup> If it is not logistically possible to establish local base rates, the examiner may consider published rates in similar forensic settings. The practical significance of this point is that use of a malingering scale in a setting with a very low base rate of the target condition (malingering) may falsely label honest people as malingerers. Use of multiple validity measures may decrease this risk.

### **The MMPI-2 in Detection of Psychological Malingering**

In most instances, the neuropsychiatric examiner will not possess a license in psychology, and therefore, consultation with a psychologist or neuropsychologist is required. However, the same caveats apply in psychological assessment as they do in neuropsychological assessment. It is incumbent upon the neuropsychiatric examiner to determine if the consulting psychologist or neuropsychologist is appropriately skilled in the interpretation of the MMPI-2. In general, individuals who possess one course in graduate school regarding the MMPI-2 and subsequent limited experience in the utilization of this test instrument are poor choices for consultation in neuropsychiatric assessment. As with medical procedures, the heart surgeon performing 200 to 300 procedures a year is generally more competent than one performing 25 procedures annually. The same is true

of the psychologist. Therefore, the neuropsychiatric examiner may want to determine the base rate of MMPI interpretations performed by the psychologist in any given year. This will help the neuropsychiatric examiner determine, at least in part, the experience base of the psychologist. Moreover, in a forensic setting, the use of computerized interpretive templates for the MMPI-2 is not recommended. The most credible use of the MMPI-2 in court rests upon careful individual interpretation by the psychology examiner who also examined the injury claimant at the request of the neuropsychiatric examiner. A case in point lies in substantial elevations on scale 8 of the MMPI-2, which is often an outcome of significant traumatic brain injury. A computerized interpretation of the MMPI-2 generally will default to a diagnosis of schizophrenia or some schizophrenia-like process. In fact, the MMPI-2 is responding to the disordered thinking being reported by the examinee as a result of impaired attentional and frontal systems following traumatic brain injury. Clinical psychologists who are inexperienced in traumatic brain injury may overlook this point, but if the examining physician testifies in court about the MMPI-2 and the finding of “schizophrenia,” this in all likelihood discredits the examiner and causes significant complications to the production of accurate medical testimony regarding the behavioral effects of traumatic brain injury.

### *Cannot Say Score*

The use of this scale determines how many questions the examinee failed to answer during MMPI administration. It is generally accepted that if more than 30 test questions are not answered, this will attenuate the profile. Most psychology experts believe the MMPI profile should not be interpreted if 30 or more items are omitted. Therefore, it is incumbent upon the psychology examiner to ensure that post-test-taking interviews are performed to answer questions the examinee may have regarding omitted items so that they can be completed at the time of the examination. Moreover, it is extremely important that the neuropsychiatric examiner insure that the examinee completed the MMPI-2 uninterrupted by interference from others. If the examinee reads poorly, the University of Minnesota Press provides auditory tapes that may be used for the administration of the MMPI-2. In almost no instance should an MMPI-2 be read to the examinee by a second party. This will usually invalidate the protocol for forensic purposes. If English is a second or third language, the MMPI-2 may require deletion from the test protocol unless appropriate other language forms are used. The examinee may be loathe to answer certain questions in the face of a second party. The reading requirements and other testing standards for the MMPI are discussed further in [Chapter 7](#).

If asked, the neuropsychiatric examiner should be aware that possible reasons for omitting items during administration of the MMPI include poor cooperation, defensiveness, indecisiveness, fatigue, depression, carelessness, poor reading comprehension, and the perception of examinees that the items are not relevant to them. It is recommended that no items be omitted, as even five or six omitted items, on a particular scale, can affect the reliability and validity of that scale.<sup>36</sup> On the other hand, if most of the omitted items occur toward the end of the booklet (after item 370 on the MMPI-2 or after item 350 on the adolescent version [MMPI-A]), some validity and all clinical scales can be interpreted. The newer validity scales, such as VRIN, cannot be used in this instance. Also, the forensic examiner cannot use data from the supplementary scale or content scales, which are included in items found toward the end of the test question booklet.

### *The Lie (L) Scale*

If the examinee elevates the L scale (> 65T), the examinee is probably involved in impression management of the testing situation. However, the L scale cannot measure lying per se. It is not a truth detector. Elevations on the L scale suggest the examinee has responded to other items in the MMPI-2 in such a manner as to deny personal weakness and present the most favorable image to the examiner. Elevation on the L scale may not be as important in the neuropsychiatric examination of traumatic brain injury as it would be in a custody evaluation or a criminal evaluation. However, if the score exceeds 72T, this suggests clear distortion of item responding on the L scale in order to manipulate what the examiner thinks of the examinee. If the examinee is attempting to create a

particular pattern of disability (e.g., brain injury), elevations of the L scale associated with other elevated MMPI-2 scores are consistent with that interpretation. Review of the TRIN scale (inconsistent true or false responding) can aid in determining whether the elevated L score is due to a frequent false response. Baer et al.<sup>84</sup> reviewed measures of underreporting psychopathology on the original MMPI. They did not find any substantial differences in the function of the L score on the MMPI-2 relative to the MMPI.

In addition to the above-mentioned reasons for elevation of the L scale, this scale may also be elevated in persons who are unrealistically proclaiming virtue. Other causes of elevation are hypermorality, a naïve self-view, an effort to deceive others about motives or adjustment, personality adjustment problems, and a lack of willingness to admit even minor flaws in one's personality or character (poor self-disclosure).

### *The K Scale*

Elevations on the K scale indicate the tendency to present a favorable self-report.<sup>36</sup> This scale can be influenced by one's socioeconomic class or educational level.<sup>85</sup> Elevations on the K scale greater than 65T suggest possible defensive responding. For instance, the individual is presenting a more favorable image than is practical. This is commonly seen in family custody evaluations or criminal proceedings and rarely in traumatic brain injury evaluations, but it may occur. Persons who are poorly educated below high school level tend to produce lower K scores than more educated persons. Causes for elevated K scores include defensiveness, a great need on the part of the examinee to present as very well adjusted, or overresponding falsely. False responding can be ruled out by examining the TRIN scale.

### *The Infrequency (F) Scale*

This is clearly the most important validity scale on the MMPI. While it cannot be interpreted in isolation, it has the greatest scientific database of all of the MMPI validity scales available to psychologists. It has been modified substantially in the MMPI-2 and MMPI-A relative to the original MMPI. Four original items were dropped from the scale because of their objectionable content. The new F scale was empirically normed using linear T-scores as opposed to the rationally derived setting of scale values with the original F scale development. An additional infrequency scale has been added, the back-page F scale, or Fb. This was developed to rule out a measure of infrequency for the items that appear in the back of the booklet (after item 370) because the original F scale contains only items that occur in the front half of the item booklet.<sup>36</sup> The F scale for the MMPI-A was further revised to address more fully the tendency of adolescents to endorse items differently than adults.<sup>86</sup>

Concern over elevations on the F scale generally is not present until the scale exceeds 80T. A score between 60T and 79T usually reflects that the examinee is approaching the items in a problem-oriented fashion. If the F scale exceeds 80T, this indicates an exaggerated response set and probably represents an attempt to claim excessive problems. This would be consistent with symptom magnification. The examiner can review the VRIN T-score, and if it is below 79, this rules out the probability of inconsistent responding. If the F scale T-score ranges from 90T to 109T, the profile may be invalid. If the F score exceeds 110T, this is an uninterpretable profile and consistent with extreme item endorsement. Where the F scale is elevated, possible causes include confusion, illiteracy, responding to the items in a random fashion, severe mental illness, symptom exaggeration, faking psychological problems, and malingering.<sup>87</sup>

A new F scale was added to the current edition of the MMPI-A and was specifically developed for persons between the ages of 14 and 18. The F1 scale functions similar to the F scale on the adult version of the MMPI-2. The F2 scale corresponds to the Fb on the adult form of the MMPI-2. In other words, F2 allows one to assess responding toward the end of the booklet vs. toward the front of the booklet with the adolescent test in the same fashion as is accomplished with the adult MMPI-2. Berry and others have found that the F and Fb scales of the MMPI-2 will significantly

differentiate patients seeking compensation for head injuries from closed-head injury patients not seeking compensation, in terms of greater scale elevation in the former.<sup>88</sup>

### *The VRIN Scale*

This new scale was added to the MMPI-2 and the MMPI-A during their development. VRIN does not exist in the original edition of the MMPI. It is a very useful scale for determining careless, inconsistent, or random responding. Its most important interpretation is made in combination with the F scale. The VRIN scale is an empirically derived measure. Throughout the MMPI-2 and MMPI-A, there are pairs of items for which some responses are semantically inconsistent. As noted above, very high elevations on the F scale are the most sensitive indicators of psychological malingering. The VRIN scale enables the psychologist to determine if extreme elevations on the F scale are in fact malingering or due to other complications within the examinee's approach to the MMPI-2 or MMPI-A. If the individual has a high score on the F scale and a low to moderate score on the VRIN scale, reasons other than randomly responding or inconsistent responding must be considered to explain the high F score. A high F score in these cases, with a relatively reduced VRIN score, may represent actual or faked psychopathology. If the F scale score is extremely elevated and the VRIN scale score also is extremely elevated, these findings are consistent with confusion, random responding, or inconsistency in the examinee's approach to the testing. If the VRIN T-score exceeds 80T on the MMPI-2 or exceeds 75T on the MMPI-A, this indicates inconsistent random responding that invalidates the MMPI profiles. If the VRIN scores range from 70T to 79T on the MMPI-2 and from 70T to 74T on the MMPI-A, this suggests a possibly invalid profile due to inconsistent responding.<sup>87</sup>

### *The TRIN Scale*

This scale was designed to measure the tendency for some individuals to respond in an inconsistent manner by endorsing many items in the same direction (either true or false).<sup>36</sup> Scoring for the TRIN scale is very complicated. Most MMPI experts feel that TRIN should be computer scored rather than hand scored to reduce scoring errors. The TRIN score is particularly useful when one interprets scores on scales L and K because all but one of the items on these two scales is keyed "false." Thus, if a person engages in an inconsistent "false" response set, this may produce elevated scores on scales L and K that have nothing to do with being defensive or with an attempt to fake good. Conversely, an individual who answers the MMPI-2 items inconsistently "true" may produce very low scores on L and K that have nothing to do with being excessively open, self-critical, or overwhelmed by stress. Thus, whenever extreme scores appear on scales L and K, the psychologist should make a careful examination of the score on TRIN. Pope et al. believe this is essential if L and K scores are in the extreme ranges.<sup>36</sup> As with VRIN, TRIN scores greater than 80T on the MMPI-2 or greater than 75T on the MMPI-A indicate inconsistent responding and probably invalidate the test protocol. In like fashion, respective scores 70T to 79T on the MMPI-2 or 70T to 74T on the MMPI-A suggest possible inconsistent responding,

Pope et al.<sup>36</sup> have described four major ways that the validity scales of MMPI-2 may raise the issue of malingering. These faking responses are: (1) rare responding, (2) defensive responding, (3) inconsistent responding, and (4) atypical MMPI-2 patterns. The F scale is the most sensitive scale in the MMPI for detecting rare responding. Defensive responding is a common indicator of complaints without actual organic problems.<sup>89</sup> Defensive individuals often respond to the test items on the MMPI-2 by claiming a high degree of virtue and denying or minimizing faults. This response set will produce high L and K scores so that their claims of physical problems will seem more credible. It is an attempt to manipulate what the examiner thinks of the person or to create a particular pattern of disability within the context of the neuropsychiatric examination. Rogers has noted<sup>90</sup> that inconsistent responding on the MMPI test items can reflect a general pattern of malingering. Some individuals will attempt to endorse extreme symptoms in an unselected fashion and endorse randomly, or respond in an "all true" or "all false" response set. The VRIN and TRIN scales on the MMPI-2 will help detect these types of malingering. The fourth possible indicator



of malingering, atypical MMPI-2 patterns, requires consultation with a psychologist very experienced and educated in the use of the MMPI-2. This is the most difficult pattern to analyze. To detect malingering, the psychologist must match behavior or symptoms from the examinee's responses to that of expected clinical patterns established by research on the particular sample involved or by the base rates for the relevant population.<sup>91</sup> There are modal or expected MMPI-2 performances that can be identified for a variety of clinical situations or phenomena, and those are published in the major texts on interpretation of the MMPI-2 (see references in [Chapter 7](#)).

### *The Fp Scale*

The F and Fb scales are effective at detecting extreme item endorsement on the MMPI-2. However, in the more moderate ranges, they are not useful for differentiating symptom exaggeration or malingering from a person who is actually quite seriously mentally ill. The examiner must understand that mentally ill or psychologically disturbed persons can elevate scores on F and Fb even though they are not exaggerating or malingering. After the original publication of the MMPI-2 in 1989, Arbisi and Ben-Porath<sup>92,93</sup> developed the scale now called Fp to help with that differentiation. If the T-score on Fp ranges from 60T to 79T, this generally reflects a problem-oriented approach to the items and the person may have true psychopathology. Scores ranging from 80T to 89T indicate an extremely exaggerated response set in which the examinee is attempting to claim extreme or unusual psychiatric symptoms. If the T-score elevates to 90T to 109T, the profile may be invalid as a result of the examinee claiming an extreme number of rare psychiatric symptoms. In those instances where Fp is greater than 110T, this indicates likely malingering of psychiatric symptoms and it results in an uninterpretable profile because of the extreme item endorsements. Elevated Fp scores (greater than 90T) usually represent symptom exaggeration, faking psychological problems, or malingering psychiatric illnesses.<sup>87</sup>

### *The F-K Index*

This dissimulation index was first proposed by Gough in 1947.<sup>94</sup> However, Gough set the cutoff at 9. In other words, he advised taking the raw score on F and subtracting the raw score from K. If that score exceeded +8 (+9 or greater), the examinee was considered to have faked the MMPI-2. Later research suggested that this index was too low, and it has been recommended that profiles with an F-K score of +12 or above be considered invalid.<sup>95</sup> The F-K index has been supported by various studies in forensic assessment that have demonstrated it will accurately detect malingering.<sup>96-98</sup> However, it is not as effective as the T-score of the F scale taken alone.<sup>99</sup>

There are numerous other scales that have been reported to be of assistance in the detection of malingering or dissimulation during psychological, psychiatric, and neuropsychiatric examinations. These include the Fake Bad Scale (FBS), the Superlative Self-Presentation Scale (S), and others that are outside the scope of this text. The reader is referred to Pope and others for more extensive data on the use of the MMPI-2 or the MMPI-A in court.<sup>36</sup> The reader can also refer to Nelson<sup>80</sup> for use of the MMPI-2 in forensic neuropsychological evaluation. [Table 9.4](#) lists methods whereby the MMPI-2 may assist in the detection of deception.

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**TABLE 9.4**  
**Using the MMPI-2 and MMPI-A to Detect Malingering**

- Consult with a psychologist expert in MMPI administration and interpretation.
  - Is there evidence of rare responding? Review MMPI profile to detect high F T-scores and high F-K raw scores. If they are elevated significantly, confirm that VRIN is also not elevated (confusion or random responding if VRIN elevated).
  - Is there evidence of defensive responding? Are L and K scores significantly elevated in order to make claims of impairment more credible? Determine from TRIN scores whether a “false” response set is present.
  - Is inconsistent responding present? Review VRIN and TRIN scores for “all true” or “all false” response sets.
  - Are atypical MMPI-2 or MMPI-A patterns present?
-

## **The Millon Clinical Multiaxial Inventory-2 in Detection of Psychological Malingering**

The *Millon Clinical Multiaxial Inventory-2* (MCMI-2) is not particularly useful in the forensic neuropsychiatric assessment of traumatic brain injury. That statement is not meant to discredit this inventory. It is very useful for personality assessment and for the assessment of persons in psychotherapy. However, the database for this test instrument in brain injury is slight and not nearly as strong as the MMPI-2, the *Personality Assessment Inventory* (PAI), or the *Structured Interview of Reported Symptoms* (SIRS).

There are four validity scales on the MCMI-2 specifically designed to assess exaggeration or minimizing of problems: (1) the validity index, (2) scale X (disclosure), (3) scale Y (desirability), and (4) scale Z (debasement). Bagby and others have independently examined all four MCMI-2 validity scales in distinguishing honest, fake bad, and fake good response sets by discriminate function analyses. Their results revealed that the MCMI was capable of not only classifying fake bad profiles accurately, but also identifying fake good and honest profiles with much accuracy. Apparently, the MCMI-2 demonstrates a slight tendency to identify faking bad profiles more accurately than honest or faking good profiles.<sup>100,101</sup>

The neuropsychiatric examiner should realize that the MCMI-2 is theory based rather than empirically based. In other words, it is based on theories of psychological functioning by Theodore Millon and is not based on empirically derived samples from mentally ill persons. This provides some potential weakness when using this instrument in forensic settings and, as a result, may be inconsistent with *Daubert* standards.

## **The Personality Assessment Inventory in Detection of Psychological Malingering**

Morey<sup>103</sup> describes random responding and malingering as negative distortion. As noted in [Chapter 7](#), the PAI, when contrasted with the MMPI-2, is much more clinically based. However, unlike the MMPI-2, there is no adolescent form for this test instrument and it is not normed on persons younger than 18 years. This test appears to be psychometrically superior to the MMPI-2.<sup>104</sup>

The PAI contains four major validity scales: (1) infrequency, (2) inconsistency, (3) negative impression, and (4) positive impression. The Infrequency (INF) Scale determines if the examinee is responding carelessly or randomly. The Inconsistency (ICN) Scale determines if the examinee is answering questions consistently throughout the task. The Negative Impression (NIM) Scale determines whether the examinee is psychologically exaggerating or malingering. The Positive Impression (PIM) Scale determines whether the examinee is trying to make a very favorable impression or is reluctant to admit to minor flaws.

### *Infrequency Scale*

This scale is used for the identification of examinees who complete the PAI in an atypical way due to carelessness, confusion, reading impairment, or other sources of random responding. The scale consists of items that were designed to be answered similarly by all examinees, regardless of their clinical status. Half of the items are expected to be answered “totally false,” whereas the other half should be answered “very true.” INF items are placed evenly throughout the PAI to identify potentially problematic responding. INF scale items have been written specifically to provide item content that would be infrequent, yet would not sound bizarre, for instance, “I have never seen a building.”

The INF scale is primarily a measure of carelessness in responding. However, examinees also could answer the PAI items in a very idiosyncratic way. If the psychologist makes a quick review of the INF items, it is easy to distinguish between these two potential sources of elevation. High scores on INF (= 75T) are consistent with the examinee attending inappropriately to item content

while responding to the items on the PAI. The completely random response will result in an average INF score of 86T. The neuropsychiatric examiner should be aware that several potential reasons for scores in this range may occur, such as reading impairment, random responding, confusion, errors in scoring the scale, or failure to follow the test instructions. Test results with INF scores in this range are assumed to be invalid and no clinical interpretation of the PAI is recommended.<sup>103</sup>

#### *Inconsistency Scale*

This is an empirically derived scale that reflects the consistency with which the examinee completed items with similar content. One commonly observed problem that can cause elevations on ICN is a failure to attend to item statements that contain the word *not*. There are few items with negative statements on the PAI, but these items are overrepresented on the ICN scale in order to specifically examine how such items are interpreted. If the examinee is paying attention poorly, an elevated scale should alert the interpreter that the examinee may not have been reading the items carefully when completing the inventory. High scores on ICN (= 73T) suggest that the examinee did not attend consistently or appropriately to item content in responding to the PAI items. If the examinee answers the questions in a completely random fashion, this generally results in an average ICN score of approximately 73T. The potential causes for scores in this range include carelessness, reading difficulties, confusion, errors in scoring, or failure to follow the test instructions. Regardless of the etiology, test results should be assumed to be invalid and no clinical interpretation of the PAI is recommended when ICN scores are 73T or higher.<sup>103</sup>

#### *Negative Impression Scale*

Morey indicates that the starting point in the detection of malingering while using the PAI is this scale.<sup>103</sup> The self-report of a high score on NIM is probably more pathological than an objective observer would report. However, patients with actual mental illness will score higher on this scale than individuals without mental illness due to negative perceptions that covary with the presence of some mental illnesses.

The NIM scale includes two types of items: some are presented as an exaggerated distorted impression of self, while others represent extremely bizarre and unlikely symptoms. Either of these tendencies may cause distortion of a self-report in a negative direction. Scores on the NIM scale lower than 73T indicate that there is little distortion in a negative direction on the clinical scales. Moreover, the examinee probably did not attempt to present a more negative impression than the clinical picture would warrant. Moderate elevations (73T to 84T) are consistent with an element of exaggeration of complaints and problems. Elevations on NIM in the range of 84T to 92T may be consistent with a “cry for help” or an extremely negative evaluation of self. This scale range also is consistent with a deliberate distortion of the clinical picture (symptom magnification or false attribution). High scores on NIM (= 92T) are consistent with examinees’ attempts to portray themselves in an especially negative manner. This score is consistent with careless responding, extremely negative self-presentation, or malingering. A completely random completion of the PAI would result in an average NIM score of 96T.

#### *Positive Impression Scale*

The content of PIM scale items includes the presentation of a very favorable impression or the denial of relatively minor faults. Morey advises that it should be recognized that the tendency for favorable self-presentation is fairly common in the normal population. PIM scores represent the level examinees are attempting to manage their clinical impression to the examiner in a positive direction. Thus, this scale roughly corresponds to the L and K scales of the MMPI and MMPI-2 in terms of measuring defensiveness or positive impression management strategies. Low scores on PIM below 44T are consistent with honest responding. Scores between 44T and 57T suggest the examinee did not attempt to present an unrealistically favorable impression. Moderate elevations ranging from 57T to 66T suggest that the examinee responded in a manner to portray a lack of common shortcomings. A PIM score of 66T is 2 standard deviations above the mean for clinical

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**TABLE 9.5**  
**Using the PAI to Detect Malingering**

- Consult with a psychologist expert in PAI administration and interpretation.
  - Review the INF scale. Is there evidence of careless or random responding?
  - Review the ICN scale. Is there evidence of inconsistent responding?
  - Review the NIM scale. Is there evidence the examinee is presenting a very negative image?
  - If the NIM scale exceeds or equals 92T, does the MAL scale indicate malingering?
- 

patients. Thus, scores of 66T are consistent with the examinee's attempts to present as exceptionally free of common shortcomings to which most individuals will admit. The validity of the entire PAI clinical scales is seriously questioned when PIM scores exceed 66T.<sup>103</sup>

#### *The PAI Malingering Index*

The PAI Malingering Index (MAL) is a specific indicator of malingering with higher specificity than NIM. It is comprised of eight configural features of the PAI profile. The reader should refer to the PAI manual or interpretive guide authored by Morey for further details and appropriate applications of this index.<sup>103</sup> However, simply put, MAL scores of 3 or above should raise questions of malingering, whereas scores of 5 or more are highly unusual in clinical samples, and they tend to occur only when mental disorder is being faked severely. [Table 9.5](#) gives guidelines for using the PAI validity scales in the detection of distortion and malingering.

#### **The Structured Interview of Reported Symptoms**

This test instrument was developed within the context of the extraordinary research of Richard Rogers, Ph.D., and his colleagues regarding malingering.<sup>105</sup> Rogers' model of malingering is based on three domains: pathogenic, criminological, and adaptational. Each model proposes a distinct primary motivation for malingering. Severe underlying pathology drives the pathogenic model. An antisocial noncompliant orientation drives the criminological model, and coping with adversarial circumstances such as being a prisoner of war drives the adaptational model. Interpretation of the SIRS is not dependent upon any one explanatory model. Rogers further evaluates malingering by four separate but integrated methods. The first method is to review the examinee for (1) endorsement of an unusually high number of rare symptoms, (2) endorsement of an unusually high number of blatant symptoms, (3) nonselective endorsement of symptoms that appear to be improbable based on the excessive number of them, and (4) endorsement of absurd and preposterous symptoms. Once the pattern of self-reported symptoms has been established, corroboration of faking is attempted by collateral interviews, examining for pronounced differences between reported prior episodes and the historical documentation, and unequivocal evidence of faking on standardized measures such as the MMPI-2. After the pattern of symptoms is established and corroborated by data gathering, then it should be determined that the examinee's motivation for faking is not based exclusively upon a desire to be a patient (factitious illness), nor is it an attention-getting device such as seen in borderline patients.<sup>105</sup>

The SIRS is a highly structured interview format based on detailed, general, and repeated inquiries. Because of its rather complex format, Rogers et al. note that it is imperative that interviewers be familiar with the structure of the SIRS and well practiced in its administration. Mechanics of SIRS administration differ greatly from many other assessment techniques used in psychology. The SIRS is able to classify the responses of examinees at two levels of classificatory certainty. Of the eight primary scales, those classified as "probable" accurately differentiate at least 75% of the criterion groups, and those classified as "definite" accurately classify 90% or more of individual subjects. Feigning is then classified based on single-scaled scores and multiple-scaled scores.

Some individuals cannot be classified as either feigners or “honest” responders. In such cases, the interpretation is a description of response styles and leads to “indeterminate” classification. It is very important to remember that the primary emphasis of the SIRS is on the classification of feigning. This test instrument is not designed to measure psychopathology or to be used as a stand-alone instrument to measure one’s mental state. This test instrument also stresses the important determination of those individuals who are honest responders. Moreover, users of this test instrument are cautioned that they must review other sources of clinical data (for example, the MMPI-2) or other collateral sources of information before the classification of malingering is made. Many experts in the field of psychology and psychiatry consider the SIRS to be the gold standard assessment instrument for the detection of psychological malingering when used within a comprehensive psychological evaluation. The examiner is again strongly cautioned that the SIRS is not a stand-alone instrument. It is best used when the issue of malingering has been raised by the clinical presentation of the examinee, by distortion of validity indices on standardized psychological test instruments, by historical information, or by the behavior of the examinee during the assessment.

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# 10 Causation, Damages, Outcome, and Impairment Determination Following Traumatic Brain Injury

## INTRODUCTION

When traumatic brain injury cases are brought to civil court, they are argued by the plaintiff as a tort. A tort is a private or civil wrong or injury produced by an actor or tortfeasor (the person responsible for causing the injury). By taking the claim of injury or harm to a court, the injured person asks the court to provide a remedy in the form of an action for damages. Damages are compensation (usually monies) that may be recovered in the courts by any person who has suffered a loss, detriment, or injury, in this case to his person.<sup>1</sup>

In this text, impairment follows the American Medical Association's *Guides to the Evaluation of Permanent Impairment* definition. This defines impairment as "a loss of use or derangement of any body part, organ system, or organ function." On the other hand, the term disability historically refers to a broad category of persons with diverse limitations in the ability to meet social or occupational demands. Several organizations are now moving away from the term disability and instead are referring to specific activity limitations to encourage an emphasis on the specific activities the individual can or cannot perform and to identify how the environment can be altered to enable the individual to perform the activities associated with various social or occupational roles.<sup>2</sup>

Competence is an issue that may arise following traumatic brain injury. The injury may so affect the individual that the person is then unable to act in a self-serving fashion. Competence is defined within the law of evidence as the presence of those characteristics or the absence of those disabilities that render a witness legally fit and qualified to give testimony in a court of justice; applied in the same sense, to documents or other written material presented as evidence. Competency differs from credibility. For instance, a witness may be competent and yet give incredible testimony; the person may be incompetent, and yet this person's evidence, if received, may be perfectly credible. Competency is an issue for a court (a judge); credibility is usually an issue for a jury.<sup>3</sup>

Competence may be general or specific. The question of a person's general competence may be raised when an allegation is made that the individual no longer has the capacity to make decisions about the entire range of his affairs. Specific competence may apply to a very focused matter, such as competence to consent to treatment, competence to make a contract, or competence to draft a living will.<sup>3</sup>

For the physician providing expert testimony in a brain injury case, causation and damages are issues for the lawyer. However, it is mandatory that the examining physician have a fundamental understanding of these legal issues in order to determine if the medical proof elements are present to enable the physician to provide expert testimony on causation or damages. With regard to

impairment and disability, these terms often are highly confused by physicians. Medical impairment is to be found by physicians, whereas disability is to be adjudicated by administrative legal bodies and courts. Competence is to be determined in most instances by a judge, but the medical and psychological underpinnings of competence may be provided by physicians or psychologists to courts.

## CAUSATION

Traumatic brain injury cases are usually brought to courts as a personal injury tort. The plaintiff brings the case and the accused mounts a defense. There must be some reasonable connection between the defendant's act or omission and the resulting brain injury to the plaintiff before liability will be found.<sup>4</sup> Numerous terms and rules are used to define causation, and these have been established by courts throughout the centuries. Some of the more common tests include the "but for" test, proximate cause or legal cause, intervening cause, cause and fact, *res ipsa loquitur*, and substantial factor-increasing risk of harm.<sup>5</sup>

The physician will often hear lawyers use a common term, *proximate cause*. This is defined as "that which, in a natural and continuous sequence, unbroken by any efficient intervening cause, produces injury, and without which the result would not have occurred."<sup>1</sup> In other words, the proximate cause of a brain injury is the primary or moving cause, that which, in a natural and continuous sequence, unbroken by any other intervening cause, produces trauma to the brain in question. Without the cause, the injury could not have occurred. In most instances of brain injury, the proximate cause is an accident resulting in trauma to the head that in a continuous and unbroken fashion produces a change in the organism (i.e., the brain).

Physicians do not need to concern themselves with complex legal points upon which causation turns. However, an examining physician hired to evaluate a person who may have sustained a traumatic brain injury by and large will be used by the hiring attorney as a causation expert for the plaintiff or, in the case of the defendant, to either refute causation or attempt to show that no injury occurred. Personal injury lawyers tend to follow a sequence to prove causation by using an expert who offers an opinion that the trauma caused by the defendant was a substantial or material contributing factor in producing the plaintiff's current symptoms.<sup>6</sup> An expert (such as a physician who examined the victim for traumatic brain injury) will be presented by either the plaintiff's or defendant's lawyer at trial to support or refute a claim for mental, emotional, or cognitive injuries, depending on the nature of the relationship between the accident and the injuries. If the relationship of the patient's cognitive and psychological state, subsequent to the brain injury, is not a matter of common knowledge likely to be possessed by the average judge or juror, expert testimony is required to avoid a directed verdict on the item of damages.<sup>7,8</sup> Traumatic brain injuries and their resulting psychological and cognitive sequelae are not likely to be understood by a typical judge or jury as necessarily following from physical trauma. Generally, the central issue in a traumatic brain injury trial is the degree of relationship between the physical injury and the organic brain injury. These cases typically turn on the requirement of reasonable medical certainty or reasonable medical probability. If an expert physician expresses uncertainty about the causal relationship between the physical injury and the organic brain injury, exclusion of the testimony and a directed verdict on the issue may result.<sup>9</sup>

An old common law test is the "but for" analysis. Stated another way: "But for the negligence of the defendant, the plaintiff would have suffered no harm." Or, from the perspective of the defendant, "A defendant's conduct is not a cause of the event if the event would have occurred without it."<sup>10</sup> Most legal experts admit that though the "but for" test is simple to use, it has no borders. Its parameters are too vague and unspecific, but there is a significant appeal for using this test with the layperson.<sup>5</sup> Many lawyers consider it inadequate, particularly in a traumatic brain injury case, because it is so overinclusive. The test also can be considered quite underinclusive if there is more than one defendant. By using the "but for" test, neither defendant could be held liable

if the injury would have occurred in any event through the negligence of the other. The two defendants would cancel each other out in this case.<sup>4</sup> However, these causation arguments are made for the edification of the physician only, and any analysis of these issues is best left to lawyers and judges skilled in civil and personal injury matters.

## DAMAGES

In most traumatic brain injury legal cases, it is the issue of damages wherein the neuropsychiatric examiner will play the greatest role at trial. In general, the plaintiff wants to prove maximal damages from the alleged traumatic brain injury, whereas the defendant wants to prove minimal or no damages as a result of the alleged traumatic brain injury. The physician expert is not an advocate, as discussed in [Chapter 9](#). However, the physician expert will garner no respect from the jury if she does not advocate for her own opinion. Obviously, if experts do not believe their opinions, that will be conveyed to the jury. Thus, advocating for one's opinion is not the same as advocacy for either the defendant or the plaintiff. Even if the lawyer for the plaintiff proves liability for the accident and causation of an injury, the monetary award will hinge upon damages.

For physicians evaluating brain injury, the major issue in damages is outcome. In other words, what are the medical outcomes of the traumatic brain injury that the plaintiff either does or does not possess? In general, plaintiffs' lawyers will attempt to prove damages at trial by using the treating doctor as an initial witness to document the causation of the brain injury. This is often the physician who completed the initial cognitive and behavioral examination. This individual then testifies on the causation of cognitive and behavioral damages. The lawyer may follow up with lay witnesses to prove issues such as impairment in daily functioning, the effect upon activities of daily living, and alteration of the individual's lifestyle.

When the neuropsychiatric examiner is hired to assess damages in a victim of traumatic brain injury, it is necessary to carefully explore for preexisting emotional conditions that may play a role in the current symptomatology expressed by the examinee or represent conditions that may have been aggravated or exacerbated by the traumatic brain injury, if it exists. When there have been preexisting conditions, determining what injuries a wrongdoer has caused presents a substantial problem to the examiner. The law generally dictates that victims are taken "as they are found." Thus, the mere fact that the examinee had either a prior injury or a prior mental disorder before the subject issue of brain injury is not a defense. If the tortfeasor has further harmed a person with a preexisting condition, that harm is compensable under the law. It is common when preexisting brain or mental disease arises for the plaintiff to argue that the present alleged brain injury was "the straw that broke the camel's back" or the commonly used "cracked-egg theory."<sup>6</sup> The critical question the neuropsychiatric examiner must attempt to answer is what the plaintiff was like before the defendant allegedly produced a brain injury. The neuropsychiatric examiner may need to assist the attorney with answers to two questions. Was the preexisting condition latent? Was the preexisting condition stable or degenerative? A trauma can trigger or activate a dormant condition.<sup>11</sup> Whether or not the condition was stable or degenerative is a major legal issue, since stability before the accident must be established or it can be argued that the present symptoms are merely the result of a preexisting progressive condition.<sup>12</sup>

If liability can be established, then damages can be awarded. Lawyers often argue that "liability provokes damages" or "damages provoke liability." To win at trial, an alleged damaged party must demonstrate organic physical losses, show that any preexisting susceptibility was either aggravated or worsened, and prove that the current level of functioning has changed relative to the premorbid status. Moreover, in most instances, at trial a medical diagnosis causing the damage will be required. The examiner, within the course and scope of a competent neuropsychiatric evaluation for forensic purposes, should also determine if there are secondary gain mechanisms at work that are perpetuating symptoms. Is there evidence of malingering or symptom magnification, and is there a direct and continuous medical relationship between the examinee's claim of injury and a demonstrable

neuropsychiatric cause? Lastly, to make an effective medical argument for either the presence or absence of damage following alleged traumatic brain injury, the medical outcome of the examinee will be one of the most important findings that the neuropsychiatric examiner must make.

## ADULT OUTCOMES FOLLOWING TRAUMATIC BRAIN INJURY

In the last 25 years, evidence-based analysis indicates that how traumatic brain injury is managed in a community hospital setting has a direct effect upon outcome. The American Association of Neurological Surgeons (AANS) has produced *Guidelines for the Management of Severe Head Injury*. A recent study evaluated application of these guidelines for efficacy. A cohort of 93 patients was selected, and 37 patients were treated before the implementation of AANS *Guidelines*; the results were statistically compared with 56 patients treated after the implementation of *Guidelines*. Implementation of the recommendations in the AANS *Guidelines* in a standardized protocol resulted in a 9.13 times higher odds ratio of a good outcome relative to the odds of a poor outcome or death, compared with a group managed before the traumatic brain injury practices changed. A dedicated neurotrauma team and comprehensive treatment algorithms were judged to be critical elements to this success. However, in this particular study, hospital charges increased by more than \$97,000 per patient, but it was felt that they were justifiable in the face of significantly improved outcomes. This study concluded that implementation of a traumatic brain injury protocol in a community hospital setting was both practical and efficacious.<sup>13</sup> It has been argued that the improved survivability and improved outcomes using criteria such as those provided by AANS can be attributed to the approach of “squeezing oxygenated blood through a swollen brain.” Cerebral perfusion monitoring of intracranial pressure and treatment of cerebral hypoperfusion in turn decrease a secondary injury, and this has improved outcomes.<sup>14</sup> Moreover, because most of the pathologic processes that determine outcome are fully active during the first hours after traumatic brain injury, the decisions of the initial emergency care providers may be crucial. Emergency management of traumatic brain injury is often directly linked to neurologic and neuropsychiatric outcome.<sup>15</sup>

There are some early indicators that may be predictive of functional outcomes after traumatic brain injury. For instance, the motor section of the *Glasgow Coma Scale* (GCS) (see [Chapter 1](#)) has been tested relative to outcome. A motor score of 5 vs. a motor score of 6 on the GCS was tested in 496 subjects who had sustained a traumatic brain injury. A motor score of 5 measures time to motor localization, whereas a motor score of 6 measures the time until the victim can follow commands. The time until commands were followed (motor score of 6) was a better predictor of all the outcomes assessed in this study than time until motor localization occurred (motor score of 5). Time to command appeared to be a more powerful predictor of outcome after severe brain injury than the ability of the patient to localize.<sup>16</sup> The release of certain biochemical markers is associated with short- and long-term neuropsychological outcome after traumatic brain injury. Two markers being studied currently are neuron-specific enolase (NSE) and protein S-100B. In a cohort of patients, in whom most had sustained a minor head injury, patients with short- and long-term neuropsychological disorders had significantly higher NSE and S-100B serum concentrations and a significantly longer-lasting release of both markers. Those patients with good neuropsychological outcome vs. those with poor neuropsychological outcome could be discriminated on the basis of these neurobiochemical serum markers.<sup>17</sup> Magnetic resonance imaging (MRI) has also been found useful at early and delayed time points after traumatic brain injury with regard to predicting the severity and clinical outcome of patients.<sup>18</sup> Recent review of MRI data on hippocampus volume following traumatic brain injury, compared against normal aging, reveals that in the subacute phase, the volume of the temporal horn may be indicative of the intellectual outcome of the patient, whereas the volume of the hippocampus appears to be indicative of the level of verbal memory function.<sup>19</sup>

Purely clinical observations and measures also have some predictive power in terms of outcome after traumatic brain injury. The level of agitation is intimately related to cognition early after traumatic brain injury. A longitudinal study of 340 consecutive patients admitted to an acute

traumatic brain injury rehabilitation unit was conducted. Lower cognitive function at admission to a rehabilitation unit was associated with the occurrence of agitation during rehabilitation, a longer length of stay in the facility, and lower cognitive function at discharge. Furthermore, this predicted a decreased likelihood that an individual would be discharged to a private residence. This study has urged the importance of systematically monitoring both agitation and cognition when applying interventions to reduce agitation.<sup>20</sup> While this study has short-term predictive ability, the early cognitive assessment with neuropsychological tests has longer-term predictive abilities. A total of 388 adults with traumatic brain injury were evaluated after their posttraumatic amnesia had resolved prior to discharge from inpatient rehabilitation. Neuropsychological tests were administered when the patient emerged from posttraumatic amnesia. Productivity status was evaluated at follow-up examinations 12 months postinjury. Persons scoring at the 75th percentile on early cognitive status (less impaired) had 1.61 times greater odds of being productive at follow-up than those scoring at the 25th percentile (more impaired).<sup>21</sup> Psychiatric disorders also play a prominent role in functional disability and outcome following traumatic brain injury. Depression and anxiety are more common in outpatients who have sustained traumatic brain injuries (see [Chapter 2](#)). Patients with depression or anxiety are more functionally disabled and perceive their brain injuries and cognitive impairments as more severe than they may be. Moreover, depressed patients report more severe postconcussion symptoms.<sup>22</sup> Fann and colleagues, who reported the effects of depression and anxiety following traumatic brain injury, have been able to demonstrate cognitive improvement with aggressive treatment of depression following mild traumatic brain injury.<sup>23</sup> Lastly, with regard to early predictors of outcome, while MRI and computed tomography (CT) may be useful for prediction of outcome, the routine use of positron emission tomography (PET) scanning during rehabilitation seems to add no predictive power to that which may be determined by MRI in children and adolescents.<sup>24</sup>

The neuropsychiatric examiner working in a legal setting may be asked to make some prediction about a brain-injured person's likelihood of returning to work in the future. Numerous guidelines have been published regarding return-to-work outcomes, factors influencing return to work, and vocational programs that enhance employment.<sup>25</sup> However, to make a specific recommendation based on a specific test is difficult if not impossible. As discussed in [Chapter 6](#), there are some significant limitations with clinical testing due to the ecological validity of determining work capacity. If outcome is defined as the patient being competitively employed or enrolled full-time in regular education following traumatic brain injury, neuropsychological testing early after brain injury has some predictive capacity. Neuropsychological testing can help predict long-term productivity even when performed before discharge. Competent testing can predict productivity, but the testing may not be able to predict whether the patient can be employed at a specific occupation in the future.<sup>26</sup>

Numerous other clinical indicators may be of some predictive value for determining outcome following traumatic brain injury. Self-awareness is often a problem following traumatic brain injury (see the emotional intelligence discussion in [Chapter 7](#)). The level of personal self-awareness following traumatic brain injury is directly associated with greater motivation to change behavior. However, the higher the self-awareness, the more likely the patient is to demonstrate depression.<sup>27</sup> As stressed in [Chapters 8](#) and [11](#), the neuropsychiatric examiner should carefully review the acute hospital record to determine length of coma and duration of posttraumatic amnesia. These factors have been studied in a group of 508 traumatic brain-injured rehabilitation patients ranging in age from 0.8 to 71 years with a mean age of 19 years. The follow-up period was between 5 and 20 years, with a mean of 12 years. The main outcome measures were functional outcome measured by the *Glasgow Outcome Scale* and postinjury occupational outcome. The length of coma and duration of posttraumatic amnesia were correlated specifically with the patient's work history after the brain injury and with functional outcome measured by the *Glasgow Outcome Scale*. This study determined that the extent of recovery and quality of life for rehabilitation patients with traumatic brain injury can be estimated early on by prognostic factors reflecting the injury severity in the

acute phase. These results suggested that the Glasgow Coma Scale (GCS) score, positive loss of consciousness, and duration of posttraumatic amnesia all have a strong predictive value in assessing functional or occupational outcome later in life.<sup>28</sup> The level of executive functioning following traumatic brain injury is likewise important and has some predictive value (see [Chapter 6](#) for executive function discussion). The *Stroop Color and Word Test* will differentiate individuals who require no assistance with activities of daily living from those who require some level of assistance. The Stroop Color and Word Test may have greater differential power to determine those who will be competitively employed from those who will remain unemployed or require sheltered employment. This test seems more powerful in this predictive ability than either the *Wechsler Adult Intelligence Scale-Revised* (WAIS-R) or the *Wechsler Memory Scale-Revised* (WMS-R).<sup>29</sup> Another test of executive function, the *Tower of Hanoi/Sevilla*, is a good tool for evaluating the executive functioning routinely in traumatically brain-injured patients with regard to outcome. In one study, the Glasgow Outcome Scale failed to detect more than 25% of patients with severe executive impairment, whereas the Tower of Hanoi/Sevilla was much more sensitive.<sup>30</sup> This test has been improved in its psychometric function and is part of the Tower Test in the *Delis-Kaplan Executive Function System* testing described in [Chapters 6](#) and [7](#).

Regardless of how outcome is measured, it is clear that many of those who sustain a traumatic brain injury will need lifelong intervention. An Australian study of 254 traumatic brain injury patients followed individuals for 2 to 5 years following injury. Visual difficulties, headache, and fatigue were persistent in a significant number of patients. Between 2 and 5 years after injury, there was increased independence in personal, domestic, and community activities of daily living in the use of transport. After 5 years, there was a slightly higher incidence of cognitive, behavioral, and emotional changes. Thirty-two percent of those who returned to work at 2 years were subsequently not employed at 5 years. Many who were students at the time of their injury had also become unemployed. The findings of this study suggest the need for intermittent lifelong intervention following traumatic brain injury, and systems of rehabilitation should be adapted to provide this.<sup>31</sup> This same Australian group had earlier reported that around two thirds of their sample reported cognitive, behavioral, and emotional changes. This underscores a need for ongoing community-based support and assistance in dealing with practical difficulties and psychological problems as they are experienced after the brain-injured person returns to the community.<sup>32</sup> A recent New Jersey study stressed that neuropsychological recovery after traumatic brain injury is not uniform across individuals or across neuropsychological domains. It identified a subset of persons with moderate to severe traumatic brain injury who demonstrated some continued neuropsychological recovery several years after injury, with a few persons demonstrating substantial recovery. For other persons, measurable impairment remained 5 years after injury. Where improvement did occur, it was most apparent on neuropsychological tests measuring cognitive speed, visuoconstruction, and verbal memory.<sup>33</sup>

For the neuropsychiatric examiner providing consultation to a plaintiff or defense lawyer within a brain injury case, it will be expected that the physician will have some understanding of outcome following brain injury for the examinee in question. The determination of impairment is discussed more fully below. However, the model one uses to determine outcome plays a role in the accuracy of outcome prediction. There is some evidence that needs-based models using subjective indicators of improvement clearly predict more variance in measures of life satisfaction, or subjective well-being, than do other types of models relying on more objective measures, such as neuropsychological assessment. This suggests that needs-based models may have greater ecological validity.<sup>34</sup> That validity concept has been discussed previously in this text (see [Chapter 6](#)). As noted previously in this text, the majority of improvement following traumatic brain injury occurs in the first 6 months postinjury, and clearly the vast majority occurs within 12 months of the injury. However, there are always statistical outliers within any normative population. Thus, there will be some individuals who will continue to demonstrate improvement even as far out as 5 years. If one examines this concept in a purely statistical fashion within a court of law, while the examiner can

predict it is possible that the person will improve further after 1 year, these possibilities may not rise to the level of “reasonable medical probability.” Since that is the standard generally required in courts of law for personal injury cases where medical testimony is offered, the examiner should keep this in mind. Moreover, the examiner may be able to say within reasonable medical probability that there is a substantial likelihood that a given individual will demonstrate a reduction in disability within 18 months of traumatic brain injury. However, that statement does not imply that cognitive skills will return to baseline.<sup>35</sup> Please note the important distinction between impairment and disability as described previously.

As was discussed earlier, in personal injury law, we “take the victim as he is found.” That implies that many factors present prior to brain injury may have an impact upon outcome. Therefore, in a medical–legal brain injury situation, the examiner should consider issues that the examinee brings to the case that were present prior to injury and that may adversely affect outcome or improvement. One issue that affects outcome is seizure disorders. In a Finnish study, a consecutive sample of 490 patients was followed for at least 5 years from the time of injury within a rehabilitation and reemployment program. Outcomes were studied separately among patients with late seizures and among a nonseizure group. Children ages 7 or younger at the time of injury were more likely to have early posttraumatic seizures than were adolescents or adults. The time elapsed between the brain injury and the first late seizure also was of greater length in older age groups. The presence of an early seizure or a depressed skull fracture had a statistically significant relationship to the origin of late-onset seizures. Risk factors for seizures included permanent posttraumatic neurological deficit, linear skull fracture, and permanent local brain lesion documented on CT scan. Late seizures worsened the functional outcome but had no significant influence on whether the person was employed at the end of the follow-up period. Adequate antiepileptic therapy improved rehabilitation goals and reemployment.<sup>36</sup> The presence of seizures may play a role in requirement for rehospitalization following traumatic brain injury. The neuropsychiatric examiner, acting as an expert witness, may be asked by the lawyer to assist in the preparation of a life-care plan. Rehospitalization is one of the costs that may require expert substantiation within a life-care plan. A Virginia study followed traumatic brain injury victims for 1 to 5 years after injury to investigate the incidence and cause of rehospitalization. This was a large study that reviewed 17 medical centers in the federally sponsored Traumatic Brain Injury Model Systems. In each setting, the continuum of care included emergency medical services, intensive and acute medical care, inpatient rehabilitation, and a spectrum of community rehabilitation services. Eight hundred ninety-five patients admitted to acute care within 24 h of traumatic brain injury between 1989 and 1999 were examined at 1-year follow-up and 5 years postinjury. The incidence of rehospitalization ranged from 22.9% at 1 year after injury to 17% at 5 years after injury. At 1 year after injury, one-third of the rehospitalizations were for elective reasons. At 5 years after injury, the incidence of readmissions for seizures or psychiatric difficulties and general health maintenance increased substantially. Thus, there remains a relatively high rate of rehospitalization in the long term after traumatic brain injury.<sup>37</sup> Another interesting study by the same research group compared the functional outcome, length of stay, and discharge disposition of individuals with brain tumors vs. those with acute traumatic brain injury; 78 brain tumor patients were matched one-to-one by location of lesion and age with 78 acute traumatic brain injury patients. The *Functional Independence Measure* was used on admission and at discharge. The brain injury population had a significantly greater change in measures of functional independence, and the tumor group had a significantly shorter rehabilitation length of stay and a greater discharge to community rate.<sup>38</sup> Rarely, a person who has had a brain tumor will subsequently suffer a traumatic brain injury, through either motor vehicle accident or a fall. Based on this study, it appears that the functional outcome is worse for traumatic brain injury than for brain tumor. Other preinjury factors that may have a negative influence on outcome following traumatic brain injury include substance abuse and older age. A Mississippi study has noted that patients with no history of preinjury substance abuse were more than eight times as likely to be employed at follow-up than those who had a



history of preinjury substance abuse.<sup>39</sup> Increasing age is a strong independent factor in prognosis, with a significant increase in poor outcome above 60 years of age.<sup>40</sup> Interestingly, there is a Canadian report that is in direct opposition to the AANS. This report concludes that it is premature to suggest that the elderly have a uniformly poor outcome following traumatic brain injury.<sup>41</sup> With the younger population, the examiner may be asked to predict function in college students. Those with moderate to severe injury generally fare similar to their older adult counterparts. On the other hand, youngsters who sustain a mild brain injury in childhood or adolescence may be intellectually unimpaired. There is evidence that they approach their studying in a manner similar to that of their uninjured classmates at college. However, emotionally they report more severe distress in terms of their general and personal functioning.<sup>42</sup>

## CHILD OUTCOMES FOLLOWING TRAUMATIC BRAIN INJURY

Interestingly, children who fare the worst following traumatic brain injury are those who sustained their injuries while riding in a vehicle, as opposed to being hit by a vehicle while walking or riding a bicycle.<sup>43</sup> For the severely injured child, there are certain predictors of probable outcome. A large university trauma center studied severely injured children using vital sign determination during the first 24 h of admission and administration of the *Pediatric Risk of Mortality* and the GCS. This study further measured the duration of mechanical ventilation and the number of pediatric intensive care unit (ICU) and hospital days required prior to discharge. Functional status was graded as normal, independent, partially dependent, or dependent in the areas of locomotion, self-care, and communication. A total of 105 children were studied over a 5-year period. Follow-up evaluations were available for 80 patients. Nineteen patients died. Mortality and dependent functional outcome were more likely in patients with younger age, lower GCS scores, and higher Pediatric Risk of Mortality scores at hospital admission. A GCS score of 5 is strongly associated with death or poor functional outcome. The Pediatric Risk of Mortality score adds to the power of the GCS to predict survival and functional outcome in those youngsters who required tracheal intubation. Of the 78 patients who survived and were available for follow-up, the number who were functionally normal or independent increased to 2 of 3. Obviously, that means that one of three was not functionally independent.<sup>44</sup> Another university study reviewed 60 children less than 6 years of age who sustained either inflicted or noninflicted traumatic brain injury. The variables measured included the GCS score, the duration of impaired consciousness, and the number of intracranial lesions visualized on CT-MRI. These variables accounted for a significant amount of the variance in the Glasgow Outcome Scale as well as the cognitive and motor scores at baseline and at 3- and 12-month evaluations. Neither age at injury nor the *Injury Severity* score accounted for a significant variability in outcomes.<sup>45</sup> Whereas motor vehicle accidents are more likely to produce a traumatic brain injury in a child than a pedestrian injury, a crush injury to an infant or young child's head is less likely to produce significant traumatic brain injury than dynamic loading of the head caused by impact (see [Chapter 1](#)).<sup>46</sup> Neuropsychological outcome after brain injury produced by static loading of the head is more favorable to the child than that after traumatic brain injury associated with dynamic loading.

The neuropsychiatric examiner who evaluates children following brain injury may be asked to testify regarding recovery trends and changes in cognitive domains after injury. In children who are moderately or severely injured, research indicates that the chronicity of neurobehavioral deficits during the first 3 years following injury shows a strong rate of improvement during the first year, but a negligible rate of change during the following 2 years postinjury in most cognitive domains. Over time, the recovery rate slows down more for those with greater brain injury severity. The greatest slowing of recovery occurs in performance IQ, adaptive problem solving, memory function, and motor skills. The examiner can testify in most instances that achievement of parity with peers by the moderately and severely injured child seems unlikely. On the other hand, mildly injured children generally exhibit negligible deficits or change in performance over time.<sup>47</sup> With regard to

IQ, for severe traumatic brain injury, the younger the age at injury, the more minimal the recovery in IQ. For older children, recovery of intellectual function is similar to that for adults. Findings from a study of 124 children divided according to age at injury indicate that sustaining severe traumatic brain injury in early childhood may be a particular risk for residual problems following injury.<sup>48</sup> Severe traumatic brain injury can have a pernicious effect on discourse abilities in children after injury compared with children with mild to moderate injuries. Verbal discourse would include telling stories or describing pictures.<sup>49</sup> Children following brain injury may also show impairment of social problem solving. Brain-injured children generate fewer positive assertive responses, and give more indirect responses, to peer group entry situations than a noninjured comparison group.<sup>50</sup> This social problem-solving difficulty may well extend into adulthood for those children injured while young. Whereas the intellectual deficits sustained by the brain-injured child persist into adulthood, it seems that the prevailing problems for the adult injured as a child are more related to social maladjustment and poor quality of life and are an outcome of behavioral and psychosocial disorders.<sup>51</sup> Refer to [Chapter 7](#) for a further discussion of emotional intelligence, which is probably the variable that adversely affects social adjustment and community integration following traumatic brain injury. Lastly, with regard to predicting outcome, it is again important for the neuropsychiatric examiner to realize that neuropsychological assessment of children contains many of the ecological validity pitfalls noted for adults.<sup>52</sup> Thus, ecologically valid assessments of children following brain injury will require multiple data sources. It is not wise to rely solely upon neuropsychological assessment for prediction.

As with the adult, the neuropsychiatric examiner can predict a significant family burden due to traumatic brain injury in a child. At trial, the physician expert may be asked to present information for a consortium claim of parents due to the loss of parent–child interaction that may occur following traumatic brain injury. This is an evolving area of the law in some states at the time of the writing of this text. It is far more of a burden upon a family to care for a child with traumatic brain injury than it is to care for a child with an orthopedic injury. The nature and severity of physical and cognitive problems in the child are most closely related to injury severity, but the family functioning and child behavior are most highly predicted by psychosocial and premorbid factors.<sup>53</sup> Therefore, the examiner must develop some ideas about the premorbid functioning of the family prior to the child’s injury. More dysfunctional families are more likely to claim a burden due to the injured child than those families that were more functional prior to the child’s injury. Also, the level of support systems available to the family correlates directly with the family’s ability to cope with the injured child. For instance, children who had a preinjury learning disability will fare worse following traumatic brain injury than their peers who have no such disability.<sup>54,55</sup> For the child with preinjury attention deficit hyperactivity disorder (ADHD), there is a positive dose–response relationship between severity of injury and change in ADHD symptoms. These changes are considered to be a direct effect of the brain damage.<sup>56</sup> Thus, in some legal cases, the physician expert will be asked to apportion preinjury factors relative to postinjury cognitive impairment.

The physician expert may be asked for an opinion about the effect of childhood traumatic brain injury upon educational attainment. While the exact educational needs of the child are best determined by educational professionals, the physician should have some familiarity with medical needs that may impact educational planning. Most, if not all, public school systems in the U.S. are required to provide educational opportunities for disabled children. This, of course, includes those children who have been traumatically brain injured. Most educational specialists recommend an integrated intervention approach.<sup>57</sup> It is recommended that when the child is in rehabilitation, multidisciplinary evaluation by educational specialists be performed at that time in order to facilitate a smooth transition to school and also to detect deficits that may require remediation.<sup>58</sup> Specifically, educational research suggests that children who have sustained severe brain injury exhibit greater deficits on reading comprehension and arithmetic, while those children with moderate to severe injuries also perform at a lower level of function in the areas of reading accuracy and spelling.<sup>59,60</sup> Many children following traumatic brain injury will require a change in placement from regular to special

education. Neuropsychological testing may be useful in identifying children with special educational needs in order to assist educational professionals with proper academic placement.<sup>61</sup> Furthermore, traditional achievement tests used by school professionals may be insensitive to detecting posttraumatic academic deficits in children following brain injury.<sup>62</sup> More sophisticated neuropsychological testing should probably be performed in children following traumatic brain injury, and those results should be made available to educational professionals for planning and placement purposes. For the physician expert, these issues speak directly to damages. How is tutoring to be paid? What is the duration of tutoring required? What impact will the child's brain injury have upon future educational and occupational attainment?

## **SEVERITY-RELATED OUTCOMES FOLLOWING TRAUMATIC BRAIN INJURY**

### **Outcome from Mild Head Injury**

A mild head injury can be defined as a trauma caused by blunt force or sudden acceleration–deceleration that produces a period of unconsciousness of less than 20 min, a GCS score of 13 to 15, no focal neurological deficit, no intracranial complications, and CT scan findings limited to a skull fracture without evidence of parenchymal contusion or hematoma.<sup>63,64</sup> Uncomplicated mild head injury makes up 80% of all hospital admissions in San Diego County. This produced an incidence rate for mild head injury of 130.8 per 100,000 persons per year.<sup>65</sup> The primary outcomes from mild head trauma are postconcussion syndrome and psychiatric sequelae. This is true in both adults and children.<sup>66</sup>

The percentage of patients reporting postconcussion syndrome within 1 week of injury ranges from 82 to 93% across three medical centers.<sup>67</sup> The three most frequent symptoms at all centers were headache (71%), decreased energy (60%), and dizziness (53%). While the severity of symptoms diminished after 3 months, mild residual complaints persisted in cognition, body aches and pains, and affective disorders. Affective disorder is considered to have a neurogenic etiology. An older study by Rutherford and others found 7% of patients reporting postconcussion syndrome symptoms 1 year after mild head injury.<sup>68</sup> However, this is considered consistent with the prevailing level of similar symptoms in the general population.<sup>69</sup> With regard to the psychiatric problems, emotional disturbance is very common after even a mild head injury. When standard psychopathology instruments are used, at 6 weeks after injury, 39% of head-injured patients report symptoms consistent with diagnoses of depression or anxiety, compared with only 4% of control patients. However, the patients who developed depression or anxiety were on the average 10 years older than the control patients and were more likely to be women.<sup>70</sup> Patients who had a preexisting psychiatric illness seem more likely to develop emotional complications following postconcussion syndrome.<sup>71</sup>

### **Outcome from Moderate Head Injury**

Unlike mild head injury, the definition of moderate head injury is more complicated and more variable. For research purposes, most authorities recommend that the patient be observed 6 to 48 h before an injury category is assigned. However, many authorities have reached a consensus that the moderate head injury is defined as a GCS score of 9 to 12 at the time of hospital admission. Moreover, patients with a GCS score of 13 to 15 who have an intraparenchymal lesion on CT scan are generally also placed into the moderate category.<sup>72</sup> The demographics seem quite different for this category of injury compared to mild head injury. An early study of moderate head injury found the patient population to be 77% male with a mean age of 33 years and a mean blood alcohol level of 0.14% at the time of admission to hospital. Forty-two percent revealed a history of prior head injury, 34% had a history of alcohol abuse, and 21% were chronically unemployed.<sup>73</sup>

Rimel's group found that 21% of patients who presented with a moderate head injury improved to a mild level within 6 h.<sup>73</sup> However, many patients with moderate head injuries by GCS criteria

will progress to significant pathological changes following the primary impact (see [Chapter 1](#)). The large study by Stein<sup>72</sup> revealed that of 447 cases of moderate head injury, 60% made a good recovery, 26% made a moderate recovery, 7% were left with severe disability, and 7% were left in a vegetative state or died. This, of course, means that 40% of those with moderate head injury in this series had less than a good recovery. Complications of moderate head injury are common and frequently major. Thirty percent or more have intracranial lesions on CT scan. All patients with moderate head injury should be treated in a hospital where neurosurgical consultation is available. However, the outcome is much worse for patients with moderate head injury by GCS criteria who demonstrate delayed brain lesions on CT scans. For those individuals, measured with Glasgow Outcome Scale at 6 months, 3% demonstrated good recovery, 28% had moderate disability, 22% had severe disability, 37% were in a vegetative state or dead, and about 10% had unknown outcome.<sup>72</sup>

### **Outcome from Severe Head Injury**

Severe brain injury is defined as a GCS score of 8 or less. Persons who remain in a coma for more than 6 h generally are also included in the severe injury classification. Elderly victims of severe traumatic brain injury have particularly poor outcomes. The mortality rate in the early 1990s was 80% for those older than 56 years who sustained a severe brain injury.<sup>74,75</sup> For the severely injured patient, outcome determinations must focus on cognitive and neuropsychological issues, since improved neurosurgical care may improve or palliate most of the physical disorders associated with severe brain injury, whereas severe disabilities may remain in the mental and cognitive spheres.

A large group of adult patients followed by Tate and others for 6 years after severe traumatic brain injury revealed that in the group as a whole, 76% were classified as having either poor or substantially limited social reintegration. Even in those with the best Glasgow Outcome Scale scores, only 50% were classified as having good reintegration with regard to employment, interpersonal relationships, functional independence, social contacts, and leisure interests.<sup>76</sup> With regard to children, Kaiser and Pfenninger followed 24 children with severe traumatic brain injury who received state-of-the-art acute care in a neurointensive care unit. After 2½ years, 42% had residual focal neurological deficits and 58% had measurable neuropsychiatric deficits, primarily characterized as altered personality. However, despite these deficits, the authors reported that few required special education.<sup>77</sup>

## **EVALUATING LEGAL COMPETENCE FOLLOWING TRAUMATIC BRAIN INJURY**

### **ADULT COMPETENCE**

The term *competency* is generally used in reference to decision-making and communicating capacity. General competency refers in many statutes to the ability of a person to manage his affairs. Specific competency is defined in relation to a particular act (e.g., to make a will).<sup>3</sup> The President's Commission for the Study of Ethical Problems in Medicine and Biobehavioral Research (1982) defined competency as "the ability to make autonomous decisions; to reason and deliberate and to understand and communicate information; and the possession of goals and values."<sup>78</sup> Hoge and others have defined decisional competence as "the capacity to: understand information relevant to the issue at hand; think rationally about alternative courses of action; appreciate one's situation as a person confronted with a specific decision; and express a choice among alternatives."<sup>79</sup> When legal standards are applied relative to medical standards, competency becomes much more narrowly defined. In general, competency is defined as cognitive capacity, and there are no established or set criteria for determining a person's specific competence. However, for consenting to medical treatment, a minimal level of decision making must exist and, generically, a person should be able to comprehend and perform at least all of the following: (1) understand the particular treatment

being offered; (2) make a discernible decision, one way or another, regarding the treatment that has been offered; and (3) communicate, verbally or nonverbally, his or her decision.<sup>80</sup>

When the law makes competence relevant, it begs the question of a person's legal capacity to act, not to determine through circumstantial reasoning what did or will occur, but to determine directly the legal significance of an actor's behavior.<sup>81</sup> Numerous legal cases have reviewed various human functions where mental competence is an issue. The U.S. Constitution prohibits the trial of an incompetent person.<sup>82</sup> It further prohibits the execution of an incompetent criminal defendant.<sup>83</sup> An incompetent person may not give consent to health care or health procedures.<sup>84</sup> A person must have basic competence to execute a will.<sup>85</sup> For the physician performing a neuropsychiatric examination of a brain-injured person, the major issues that may arise can affect any area of human life. The term *competency* is used by laypersons, and even many physicians, as a general, broad concept. The forensic examiner must ask a question: Where competency of a brain-injured person is raised as an issue at law, competency to perform or agree to what? This is a critical question that must be answered during competency assessment. Unfortunately, what often transpires in a competency issue is that a physician evaluates a person, determines if she is oriented to person, place, and time and if she knew what she had for breakfast, and pronounces her competent. If the question before the examiner is, "Can this individual manage her financial affairs?" then that is a very different issue and requires a very different examination than a question as to whether the injured person can give consent for a medical procedure. The elements necessary to make a rational decision are different for each of these two specific competencies. As has been discussed in the clinical section of this book, examinations of mental state should strive to have relevance to the real world and prove themselves ecologically valid. Therefore, an examination to see if a person can manage her finances requires some examination of arithmetic skill, number memory, and other issues. For instance, the examiner must have some idea about the individual's assets in order to determine if the examinee understands the range and nature of her assets. It should be clear that for the gentleman under consideration for an elective surgical procedure, a very different set of questions and examinations must transpire relative to the woman who may be unable to manage her financial affairs. Thus, in this context, competency refers to some minimal mental, cognitive, or behavioral ability, trait, or capability required to perform a specific, legally recognized act or to assume some legal role. The term *capacity* is often interchanged with the term *competency* and generally refers to an individual's actual ability to understand or to form an intention with regard to some specific act.<sup>80</sup>

As the neuropsychiatric examiner approaches a brain-injured person regarding competency, there are four general standards that should apply to the examiner's mental assessment for general competence.<sup>86</sup> Table 10.1 outlines a mental examination schema for determining general competence in decision making. Moreover, when performing any type of competency examination within a medical–legal framework, the physician must first be aware of the applicable competency standards and their particular jurisdiction. Legal consultation may be required to determine these standards. The examination then should focus on the four basic capacities of competence: the ability to understand relevant information, the ability to appreciate the nature of the situation and its likely consequences, the ability to manipulate information rationally, and the ability to communicate a choice.<sup>81</sup> A comprehensive mental status examination will be critical to answering the questions in Table 10.1. Moreover, ancillary measures may be required to support findings obtained during the mental status examination. For instance, is there evidence of executive dysfunction on psychological testing? Is there evidence of a significant reduction in intellectual capacity as a result of brain injury? Has the brain injury produced evidence of dementia on measures such as the *Mini-Mental State Examination*? Ancillary testing is used merely for confirmation of the mental status examination and to add information that may be of use to a court in the determination of competency. In most instances, competency is determined by a judge rather than a jury, and therefore, the examiner's database can be established at a detailed and complex level appropriate for judicial use. Where a person's capacity to consent to medical treatment is called into question, a semistructured

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**TABLE 10.1**  
**Neuropsychiatric Assessment of General Competency**

Ability to understand relevant information:	Is the person generally aware of his current circumstances? For example, where he lives, the sources of income, the general nature of his assets, any significant persons who assist him, and any threats to his person and financial security (e.g., depletion of his estate).
Ability to understand the issues at hand:	Can the person demonstrate her capacity to understand the relevant facts? For instance, does she know that a monthly payment reduction is required in order to extend the payout period of her IRA?
Ability to appreciate likely consequences:	Does he understand that if the home is sold to provide an income for life he will no longer be allowed to live there? Does she understand that lack of payment to the water company will result in the turning off of the water supply?
Ability to manipulate information and communicate a choice:	Can the person demonstrate his orientation, memory, judgment, logic of thought, and regulation of affect? Can the person demonstrate by explicit examples that she can manipulate data about her assets and express her wishes for their use? Can the person understand, appreciate, reason, and express a choice?
Apply appropriate cognitive, psychological, and imaging measures to confirm clinical findings:	Perform appropriate mental status examination, neurological examination, standardized cognitive and psychological testing, brain neuroimaging, and laboratory studies.

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interview format is available for clinical use. The *MacArthur Competence Assessment Tool-Treatment* (MacCAT-T) is available to assist in either a forensic examination or a clinical examination of this important capacity.<sup>87</sup> However, it is important to recognize that the MacCAT-T does not provide scores that translate directly into determinations of legal competence or incompetence. The examiner's judgment and other forms of examination will be required for a complete assessment of competence to enter into medical treatment.

## CHILD COMPETENCE

Children, by definition, are dependent persons and have parents or guardians. However, there are occasions wherein a brain-injured child could come under the scrutiny of the juvenile court system. Moreover, older teenagers who commit serious crimes against others are often waived to adult court and are tried as adults. Children also may, at times, be witnesses in a civil or criminal court action, and the brain-injured child's competency to testify may be rightfully raised as an issue.<sup>88</sup> The credibility of child witnesses often is an issue before the court, and if the child witness has sustained a traumatic brain injury, almost assuredly, the child's credibility will be questioned. Other child issues in a traumatically brain-injured youngster that may come to the attention of an examiner are assessing risk of harm to others. This could be an issue as a direct outcome of traumatic brain injury, or as is more often the case, a child with oppositional defiant disorder or attention deficit disorder sustains a traumatic brain injury and then demonstrates some marked exacerbation of behavior. The methods and procedures for evaluating juveniles have their own specific ethical rules, and in many instances, these are distinct from the ethics involved in adult assessment.<sup>89</sup> However, the basic evaluation of child competence is structurally no different than that of the adult. Thus, the forensic examination of child competence, as described for the adult, should determine the youngster's capacities for understanding, appreciating, reasoning, and expressing a choice.

## DETERMINING IMPAIRMENT FOLLOWING TRAUMATIC BRAIN INJURY

As noted earlier in the introduction, the physician directly determines impairment. Thus, the physician can determine impairment in one's patient by the methods discussed in [Chapters 1 to 8](#), or the

forensic physician can determine impairment at the request of another party for purposes of litigation. With one's patient, the level of impairment determined by the physician may be used by others to assist in disability determination. Determining whether an injury or illness results in a permanent impairment requires a medical assessment performed by a physician.<sup>2</sup> Once an impairment is established, this may lead to functional limitations or the inability to perform activities of daily living. When evaluating a brain-injured individual, an examining physician has two options: consider the individual's healthy preinjury or preillness state, or consider the condition of the unaffected side as "normal" for the individual, if this is known. The physician also can compare that individual to a normal value defined by population averages of healthy people. Both methods are generally used for impairment determination. With regard to traumatic brain injury, one cerebral hemisphere may be compared to the other, the cognitive assessment may be compared to normative standards for persons of like age and gender, or the individual's cognitive and psychological state may be compared to an estimate of preinjury functioning (such as described in [Chapter 6](#)). While experts may argue that there are other methods for determining impairment, no other impairment rating system is used by physicians in the U.S. that has such wide acceptance and sound scientific database as the American Medical Association's *Guides*. However, in assessment of impairment for litigation purposes, the *Daubert* rule must be kept in mind by the examiner. The *Guides*, probably without exception, will meet the standards of that rule. As discussed earlier in this text, during history taking, the physician should inquire as to activities of daily living. These are also called instrumental activities of daily living (IADLs).<sup>90,91</sup> These activities have been described as (1) self-care and personal hygiene, (2) communication skills, (3) physical activity ability, (4) sensory function ability, (5) nonspecialized hand activities, (6) traveling ability, (7) sexual functioning, and (8) sleep functioning. With regard to a neuropsychiatric assessment, almost all scales for measurement of either instrumental activities of daily living or activities of daily living are based primarily on a physical medicine model. For a significantly brain-injured person, these models work quite well. For the person who has only a mental or behavioral impairment following a brain injury, in the absence of physical impairments, these various rating scales measure poorly. Some of the common activities of living scales in use include the *OECD Long-Term Disability Questionnaire*, the *Health Assessment Questionnaire*, the *Functional Independence Measure*, and the *Barthel Index*.<sup>92-95</sup>

The *Guides* provide information to assist the examining physician with determination of causation, apportionment, and aggravation. These are important issues in the adjudication of workers' compensation and personal injury claims. Causation used in this medical sense is different than legal causation described above in this chapter. For purposes of using the *Guides*, causation means an identifiable factor (e.g., accident or exposure to hazards of a disease) that results in a medically identified condition.<sup>2</sup> The examining physician needs to be aware that the legal standard for causation in civil litigation or workers' compensation adjudication varies from jurisdiction to jurisdiction. It is the examining physician's responsibility to determine those standards when they are being applied within the context of a forensic medical evaluation.<sup>96</sup>

Apportionment analysis may be required in workers' compensation cases depending on the jurisdiction. This analysis derives from the fact that multiple factors may cause or significantly contribute to the injury or disease resulting in the impairment being assessed by the physician. For instance, the examinee may have a preexisting injury or impairment that plays a role in the genesis of the accident or injury under evaluation by the examiner. The examining physician may be asked by the attorney, workers' compensation carrier, or other third party to apportion or distribute a permanent impairment rating between the effects of the current injury and a prior injury or impairment rating. The *Guides* recommend following a protocol for the analysis of apportionment. The physician needs to verify that all the following information is true for an individual:<sup>2</sup> (1) there is documentation of a prior factor, (2) the current permanent impairment is greater as a result of the prior factor (i.e., prior impairment, prior injury, or illness), and (3) evidence indicates that the prior factor caused or contributed to the impairment, based on a reasonable medical probability (> 50% likelihood).

For the forensic neuropsychiatric examiner, this is not an unusual circumstance. Many persons sustaining brain injuries have had prior head or brain injuries. Thus, the physician performing a neuropsychiatric examination following brain injury, in some instances, may need to determine by apportionment analysis the contribution of the first brain injury to the second brain injury. The combined effect of the first and second brain injuries may result in an impairment rating that exceeds the mere additive effects of both. In other words, the outcome may be exponential rather than arithmetically additive. In workers' compensation cases, it often derives from a prior industrial injury that the employer shall be liable only for the additional disability from the injury or occupational disease due to the subsequent injury and not the prior injury.<sup>2,97</sup>

For purposes of using the *Guides*, aggravation refers to a factor, or factors, that alter the course of progression of the medical impairment.<sup>2</sup> With regard to a traumatic brain injury, this could be an individual who had a substantial attention deficit disorder or learning disorder as a youngster. If this disorder remained manifest into adulthood and that individual then sustained a traumatic brain injury, the traumatic brain injury could be an aggravating factor to the preinjury psychiatric condition. When evaluating a person for impairment, permanency should not be considered until the clinical findings indicate that the medical condition has become static and well stabilized and has reached maximum medical improvement. With regard to traumatic brain injury, improvement generally becomes static by 12 to 18 months postinjury. There are, of course, exceptions to this rule, but in most instances, and at least within reasonable medical probability, by 18 months the improvement in traumatic brain injury will have plateaued. Thus, a maximum medical improvement refers to a time specific from which further recovery or deterioration is not anticipated, even though there may be some slight expected changes or improvement. To determine a whole-person, or whole-body, impairment, the examining physician should first estimate the impairment for the person's most significant injury and evaluate other impairments secondary to or in relation to the primary impairment. If two or more significant medical conditions exist, each impairment rating is calculated separately unless they are related. For instance, following a traumatic brain injury, a person may have executive dysfunction and dysphasia. However, these would be interrelated conditions as an outcome of the primary brain injury rather than separate mental or cognitive constructs. Where separate unrelated conditions exist, the *Guides* provide a Combined Values Chart for combining impairments.

Following the examination of a brain-injured person, the physician examiner should complete a neurobehavioral analysis taking into account the cognitive and behavioral impairments in the person. Depending on the outcome of the neuropsychiatric evaluation, the physician examiner then may utilize, in most instances, two specific chapters of the *Guides*, fifth edition. These are Chapter 13 ("The Central and Peripheral Nervous System") and Chapter 14 ("Mental and Behavioral Disorders").<sup>2</sup> With regard to Chapter 13 of the *Guides*, the most relevant section for the neuropsychiatric examiner is 13.3d ("Mental Status, Cognition, and Highest Integrative Function"). Table 13-5 of the *Guides* enables the neuropsychiatric examiner to determine an impairment level and a Clinical Dementia Rating (CDR) score. These determinations include memory, orientation, and executive function as well as activities of daily living and self-care. Table 13-6 of the *Guides* provides criteria for Rating Impairment Related to Mental Status wherein the neuropsychiatric examiner can place the examinee into one of four classes. The Clinical Dementia Rating system was developed by Morris and provides the physician examiner with a schema for categorizing dementia syndromes as a result of traumatic brain injury or other medical causes.<sup>98</sup> Table 10.2 provides the Clinical Dementia Rating Scale, and Table 10.3 provides criteria for Rating Impairment Related to Mental Status. Chapter 11 of this text provides the physician with case examples for using these scales to determine impairment following traumatic brain injury.

Where an emotional or behavioral impairment is the direct outcome of brain trauma, Section 13.3f ("Emotional or Behavioral Impairments") of the *Guides* may be useful to the examining physician. Table 13-8 of the *Guides* describes criteria for rating impairment due to emotional or behavioral disorders into one of four classes. A Class I impairment describes a person with mild limitation of activities of daily living, whereas Class II describes a person with moderate limitation



**TABLE 10.2**  
**Clinical Dementia Rating**

	Impairment Level and CDR Score				
	None 0	Questionable 0.5	Mild 1.0	Moderate 2.0	Severe 3.0
Memory (M)	No memory loss or slight inconsistent forgetfulness	Consistent slight forgetfulness; partial recollection of events; “benign” forgetfulness	Moderate memory loss; more marked for recent events; defect interferes with everyday activities	Severe memory loss; only highly learned material retained; new material rapidly lost	Severe memory loss; only fragments remain
Orientation (O)	Fully oriented	Fully oriented except for slight difficulty with time relationships	Moderate difficulty with time relationships; oriented for place at examination; may have geographic disorientation elsewhere	Severe difficulty with time relationships; usually disoriented to time, often to place	Oriented to person only
Judgment and Problem Solving (JPS)	Solves everyday problems and handles business and financial affairs well; judgment good in relation to past performance	Slight impairment in solving problems, similarities, and differences	Moderate difficulty in handling problems, similarities, and differences; social judgment usually maintained	Severely impaired in handling problems, similarities, and differences; social judgment usually impaired	Unable to make judgments or solve problems
Community Affairs (CA)	Independent function at usual level in job, shopping, volunteer and social groups	Slight impairment in these activities	Unable to function independently at these activities, although may still be engaged in some; appears normal to casual inspection	No pretense of independent function outside home; appears well enough to be taken to functions outside a family home	No pretense of independent function outside home; appears too ill to be taken to functions outside a family home
Home and Hobbies (HH)	Life at home, hobbies, and intellectual interests well maintained	Life at home, hobbies, and intellectual interests slightly impaired	Mild but definite impairment of function at home; more difficult chores abandoned; more complicated hobbies and interests abandoned	Only simple chores preserved; very restricted interests; poorly maintained	No significant function in home
Personal Care (PC)	Fully capable of self-care	Fully capable of self-care	Needs prompting	Requires assistance in dressing, hygiene, keeping of personal effects	Requires much help with personal care; infrequent incontinence

From Morris, J.C., The Clinical Dementia Rating (CDR): current version and scoring rules, *Neurology*, 43, 2412, 1993. Reprinted with permission.

**TABLE 10.3**  
**Criteria for Rating Impairment Related to Mental Status**

<b>Class 1</b> <b>1–14% Impairment of the Whole Person</b>	<b>Class 2</b> <b>15–29% Impairment of the Whole Person</b>	<b>Class 3</b> <b>30–49% Impairment of the Whole Person</b>	<b>Class 4</b> <b>50–70% Impairment of the Whole Person</b>
Paroxysmal disorder with preimpairment exists, but is able to perform activities of daily living CDR = 0.5	Impairment requires direction of some activities of daily living CDR = 1.0	Impairment requires assistance and supervision for most activities of daily living CDR = 2.0	Unable to care for self and be safe in any situation without supervision CDR = 3.0

From *Guides to the Evaluation of Permanent Impairment*, 5th ed., AMA Press, Chicago, 2000, p. 320. Used with permission.

of some activities of daily living and some limitation of daily social and interpersonal functioning. Class III impairment is a fairly significant limitation in that the person demonstrates severe limitation in performing most activities of daily living that impedes useful action in most daily social and interpersonal functions. A Class IV behavioral impairment due to brain injury would cause severe limitation of all daily activities, requiring total dependence upon another person.<sup>2</sup> Thus, an individual with a severe infraorbital behavioral syndrome (see [Chapter 2](#)) as a result of a traumatic brain injury can be aptly described for impairment rating purposes in Chapter 13, Section 13.3f of the *Guides*. On the other hand, if the individual has a mood disorder or posttraumatic stress disorder as a result of a head injury with no evidence of organic mental impairment, Chapter 14 of the *Guides* may prove more useful to the examining physician.

For a purely psychiatric outcome following a traumatic brain injury, the method of evaluating psychiatric impairment in Chapter 14 is somewhat different than the method used in Chapter 13 of the *Guides*. In Chapter 14, the *Guides* specifically avoid the use of percentage ranges of impairment, whereas they are used in Chapter 13. The *Guides* point out in Chapter 14:

Percentages are not provided to estimate mental impairments in this edition of the *Guides*. Unlike cases with some organ systems, there are no precise measurements of impairment in mental disorders. The use of percentages implies a certainty that does not exist. Percentages are likely to be used inflexibly by adjudicators, who then are less likely to take into account the many factors that influence mental and behavioral impairment .... After considering this difficult matter, the Committee on Disability and Rehabilitation of the American Psychiatric Association advised *Guides* contributors against the use of percentages in the chapter on mental and behavioral disorders of the 4th Edition, and that remains the opinion of the authors of the present chapter.<sup>2</sup>

The *Guides* further point out that little relationship exists between psychiatric signs and symptoms identified during a mental status examination and the ability to perform competitive work. Four categories can be used to assess areas of function that are related to work ability: (1) ability to perform activities of daily living, (2) social functioning, (3) concentration, persistence, and pace, and (4) deterioration or decompensation in work or work-like settings. Thus, Table 14-1 of the *Guides* defines classes of impairment due to mental and behavioral disorders. Unlike Chapter 13, this classification system uses five classes ranging from Class I (no impairment noted) to Class V (impairment levels preclude useful functioning). A Class II impairment is at the mild level, and impairment levels are compatible with most useful functioning. A Class III mental or behavioral impairment is a moderate level, and impairments are compatible with some, but not all, useful functioning. A person is assigned to Class IV, marked impairment level, when impairments significantly impede useful functioning. [Table 10.4](#) lists classes of impairment due to mental and behavioral disorders that may provide useful assistance in the determination of behavioral incapacity

**TABLE 10.4**  
**Classes of Impairment Due to Mental and Behavioral Disorders**

Area or Aspect of Functioning	Class 1 No Impairment	Class 2 Mild Impairment	Class 3 Moderate Impairment	Class 4 Marked Impairment	Class 5 Extreme Impairment
Activities of daily living	No impairment noted	Impairment levels are compatible with most useful functioning	Impairment levels are compatible with some, but not all, useful functioning	Impairment levels significantly impede useful functioning	Impairment levels preclude useful functioning
Social functioning					
Concentration					
Adaptation					

From *Guides to the Evaluation of Permanent Impairment*, 5th ed., AMA Press, Chicago, 2001, p. 363. Used with permission.

following a traumatic brain injury. The reader is advised that, if impairment ratings are part of the customary practice of the physician, frequent consultation with the *Guides* will assist to standardize the examination process. Chapter 11 of this text describes in further detail specific use of the *Guides* for determining impairment levels following traumatic brain injury.

## DISABILITY DETERMINATION FOLLOWING TRAUMATIC BRAIN INJURY

According to a 1997 Institute of Medicine report, “Disability is a relational outcome, reflecting the individual’s capacity to perform a specific task or activity, contingent on the environmental conditions in which they are to be performed.”<sup>99</sup> The alternative definition of disability used by the *Guides* was noted previously. Since this section deals primarily with forensic issues, disability is described here, yet it is determined judicially or by an administrative law judge. However, it must be stressed that treating physicians, including psychiatrists, may be asked to evaluate their patients for disability within the context of a workers’ compensation claim or a social security claim. The physician will determine a level of impairment that can then be used by the adjudicating body to determine whether the patient has a disability. When the physician is conducting an independent medical evaluation, and not functioning on behalf of the patient, the evaluation is performed within the context of a forensic or non-patient-centered format.

Social Security Disability is determined under the Social Security Disability Insurance Benefits Program. The disability standard of this program requires that the claimant demonstrate an “inability to engage in any substantial gainful activity by reason of any medically determinable physical or mental impairment which can be expected to result in death or which has lasted or can be expected to last for a continuous period of not less than 12 months.”<sup>99</sup> Within 42 U.S.C. is contained the Administration’s Listing of Impairments.<sup>100</sup> The mental impairments are found in Listing 12.00 (“Mental Disorders”). Listing 12.02 contains organic mental disorders. This listing is where persons suffering traumatic brain injury would be categorized and classified. The listing for organic mental disorders requires satisfaction of requirements A and B:

- A. Demonstration of a loss of specific cognitive abilities or affective changes and the medically documented persistence of at least one of the following:
  1. Disorientation to time and place
  2. Memory impairment, either short term (inability to learn new information), intermediate, or long term (inability to remember information that was known sometime in the past)

3. Perceptual or thinking disturbances (e.g., hallucinations, delusions)
4. Change in personality
5. Disturbance in mood
6. Emotional lability (e.g., explosive temper outbursts, sudden crying, etc.) and impairment in impulse control
7. Loss of measured intellectual ability of at least 15 IQ points from premorbid levels or overall impairment index clearly within the severely impaired range on neuropsychological testing (e.g., the Luria–Nebraska, Halstead–Reitan, etc.)

AND

- B. Resulting in at least two of the following:
  1. Marked restriction of activities of daily living
  2. Marked difficulties in maintaining social functioning
  3. Deficiencies of concentration, persistence, or pace resulting in frequent failure to complete tasks in a timely manner (in work settings or elsewhere)
  4. Repeated episodes of deterioration or decompensation in work or work-like settings that cause the individual to withdraw from that situation or to experience exacerbation of signs and symptoms (which may include deterioration of adaptive behaviors).

Should the examinee or patient have a pure psychiatric syndrome due to traumatic brain injury, other categories of impairments may apply. Listing 12.03 covers schizophrenic, paranoid, and other psychotic disorders. Listing 12.04 covers affective disorders, including depressive, manic, or bipolar syndromes. Listing 12.05 covers mental retardation and autism. Listing 12.06 includes anxiety-related disorders. This would include posttraumatic stress disorder if it were present. Listing 12.07 includes somatoform disorders. Listing 12.08 includes personality disorders and would include a personality change due to a traumatic brain injury. Listing 12.09 contains the criteria for substance addiction disorders.

Druss and others recently reviewed the 1994–1995 National Health Interview Survey of Disability, the largest disability survey ever conducted in the U.S. Their review noted that 1.1% of adults reported a functional disability from a mental condition vs. 4.8% of adults who reported a disability from a general medical condition. They estimated that 3 million Americans (one-third of all disabled people) reported that a mental condition contributes to their disability.<sup>101</sup> However, review of this data does not allow one to determine what percentage of those with mental disability are disabled by virtue of traumatic brain injury. The *Federal Register* routinely publishes criteria for evaluating mental disorders in traumatic brain injury as they are revised.<sup>102</sup> Rules for determining medical equivalents in childhood disability claims when a child has marked limitations in cognition and speech are also routinely reported in the *Federal Register* as a Social Security Ruling.<sup>103</sup>

When the physician is performing an examination for Social Security Disability as a result of a traumatic brain injury, it is recommended that the criteria of Listing 12.02 be followed. Both criteria A and B in that listing enable the examining physician to determine by mental status examination, cognitive testing, and an examination of activities of daily living the current functioning of either a patient or an examinee. The report written on behalf of the patient or for the person who hired the physician to perform the independent medical examination (IME), should be clear, direct, and to the point in describing which of the specific criteria are met by the examination and the bases for the determination.

## FORENSIC MEDICAL HISTORY

A suggested format for an adult or postpubescent child Forensic Medical History is presented next. It should be completed by the examinee whenever possible. If the examinee is too injured to complete the form or the examinee is a minor, then the attendant, guardian, or parent should do so. The purpose of the Forensic Medical History is to collect written data from the examinee. Note

at the latter part of the history form that the person certifies by his signature that he is answering the information truthfully and accurately. This can have significant importance in a forensic examination of a potentially brain-injured person wherein the individual is symptom magnifying or malingering the head injury (see [Chapter 9](#)). At deposition or trial, the entirety of the form may be introduced by a lawyer as an exhibit.

The Forensic Medical History Questionnaire is not sufficient to take into account all of the elements that may occur in the evaluation of a traumatic brain injury. The forensic examiner is advised to consult [Chapter 3](#) for questioning specific to that of traumatic brain injury. These, of course, are clinical questions that should be asked of any patient during a comprehensive brain injury assessment, but they also are the same questions one would ask an examinee in a forensic brain injury situation. The use of a written Forensic Medical History provides other useful information. It is an indirect mental status examination of certain cognitive functions if completed by the examinee. For instance, the General Information page will provide information regarding the examinee's orientation and awareness of common information such as phone number, social security number, and zip code. This provides some useful information about the intactness of the person's biographical (episodic) memory. The Activities of Daily Living section is very important in disability determination. When an examiner is providing information for workers' compensation, social security, or other forms of potential disability, one of the important factors to be determined is how does the person function on a daily basis? It may be necessary to supplement this section with collateral information from attendants or family members familiar with the examinee's daily activity.

The Past Medical History is straightforward and helps the forensic examiner to determine if issues other than a traumatic brain injury may play a role in the symptom presentation of the examinee. Moreover, throughout the Forensic Medical History, information is requested from the examinee as to when symptoms first began. The examiner should note under History of Presenting Problem that the examinee is asked the year the mental problems first began. The same question is asked regarding physical problems. Under Past Medical History, the examinee is asked to list permanent physical or mental problems from childhood while at the same time determining if there were behavioral abnormalities during school. A history of prior motor vehicle accidents is taken, and in addition, the examinee is asked about prior losses of consciousness, coma, or bone fractures. These issues are important in terms of causation and determining the temporal relationship of symptoms presented within a forensic examination to the injury itself.

The Past Psychiatric History asks specific questions in an attempt to document the onset of mental disorder. Prior use of psychiatric medicines is explored. A specific question inquires as to the first time in life the examinee took psychiatric medications, if they were used. Moreover, in issues of contributory negligence, the examinee is asked regarding the refusal of mental treatment when recommended by a doctor and whether there is any past history of intentional overdose of medications or prior attempts to take one's life.

The Family History is straightforward, but it does play an important role in determining if there are genetic components to the brain-injured person's medical history. The Social History is extremely important in a forensic situation. This explores issues of abuse, violence, harassment, academic performance, and relationship issues. The Legal History can provide very specific information useful in the forensic neurobehavioral analysis. Does this individual have a past history of violence or a significant history of violent relationships? Have restraining orders or emergency protective orders been required? Has the individual ever been charged with terroristic threatening or abuse? Is there a past history of felony or misdemeanor convictions? The important issue of drug arrest and DUI is explored. How the person behaved in employed situations is determined. Military History further asks issues of psychiatric importance regarding disciplinary actions. For instance, individuals with antisocial tendencies prior to brain injury, personality disorders, or substance abuse histories may have a military discharge that is other than honorable. This may be an important marker for a preinjury behavioral disturbance.

The Review of Systems is straightforward and proceeds in a fashion similar to an ordinary medical inquiry. Its purpose is to localize symptoms of importance that may or may not be related to a traumatic injury. At the completion of the questionnaire, it is recommended that the forensic examiner, during the history taking, verify the information face-to-face. A useful technique in this regard is to write contemporaneous notes directly on the Forensic Medical History form but do so in red ink. Therefore, at deposition, should the form become attached to the transcript, it is very clear who put what information onto the form as the examining physician's notes will clearly be differentiated from the notes of the examinee. Obviously, the examinee must write in blue or black for this to occur. The examiner should be aware, however, that if the Forensic Medical History is photocopied, what is written in red will appear as black unless the photocopying is performed in color.

## FORENSIC MEDICAL HISTORY QUESTIONNAIRE

**WARNING:** Because you are being examined for purposes of your mental fitness or a legal action (workers' compensation, social security, civil rights, civil or criminal, etc.), please be aware that the information you supply in this questionnaire or tell the doctor may not be confidential.

This is a medical-legal or an independent medical examination. Forensic Doctor, M.D., *will not* have a doctor-patient relationship with you. This examination is *not* for treatment or counseling. Forensic Doctor, M.D., may video- or audiotape-record the interview. If he does, your lawyer can request a copy of the tape.

### General Information

Name: \_\_\_\_\_ Today's date: \_\_\_\_\_  
Address: \_\_\_\_\_ City: \_\_\_\_\_ State: \_\_\_\_\_  
Zip code: \_\_\_\_\_ Date of birth: \_\_\_\_\_ Age: \_\_\_\_\_  
Phone number: \_\_\_\_\_ Social security number: \_\_\_\_\_  
Which is your dominant hand (right, left, both)? \_\_\_\_\_ Your present weight: \_\_\_\_\_  
Can you read a newspaper? Yes \_\_\_ No \_\_\_ Education (highest grade completed): \_\_\_\_\_  
Employment (current): \_\_\_\_\_  
Address: \_\_\_\_\_ Phone: \_\_\_\_\_  
Lawyer or other agency who referred you to this office: \_\_\_\_\_  
If you are being examined for disability, workers' compensation, Social Security Disability, a lawsuit, or criminal charges, who is your lawyer? \_\_\_\_\_  
Did you drive yourself here today? Yes \_\_\_ No \_\_\_ If no, who brought you? \_\_\_\_\_  
What is his or her relation to you (friend, relative, hired by your lawyer, etc)? \_\_\_\_\_  
Who do you live with at this time? \_\_\_\_\_

### History of Presenting Problem

Have you been experiencing any mental or nervous problems in the last month? Yes \_\_\_ No \_\_\_ If yes, describe:

\_\_\_\_\_

What year did your mental problems first begin? \_\_\_\_\_

Have you been experiencing any physical problems in the last month? Yes \_\_\_ No \_\_\_ If yes, describe:

\_\_\_\_\_

What year did your physical problems first begin? \_\_\_\_\_

### Activities of Daily Living

Are you currently working? \_\_\_\_\_ How many hours per week? \_\_\_\_\_

Does your town contain more than 2500 people? Yes \_\_\_ No \_\_\_

What time do you get up in the morning? \_\_\_\_\_ What time do you go to bed at night? \_\_\_\_\_  
 Who fixes your breakfast? \_\_\_\_\_  
 Do you drive your car or truck? \_\_\_\_\_  
 Do you use a checkbook? \_\_\_\_\_ Who pays your bills? \_\_\_\_\_  
 Who cleans your home? \_\_\_\_\_  
 Who fixes your meals? \_\_\_\_\_  
 Do you attend church? \_\_\_\_\_ How often? \_\_\_\_\_  
 What hobbies do you now have? \_\_\_\_\_  
 What video games do you play? \_\_\_\_\_  
 What do you read? \_\_\_\_\_ Can you write? \_\_\_\_\_  
 Do you watch TV? \_\_\_\_\_ How many hours per day? \_\_\_\_\_  
 What do you do with your children? \_\_\_\_\_  
 What was your last overnight trip? \_\_\_\_\_  
 Who mows your yard? \_\_\_\_\_  
 What work do you do around your home or farm? \_\_\_\_\_  
 How many movies do you rent per month? \_\_\_\_\_ How many times do you go to the movie theater a year?  
 \_\_\_\_\_  
 How many times do you sleep away from home in a year? \_\_\_\_\_  
 How many ball games do you attend in a year? \_\_\_\_\_ How many times do you hunt in a year?  
 \_\_\_\_\_ How many times do you fish per year? \_\_\_\_\_  
 How many times do you eat out in a month? \_\_\_\_\_ How many times a month do friends or family  
 visit you in your home? \_\_\_\_\_ How many times a week do you call someone on your phone?  
 \_\_\_\_\_  
 What plants do you grow? \_\_\_\_\_  
 Can you dress yourself? \_\_\_\_\_ Can you bathe or shower yourself? \_\_\_\_\_  
 Can you have sex? \_\_\_\_\_  
 Do you have any problems using the bathroom? \_\_\_\_\_

### Past Medical History

List any serious childhood illnesses you had: \_\_\_\_\_  
 Were you born prematurely? Yes \_\_\_ No \_\_\_ What did you weigh at birth? \_\_\_\_\_  
 Did you have growth problems? Yes \_\_\_ No \_\_\_ Did you have a birth injury? \_\_\_\_\_  
 As a baby: How old were you when you could sit alone? \_\_\_\_\_ Crawl? \_\_\_\_\_ Pull yourself up?  
 \_\_\_\_\_ Stand alone? \_\_\_\_\_ Walk alone? \_\_\_\_\_ Were potty trained?  
 \_\_\_\_\_  
 Were you sad or depressed or happy as a child? Sad \_\_\_\_\_ Happy \_\_\_\_\_  
 List any permanent physical or mental problems from childhood: \_\_\_\_\_  
 \_\_\_\_\_  
 As a child, did you have trouble sitting still in school? Yes \_\_\_ No \_\_\_ Did you have trouble learning in school? Yes \_\_\_  
 No \_\_\_ Did you have trouble keeping your mind on tasks as a child? Yes \_\_\_ No \_\_\_ Did you have trouble learning to  
 read? Yes \_\_\_ No \_\_\_ Did teachers complain you were too active? Yes \_\_\_ No \_\_\_  
 Check any serious illnesses you have now or have been treated for in the past.  

_____ Seizures (epilepsy)	_____ Depression
_____ Cancer	_____ Panic disorder
_____ Diabetes	_____ Nerves
_____ Thyroid disease	_____ Alcoholism
_____ Anemia (low blood)	_____ Drug abuse
_____ High blood pressure	_____ Overdoses of medication
_____ Heart disease	_____ Suicide attempts
_____ Lung or breathing problems	_____ Violence toward others
_____ Joint or back disease	_____ Attention deficit disorder
_____ Stomach or bowel disease	_____ Learning disorder
_____ Female problems	_____ Manic-depressive illness (bipolar)
_____ Pregnancy problems	_____ Schizophrenia
_____ Urinary tract problems	_____ Eating disorders (e.g., anorexia)

- Sexual problems
- Prostate problems
- Sleep problems
- HIV or AIDS
- Neurological disease
- Spouse abuse
- Child abuse or neglect
- Pain disorder

If you were hospitalized for any of these illnesses, list the hospital(s): \_\_\_\_\_

Have you been injured in any motor vehicle accidents? Yes \_\_\_ No \_\_\_ If yes, list below:

Year	Your Age at the Time	Type of Injury	Treatment/by Whom

Have you ever been knocked out or had a brain injury? Yes \_\_\_ No \_\_\_ If yes, describe what happened:

\_\_\_\_\_

Have you ever been in a coma? Yes \_\_\_ No \_\_\_

Have you ever broken any bones? Yes \_\_\_ No \_\_\_ If yes, describe which bones were broken, right or left side:

\_\_\_\_\_

Have you had any surgeries or operations? Yes \_\_\_ No \_\_\_ If yes, list below:

Year	Your Age at the Time	Hospital Where Performed	Type of Surgery

Are you now taking any medications? Yes \_\_\_ No \_\_\_ If yes, please list below the milligrams and how often you take your medicine.

Medications	Milligrams	Times Per Day

Are you taking any over-the-counter medicines (you do not need a prescription)? Yes \_\_\_ No \_\_\_ If yes, list them:

\_\_\_\_\_

Are you taking any herbs or natural products? Yes \_\_\_ No \_\_\_ If yes, list them:

\_\_\_\_\_

Who keeps track of your medications? You \_\_\_ Your spouse \_\_\_ Someone else \_\_\_\_\_

Do you have any drug allergies or reactions? Yes \_\_\_ No \_\_\_ If yes, list below:

Drugs	Allergic Reaction
	(rash, nausea, hives, etc.)

Do you use tobacco now? Yes \_\_\_ No \_\_\_ Not now, but previously \_\_\_ If you answered yes or have used tobacco in the past, please describe how much, when you started, and when you stopped:

\_\_\_\_\_

Do you use alcohol now? Yes \_\_\_ No \_\_\_ Not now, but in the past \_\_\_ If yes to any use of alcohol, then describe:

\_\_\_\_\_

Type of alcohol you currently use (whiskey, beer, wine, etc.): \_\_\_\_\_

Number of alcohol drinks you have per day: \_\_\_\_\_

When did you first start using alcohol? When did you stop? \_\_\_\_\_

Describe any past alcohol problems in your life (DUIs, AIs [alcohol intoxication arrests], alcoholism, etc.): \_\_\_\_\_



Describe any medical treatment for alcohol problems: \_\_\_\_\_

Have you ever taken a medication or drug that you bought off the street? Yes \_\_\_ No \_\_\_

If yes, describe: \_\_\_\_\_

Have you ever used illegal drugs (i.e., marijuana, heroin, cocaine, uppers, downers, crack, etc.)? Yes \_\_\_ No \_\_\_

Have you ever sniffed paint, glue, or gasoline to get high? Yes \_\_\_ No \_\_\_ If yes, what did you sniff and how long?

\_\_\_\_\_

Have you ever used LSD, peyote, mescaline, PCP, mushrooms? Yes \_\_\_ No \_\_\_ If yes, what and when?

\_\_\_\_\_

Have you ever used Ecstasy or "designer drugs?" Yes \_\_\_ No \_\_\_

Have you ever used illegal intravenous drugs (IV drugs)? Yes \_\_\_ No \_\_\_

Have you ever received treatment for drug/substance abuse? Yes \_\_\_ No \_\_\_ If yes, what hospital and what year?

\_\_\_\_\_

Do you drink coffee or tea? Yes \_\_\_ No \_\_\_ How many cups per day? \_\_\_\_\_ Do you drink caffeinated soft drinks? Yes \_\_\_ No \_\_\_ What soft drinks? \_\_\_\_\_ How many per day? \_\_\_\_\_

### ***Next Five Questions for Women:***

1. How many pregnancies have you had? \_\_\_\_\_ How many living children have you had? \_\_\_\_\_ How many miscarriages have you had? \_\_\_\_\_

2. Were you depressed after having a baby or miscarriage? Yes \_\_\_ No \_\_\_ If yes, when? \_\_\_\_\_ Were you medically treated? Yes \_\_\_ No \_\_\_

3. Have you had any babies by cesarean section? Yes \_\_\_ No \_\_\_

4. Could you be pregnant? Yes \_\_\_ No \_\_\_

5. When was your last menstrual period? \_\_\_\_\_

### **Past Psychiatric History**

Have you ever been hospitalized for psychiatric, drug abuse, alcohol, or mental problems? Yes \_\_\_ No \_\_\_ If yes, please explain below:

<b>Psychiatric Hospital Admissions</b>	<b>Year Hospitalized</b>	<b>Hospital Name</b>	<b>Treating Physician or Psychiatrist</b>	<b>Diagnosis or Reason for Admission</b>	<b>Type of Treatment Received</b>
1st admission					
2nd admission					
3rd admission					
4th admission					

Have you ever been discharged from any hospital against medical advice (AMA)? Yes \_\_\_ No \_\_\_ If yes, describe:

\_\_\_\_\_

Have you ever been prescribed any form of nerve medicines, antidepressants, tranquilizers, or other psychiatric medications? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

When is the first time in your life you ever took nerve medicines, tranquilizers, or antidepressants?

\_\_\_\_\_

Have you ever stopped nerve pills without asking the doctor? Yes \_\_\_ No \_\_\_

Have you ever had shock treatments (electroconvulsive therapy)? Yes \_\_\_ No \_\_\_ If yes, describe when and where:

\_\_\_\_\_

Have you ever been advised by any doctor or health practitioner to get mental or psychological treatment? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Have you ever been legally committed or admitted involuntarily to a mental hospital or psychiatric unit? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Have you ever refused mental treatment when recommended by a doctor? Yes \_\_\_ No \_\_\_ If yes, describe:

\_\_\_\_\_

Have you ever received any type of office treatment by your family doctor, psychiatrist, psychologist, or therapist (medication, counseling, therapy) for any nervous condition or psychological, psychiatric, family, or marital problem? Yes \_\_\_ No \_\_\_ If yes, describe:

Year of Treatment	Treating Therapist/Physician	Diagnosis or Problem	Type of Treatment (e.g., Drugs, Therapy)

Have you ever intentionally overdosed yourself on drugs or medicines? Yes \_\_\_ No \_\_\_ If yes, describe:

Have you ever attempted to take your life? Yes \_\_\_ No \_\_\_ If yes, describe:

Have you ever intentionally cut, burned, or disfigured yourself? Yes \_\_\_ No \_\_\_ If yes, describe:

Did you set fires as a child? Yes \_\_\_ No \_\_\_

Did you harm or kill animals as a child? Yes \_\_\_ No \_\_\_

### Family History

Please check if any of these illnesses or acts have occurred in any of your parents, brothers, sisters, or children (do not list grandparents, aunts, uncles, or cousins):

- |  |   |
|--|---|
| <input type="checkbox"/> High blood pressure               | <input type="checkbox"/> Nervous breakdown                            |
| <input type="checkbox"/> Thyroid illnesses                 | <input type="checkbox"/> Mental illness/nerve problems                |
| <input type="checkbox"/> Diabetes                          | <input type="checkbox"/> Depression                                   |
| <input type="checkbox"/> Cancer                            | <input type="checkbox"/> Alcohol/drug problems                        |
| <input type="checkbox"/> Heart disease                     | <input type="checkbox"/> Eating disorders (anorexia nervosa, bulimia) |
| <input type="checkbox"/> Lung disease                      | <input type="checkbox"/> Attention deficit disorder                   |
| <input type="checkbox"/> Kidney disease                    | <input type="checkbox"/> Learning disorder                            |
| <input type="checkbox"/> Liver or gastrointestinal disease | <input type="checkbox"/> Suicide                                      |
| <input type="checkbox"/> Seizures (epilepsy)               | <input type="checkbox"/> Killing another person                       |
| <input type="checkbox"/> Neurological disease              | <input type="checkbox"/> Violence toward others                       |
| <input type="checkbox"/> Alzheimer's disease               | <input type="checkbox"/> Child abuse                                  |
| <input type="checkbox"/> Strokes                           | <input type="checkbox"/> Spouse abuse                                 |
| <input type="checkbox"/> HIV or AIDS                       |   |

If you checked any of the above, please explain which relative had the illness or performed the violent act:

If father alive, his age: \_\_\_\_\_ If mother is alive, her age: \_\_\_\_\_

If a relative is dead, list what your father, mother, brother, sister, or child died of and his or her age at death:

### Social History

Where were you born? \_\_\_\_\_

Date of birth: \_\_\_\_\_

Of your siblings, how many sisters? \_\_\_\_\_ Brothers? \_\_\_\_\_ Where do you come in the family (first child, last child, etc.)?

What did your father do for a living? \_\_\_\_\_

What did your mother do for a living? \_\_\_\_\_

Did your family have enough money? \_\_\_\_\_ Not enough money? \_\_\_\_\_ Live in poverty? \_\_\_\_\_

Is your father living? \_\_\_\_\_ Year he died? \_\_\_\_\_ Your mother? \_\_\_\_\_ Year she died? \_\_\_\_\_

Are (were) your parents divorced? \_\_\_\_\_ If yes, when? \_\_\_\_\_ How old were you at the time? \_\_\_\_\_

Who raised you? \_\_\_\_\_ Did your parent(s) own your home? Yes \_\_\_ No \_\_\_

Was your home life happy? Yes \_\_\_ No \_\_\_ Abusive? Yes \_\_\_ No \_\_\_ Threatening? Yes \_\_\_ No \_\_\_ Hard on you? Yes \_\_\_ No \_\_\_ Make you depressed? Yes \_\_\_ No \_\_\_

Did your father abuse your mother? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever been sexually abused? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever been raped? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever been physically abused? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Are you presently being sexually or physically abused by anyone? Yes \_\_\_ No \_\_\_ If yes, who? \_\_\_\_\_

Have you ever been violent to or harmed a person? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever shot, stabbed, or beaten another person? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever threatened to kill another person? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever torn up property? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever killed another person, even if by accident? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Are there guns in your home? Yes \_\_\_ No \_\_\_ If yes, what type or caliber (e.g., .357 handgun, 12-gauge shotgun)? \_\_\_\_\_

Have you ever been in legal trouble for your sexual behavior? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Have you ever sexually abused or harassed a child or adult? Yes \_\_\_ No \_\_\_ If yes, explain: \_\_\_\_\_

Highest grade you *completed* in school? \_\_\_\_\_

If you did not finish high school, what was the reason you quit? \_\_\_\_\_

What were your grades in high school? \_\_\_\_\_ Did you require special education classes? Yes \_\_\_ No \_\_\_ In grade school or high school, did the teachers think you were hard to control or was it hard to get your attention? Yes \_\_\_ No \_\_\_

If yes, explain: \_\_\_\_\_

If you attended any college or trade school, list degree, diploma, date of graduation, and college/university/trade school you attended:

Degree/Diploma/Major	Dates of Graduation	College/University/Trade School

How many times have you been married? \_\_\_\_\_.

How many times have you been divorced? \_\_\_\_\_.

Are you now divorced? \_\_\_\_\_ Married? \_\_\_\_\_

How long have you been divorced or married? \_\_\_\_\_

Please complete:

Marriage	Year Married	Year Divorced	Spouse's Name	Any Natural Children and Their Ages	Reason for Divorce
First marriage					
Second marriage					
Third marriage					
Fourth marriage					

How many natural children do you have? \_\_\_\_\_

How many stepchildren do you have? \_\_\_\_\_

How would you describe your marriage, if you are presently married? Good relationship \_\_\_ Fair relationship \_\_\_

Bad relationship \_\_\_ Terrible or abusive relationship \_\_\_

If you are not married but have a lover, describe your relationship. Good \_\_\_ Fair \_\_\_ Bad \_\_\_ Terrible or abusive \_\_\_

Describe your relationship with your children. Close \_\_\_ Could be better \_\_\_ Distant \_\_\_ Poor \_\_\_

If you do not have a relationship at this time, how do you feel about this? Satisfied \_\_\_ Lonely but OK \_\_\_ Not satisfied and want a relationship \_\_\_ Very sad or lonely \_\_\_

## Legal History

Have you ever been in prison or jail? Yes \_\_\_ No \_\_\_ If yes, where and when?

Have you had any criminal felony or misdemeanor convictions, drug arrests, DUIs, or public intoxication arrests? Yes \_\_\_ No \_\_\_ If yes, fill in below:

Arrest Date	Charge(s)	Where (City or State)	Were You Convicted?	Length of Time in Prison/Jail

Have you been involved in any lawsuits as either the plaintiff or defendant? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Has your spouse or anyone else ever gotten a restraining order or emergency protective order against you? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Have you ever gotten a restraining order or emergency protective order against your spouse or anyone else? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Have you ever been charged with spouse abuse, child abuse or neglect, or terroristic threatening? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Have you ever filed a workers' compensation claim? Yes \_\_\_ No \_\_\_ If yes, what was (were) the work injury(ies)? \_\_\_\_\_

Have you ever been declared bankrupt? Yes \_\_\_ No \_\_\_

## Employment/Vocational History

Employment status (check one): Full-time \_\_\_ Part-time \_\_\_ Not employed \_\_\_ Student \_\_\_ If not employed, reason you are not presently employed: \_\_\_\_\_

If presently employed, who is your employer? \_\_\_\_\_

Employer address: \_\_\_\_\_

Describe your job duties: \_\_\_\_\_

Length of time on this employment: \_\_\_\_\_

If you are presently disabled, year of and reason for your disability: \_\_\_\_\_

What are your present sources of all monthly income? \_\_\_\_\_

Were you ever fired or asked to resign from employment? Yes \_\_\_ No \_\_\_ If yes, reason: \_\_\_\_\_

Have you ever threatened an employer or a coworker? Yes \_\_\_ No \_\_\_

Where is your spouse presently employed? \_\_\_\_\_

If you are not working, do you plan to return to work at anytime in the future? Yes \_\_\_ No \_\_\_

List past employment below (beginning with your most recent job):

Employer	Job Title	Start Date	Finish Date	Reason for Leaving	Other

## Military History

Have you ever tried to enter military service or a service academy (e.g., Naval Academy, West Point)? Yes \_\_\_ No \_\_\_

Were you ever turned down for military service? Yes \_\_\_ No \_\_\_

If you have had any military service, list below:

Branch of Service	Years Served	Rank at Time of Discharge	Type of Discharge	Job Duties

Were there any disciplinary actions against you? Yes \_\_\_ No \_\_\_ If yes, describe: \_\_\_\_\_

Were you ever in the brig or stockade? Yes \_\_\_ No \_\_\_

Where was your basic training? \_\_\_\_\_

Where was your advanced training? \_\_\_\_\_

If you ever served in a combat zone, list year and area: \_\_\_\_\_

If wounded in military service, describe: \_\_\_\_\_

Describe any military pension or disability you may receive: \_\_\_\_\_

## Review of Systems

CIRCLE THOSE SYMPTOMS PRESENT.

**GENERAL:** Fever, shaking, chills, change in appetite, loss in weight, gain in weight, fatigue, change in sleeping patterns, soaking night sweats

Explain any circled items. If you have lost or gained weight, how many pounds in the last 3 months?

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**HEAD, EYES, EARS, NOSE, THROAT:** Headache, changes in vision, double vision, blurred vision, eye pain, excessive tearing, discharge from the eyes, changes in hearing, ringing in ears, ear pain, discharge from ears, nosebleeds, odd odors, hoarseness, dental pain, sore tongue, sore throat, mouth sores, trouble swallowing

Explain any circled items:

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**CHEST:** Cough, sputum production, shortness of breath, wheezing, blood in sputum, abnormal chest x-ray, positive TB test, lump(s) in breast, nipple discharge, nipple bleeding, breast pain

Explain any circled items:

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**HEART:** Chest pain with exercise, shortness of breath walking, shortness of breath upon lying down, heart murmur, rheumatic fever, shortness of breath that wakes you up at night, swelling in legs, fainting

Explain any circled items:

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**STOMACH, BOWEL:** Change in appetite, nausea, vomiting, blood in vomit, dark brown vomit, diarrhea, constipation, change in stool size, blood in stool, dark black tarry-colored stool, food intolerance, trouble swallowing, heartburn, indigestion, laxative use, excessive gas, abdomen pain, weight loss, weight gain

Explain any circled items:

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**URINARY, GENITAL:** Trouble starting urination, excessive urination, dribbling of urine, pain upon urination, blood in urine, excessive urination after going to bed, unable to hold urine, bed-wetting, sores on genitals

Explain any circled items:

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FEMALE: Menstrual irregularity, premenstrual distress, menopause symptoms, excessive female bleeding  
Explain any circled items:

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MENTAL: Do you have a present plan to kill yourself? Yes \_\_\_ No \_\_\_ Do you have a plan to kill someone else? Yes \_\_\_ No \_\_\_

Depression, sadness, nervousness, panic, thoughts of suicide, poor concentration, loss of memory, too happy, word-finding difficulty, confusion, inability to know month/year, hearing voices, seeing things, paranoid thoughts, irritability, excessive anger, arguing, crying for no reason, trouble thinking, flashbacks, thoughts of killing another person, counting things, checking things, afraid of germs, afraid to touch doorknobs, wash hands more than 10 times daily, take more than 2 baths or showers daily

Explain any circled items:

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NEUROLOGIC: Blackouts, seizures, double vision, partial blindness, headaches, numbness, tingling, weakness, poor balance, shaking or tremors, abnormal movements of face or body, poor coordination, paralysis, loss of reflexes, pain

Explain any circled items:

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MUSCLE SKELETAL: Muscle spasms, joint pain, bone disorders, difficulty walking, difficulty sitting, difficulty using hands, difficulty bending, difficulty lifting

Explain any circled items:

---

SLEEP: How many hours do you sleep at night? \_\_\_\_\_ How many days weekly do you nap? \_\_\_\_\_

Cannot fall asleep, cannot stay asleep, wake up too early, fall asleep anytime, night terrors, nightmares, sleep walking, restless legs before sleep, cannot stay awake during or while sitting, severe snoring that bothers others, choking during sleep, cannot stay awake to drive, others have observed you to stop breathing during sleep, fall or stagger if angry or laugh, hear things when falling asleep or waking up, paralyzed for short time after waking up

Explain any circled items:

---

SEXUAL: Men: Cannot get erection, cannot ejaculate, ejaculate too soon, no sexual desire, partner does not meet my needs  
Women: Cannot lubricate, cannot have orgasm, no sexual desire, partner does not meet my needs

How many times per month do you engage in sexual activity with another person or a spouse? \_\_\_\_\_

Explain any circled items:

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HIV: Have you been tested for HIV? Yes \_\_\_ No \_\_\_ Results if tested: Positive \_\_\_\_\_ Negative \_\_\_\_\_

### Authorization Information

I authorize Forensic Doctor, M.D., to examine and test me. (If you are under 18 years of age, your parent or guardian must sign this form.)

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

I authorize Forensic Doctor, M.D., to send a copy of this evaluation to the person or agency who requested me to be examined or to those parties involved in my case.

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Signature

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Date

I authorize the licensed or certified psychologists consulting to Forensic Doctor, M.D., to perform whatever psychological testing Forensic Doctor, M.D., thinks is necessary to evaluate me.

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Signature

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Date

By my signature, I certify all statements I answered on this questionnaire are true and accurate.

---

Signature

---

Date

If this form was filled out by someone other than you, please give name: \_\_\_\_\_  
Relationship to you (spouse, friend, parent, guardian, etc.): \_\_\_\_\_

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# 11 Forensic Neurobehavioral Analysis Following Traumatic Brain Injury

## INTRODUCTION

In [Chapter 8](#), the neurobehavioral analysis was directed at analyzing data from the neuropsychiatric examination in order to develop treatment planning strategies and tactics for one's patient. For a forensic examination, the focus is entirely different. While many of the elements of the neurobehavioral analysis are exactly the same as those within a clinical context, the philosophical approach is quite different. The reader, at this point, may want to review the critical differences between a clinical and forensic assessment of brain injury as described in [Chapter 9](#). Forensic analysis lends itself following brain trauma primarily to determining causation and to the delineation of damages, if they have occurred, in the brain-injured person. The analysis of the data is exactly the same whether the examiner is functioning as an agent for the plaintiff lawyer or as an agent for the defense lawyer. Honesty and objectivity must prevail at all times. The goal of forensic neurobehavioral analysis following traumatic brain injury is to provide a forensic database upon which an examining expert may rely to provide helpful and appropriate testimony at trial or settlement, if requested.

## ANALYSIS OF THE DATA FOLLOWING TRAUMATIC BRAIN INJURY

### THE POLICE RECORD OR INJURY REPORT

[Table 11.1](#) outlines a suggested schema for collecting and analyzing data when providing a neuropsychiatric evaluation following traumatic brain injury. Most traumatic brain injuries occur in locations requiring a police investigation or at work sites requiring an employer's report of injury. There are, of course, exceptions. Whatever the exception, the forensic examiner should make every effort to get a report of the initial investigation or a report of injury.

With regard to the police record, this forms the basis for the initial gathering of factual information regarding the nature of the accident. For instance, in issues of brain injury associated with vehicular accidents, the police report generally is done in a technical style, and most police departments throughout the U.S. have significant standardization of reporting of motor vehicle accidents. While there may be no specific medical facts within the police record, the forensic examiner should carefully review this record to determine the nature of the trauma, and whether the police officer observed the examinee to have been injured at the scene. Most police traffic accident reports have a location on the document that indicates the type of injury the examinee sustained, whether the examinee was wearing a restraint device, how the accident scene was disposed, who transported the examinee from the accident site, and whether the vehicle remained in service. This information can be quite important with regard to causation. For instance, if the

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**TABLE 11.1****Schema for Forensic Neuropsychiatric Data Collection and Review**

Police Report:	This document is important to lawyers as it aids in establishing liability. For the forensic examiner, it aids in establishing causation of brain injury. Review for contributing factors: drugs, alcohol, violence, rape, etc.
Photographs:	If the examinee was injured in a motor vehicle accident, what is the extent of obvious force applied to the victim's vehicle? Were photographs of the examinee's body obtained? What is the visual evidence of the trauma?
Ambulance Report:	Review for GCS, RTS, and documentation of apparent injury. Did the GCS or RTS improve or worsen during transport? Was intubation required?
Emergency Department:	What were the GCS and RTS? Does neuroimaging aid in establishing causation? Do focal neurological signs or mental status changes aid in establishing causation? Was hospitalization required? If discharged from the E.D., were head injury instructions given? Was a follow-up with a neurologist or neurosurgeon ordered?
Hospital Record:	Was ICU required? Is there evidence of respiratory failure? Were secondary injuries present (e.g., hypovolemia, blood loss, cardiac or lung contusion, organ trauma or failure, etc.)? Was assisted ventilation required? Were neuronal salvage medications administered?
Rehabilitation Record:	What was the "Rancho" score at discharge? Was there evidence of cognitive/behavioral impairment? Was speech/language therapy required? Could the examinee complete ADLs by discharge? Was neuropsychological assessment provided?
Outpatient Record:	Is there evidence of posttraumatic seizures, headaches, or hypersomnolence? Is focal neurologic dysfunction present? Was neuropsychological assessment obtained? Was the examinee independent in ADLs? Was speech/language therapy continued? Was psychiatric treatment required? Was there evidence of family or caregiver stress?
School Record:	An important marker of damages in the child is alteration or reduction in school performance. Was an individual education plan required after brain injury? Was an educational diagnostic evaluation required?
Preinjury Records:	These are important for determining damages due to intellectual changes, reduction in employability, change in work product, reduction of school performance, or requirement for added medical treatments. These are also important in order to establish preinjury conditions that may have been aggravated (e.g., learning disorder, ADHD, PTSD, etc.) or that contribute to a poorer outcome (e.g., Alzheimer's disease, prior brain injury, prior psychiatric illness, substance abuse, diabetes, etc.).

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police record indicates that the level of damage to the vehicle was extremely slight and the vehicle remained in service and was driven away from the accident site, this information is consistent with a low likelihood of a severe injury in most people. In contrast, the police record may also indicate severe and extensive damage to the examinee's vehicle and may state that mechanical extrication was required to release the examinee from the vehicle for transport to hospital.

For those examinees injured at work, police reports generally are not used. The employer is required to generate some documented record of the injury in keeping with workers' compensation statutes. The employer's report of injury will generally give a factual description of the nature of the injury, the location of the examinee when found following the injury, and the apparent level of injury to the examinee at the time the individual was removed from the work site for medical evaluation. Likewise, this document can be quite revealing in the examiner's attempts to determine causation and level of injury. For instance, it is not unusual to see within the employer's report of injury that the individual was not removed from the work site, but was evaluated by the company nurse and allowed to return home; no further medical care may have been given. When an examinee alleges significant brain trauma at a later date, the employer's report may reveal inconsistencies with the examinee's allegations. On the other hand, for those workers who have been significantly injured, the employer's record generally will adequately document the level of and nature of the

injury. Both the police record and the employer's injury report — or any other initial report of injury — will assist the physician conducting a neuropsychiatric examination, where called upon to give forensic testimony, by providing a clear document upon which to base a decision about causation of an apparent brain injury.

### **EMERGENCY MEDICAL SERVICES RECORD**

In most accidents of any apparent significant nature, an ambulance or emergency medical services squad will be called to the accident scene. The American College of Surgeons first developed criteria for the establishment of trauma centers in the U.S. and developed a trauma system regionalization.<sup>1</sup> The Joint Section of Trauma of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons supported these criteria and further endorsed the establishment of regional neurotrauma centers.<sup>2</sup> Where a systemized approach to trauma care has been developed, studies have subsequently shown a marked reduction in the percentage of preventable deaths from trauma.<sup>3,4</sup>

As previously discussed in [Chapters 1](#) and [8](#), the emergency medical services record usually contains the first documented record of the *Glasgow Coma Scale* (GCS). Most emergency medical service personnel or ambulance services in the U.S. currently use the GCS.<sup>5</sup> Review of the GCS may or may not be useful in determination of causation. As has been previously outlined in this text, a person can sustain a mild brain injury and still present at the accident scene with a GCS score of 15. On the other hand, evidence of a GCS score of less than 15 is consistent with the potential for having sustained a traumatic brain injury. Evidence of reduced scores on the GCS may provide confirmatory evidence of brain injury, consistent with what was reported in the police report or other injury reports. However, this is not medical evidence, since the initial Glasgow Coma Scale score at the scene is rarely measured by a physician.

The forensic examiner should review further the ambulance report or emergency medical services report for evidence of the Revised Trauma Score (RTS) (see [Chapters 1](#) and [8](#)). This provides information to the forensic examiner regarding observations made at the accident scene by emergency personnel and will help the examiner understand the apparent level of trauma at the time the examinee was first attended at the accident site. For more severely injured persons, or individuals injured so severely that time of transport is a significant issue, helicopter transport is now routinely used and the flight record should be reviewed carefully in those instances.

### **EMERGENCY DEPARTMENT RECORDS**

In almost all instances, the GCS will be repeated again when the injured person arrives in the trauma center or emergency department. It is important to carefully examine the scores and compare those obtained by the emergency medical services to those obtained in the emergency department. In a deteriorating patient, the scores clearly will decline. On the other hand, most persons who have sustained a mild head injury will demonstrate an improvement in GCS scores during transportation to the emergency department. If there is any significant clinical indication that the person sustained a brain injury, a computerized tomography (CT) scan of the head will be ordered. The forensic examiner should review CT reports from the emergency department, as these generally will reveal pathology if present. It must be remembered, however, in those patients who are admitted to the hospital, that serial CT head scans often are obtained due to the potential evolution of intraparenchymal injury over time. The emergency department CT scan (see [Chapter 5](#)) will in most instances help delineate epidural hematomas, subdural hematomas, contusions, intracerebral hematomas, and diffuse axonal injuries. If the CT of the head is unrevealing, and clinical evidence indicates that the person has sustained a mild head injury, it is likely that the individual will be discharged from the emergency department. Therefore, the forensic examiner will have to secure outpatient records.

The emergency department record, in those instances where patients are evaluated and released, should be scrutinized to determine if the person was sent home with a head injury warning discharge instruction. If there is no evidence in the emergency department record that an accident victim was sent home with such a warning, it is reasonable to conclude that there was no substantial evidence of trauma to the head. Individuals with obvious or suspected brain trauma will be admitted to the hospital.

## **THE HOSPITAL RECORD**

Review of the hospital record was covered in some detail in [Chapter 8](#). As discussed in [Chapter 1](#), victims of head trauma often sustain secondary injury. The forensic examiner should carefully review the hospital record to determine whether the individual was sent to a trauma floor or required neurosurgical intensive care monitoring. By this time in the trauma victim's treatment, it should be clear whether focal neurological signs are present. For instance, is there evidence of hemiparesis, spasticity, or bone fractures? Did the examinee require ventilator support or tracheostomy? Was intracranial surgery or cerebral pressure monitoring required? What ancillary specialists were called to assist in the management of the patient? Is there evidence in the record of pulmonary contusion, myocardial contusion, viscous injury, or other injuries concurrent with head trauma?

It is particularly important for the forensic examiner to scrutinize nursing records. The nursing records often contain more detailed information about the person's day-to-day mental capacity during hospitalization than physician records. Neurosurgeons and trauma surgeons are more concerned with life-threatening posttraumatic issues, and their observations are oftentimes less specific regarding the patient's mental state than the nurses' observations. The specific content of the hospitalization record may correlate poorly with eventual neuropsychiatric outcome, but the forensic value of these records lies in their ability to assist the neuropsychiatric examiner in focusing the examination into areas that may prove or disprove medical damages.

## **REHABILITATION RECORDS**

If the patient is referred for rehabilitation following hospitalization, this is usually a significant marker of injury. In most instances, the neurosurgeon, or other ancillary trauma personnel, has determined that the examinee's cognitive function and level of independent skills warrant further inpatient treatment. From a forensic standpoint, the rehabilitation records are where the examiner will generally find the first evidence of a substantial cognitive assessment following the injury. [Chapter 8](#) has outlined methods of cognitive rehabilitation, and the reader may want to review these at this point. A careful analysis of the rehabilitation record will assist the examiner in determining results from speech and language therapy, physical therapy, occupational therapy, and other assessments and treatments provided during the rehabilitation process. These are important data points that the forensic examiner can use to determine the level of recovery in the examinee at the time of the rehabilitation examination. The Rancho Scale was discussed in [Chapter 1](#); its use will be documented in most rehabilitation units in the U.S.

It is important to establish within the rehabilitation records whether significant levels of agitation were present, as these are predictive of future cognitive complications. Moreover, the rehabilitation record is probably the first instance where the examinee was treated with psychotropic agents, and a review of their pharmacology is warranted. The record should be reviewed to determine the presence of spasticity, swallowing or feeding disorders, nutritional status, bowel and bladder difficulties, respiratory impairment, orthopedic impairments, neuroendocrine disorders, dysautonomia, and other possible complications as a result of the trauma sustained by the examinee.<sup>6</sup>

## **THE NEUROPSYCHOLOGICAL RECORD**

Neuropsychological assessment following traumatic brain injury generally first occurs either in the rehabilitation unit or, later, in outpatient treatment when requested by a neurologist or psychiatrist.

The forensic examiner should be aware, as was cautioned in [Chapters 6 and 9](#), that effort measures probably were not used at the time of the neuropsychological assessment. The assessment during rehabilitation will have been performed for clinical and therapeutic reasons. It is important for the forensic examiner to determine whether the neuropsychological assessment was performed prior to the filing of a lawsuit on behalf of the examinee. If the neuropsychological assessment was performed prior to the lawsuit, this may be the best cognitive evidence of the examinee's mental state during the recovery period following brain trauma. If it was performed after the filing of a lawsuit, and no effort controls were in place, then it may not be the best of cognitive data. Depending upon the legal context, it may offer the forensic examiner a neuropsychological standard to compare with the present neuropsychiatric examination for determination of levels of improvement and recovery. It is important to recognize that a neuropsychological evaluation performed during the recovery phase will not be fully capable of determining outcome from a head injury. As has been discussed earlier in this text, recovery generally does not plateau following traumatic brain injury until 6 to 18 months postinjury, depending on the individual case. It is also important to determine whether all cognitive domains were assessed at the time the neuropsychological evaluation was completed. In many instances, neuropsychologists during the recovery phase will perform a limited examination due to the level of impairments of the examinee.<sup>7</sup>

## OUTPATIENT TREATMENT

Here, the forensic focus should be upon psychiatric and neurological treatment. On the other hand, it is important not to overlook other important treatments that may occur following rehabilitation. These include physical therapy, speech and language therapy, and outpatient cognitive rehabilitation. The forensic examiner should focus particularly upon psychiatric or psychological treatment if it is undertaken following brain injury. There are two reasons for this. The first reason is to discover whether, within the psychiatric–psychological database, there is evidence that the person had a preinjury psychiatric state that may currently play a role in the genesis of psychiatric symptoms (e.g., antisocial personality disorder) or has been exacerbated by a traumatic brain injury (e.g., attention deficit disorder). The second reason speaks to damages assessment. The outpatient's psychiatric record, if this is the first psychiatric treatment for the examinee, will help the examiner delineate psychiatric symptoms specifically related to the traumatic brain injury. The neurological record should be closely examined in a similar vein. For instance, is there evidence in the neurological intake history of preinjury seizure disorder or other neurological condition that may place a burden upon the traumatic brain injury, or account for the current symptoms in a case where the head injury was very mild and there is little evidence of a traumatic brain injury? The examiner must remember that oftentimes when a person is traumatically brain-injured and provides a postinjury history to a treating physician, that history may be quite different than the history presented to the neuropsychiatric examiner after the examinee has filed a lawsuit. For those examinees who have new neurological symptoms following traumatic brain injury, the outpatient neurological record will be very helpful to the neuropsychiatric examiner in the delineation of posttraumatic issues such as seizures, migraine headaches, focal neurological deficits, or other neurological signs and symptoms. With regard to speech and language therapy, some brain-injured persons require this therapy for a year or more following a brain injury where there has been a substantial impact upon language and speech systems. The outpatient record will be very helpful to the forensic examiner in this regard. From a forensic standpoint, the speech and language record should be scrutinized to determine if the speech and language pathologist documented a congenital speech and language disorder or reported a preinjury disorder from review of academic records. The physical therapy record will assist the neuropsychiatric examiner to determine continuing posttraumatic issues such as contractures, therapy for hemiparesis, training for transfers, and other issues that may affect the traumatically brain-injured person who has also sustained a substantial physical injury.



## NEUROPSYCHIATRIC EXAMINATION

As neuropsychiatric examiners analyze their own data, they should first focus upon the significant major categories of the examination and then analytically determine whether there is in fact evidence of a neuropsychiatric injury. The history will contain two major elements: (1) the history reported by the examinee, and (2) the history developed by the neuropsychiatric examiner from the data sources mentioned above, as well as from collateral sources, preinjury medical records, academic and employment records, legal records, and depositions taken in the case, if the examination is within the context of a lawsuit.

The examiner should review her own history and determine if the elements associated with a traumatic brain injury, as described in [Chapter 3](#), are present. If they are, of course, the neuropsychiatric examination will be directed toward evaluating these historical elements. On the other hand, the history also is important from a forensic standpoint to delineate whether the expressed symptoms of the examinee are truly consistent with a brain injury. As noted in Case 1, a significant disparity in symptoms expressed by the examinee may not comport with objective data from the examination, and this may lead to a determination of symptom magnification or malingering. The mental status examination is a core feature of the neuropsychiatric examination. Its forensic purpose is to focus the examiner, during a face-to-face examination of the examinee, upon elements that are predictive of organic mental dysfunction. These then will require further elucidation by objective testing techniques. The neurological examination has great forensic importance (see Case 1), and where findings are inconsistent with brain injury, their forensic importance cannot be underestimated. On the other hand, positive focal neurological findings consistent with traumatic brain injury will affirm the history and mental status data if an actual brain injury is present, but absence of focal neurological signs does not rule out injury.

Whereas brain imaging may not be significantly utilized in many clinical examinations for brain injury or, for example, when a physician is treating a brain-injured person who has already been imaged, in a forensic situation imaging may prove highly important. For instance, a negative magnetic resonance imaging (MRI) or single photon emission computed tomography (SPECT) scan, in the face of mental status findings inconsistent with brain injury and neuropsychological findings inconsistent with brain injury, provides powerful medical evidence that no significant brain injury occurred, even though there may have been a head injury. On the other hand, in a truly injured person, from a forensic standpoint, brain imaging is the only portion of the neuropsychiatric examination where the examinee can have no direct influence upon the outcome of the results. An examinee can give incorrect history, distort the mental status examination, provide poor cognitive effort on the neuropsychological examination, or demonstrate confusing findings on neurological examination. However, an examinee cannot modify how the MRI signal influences the imaging detectors, nor can the examinee consciously produce a focal perfusion deficit or hypometabolic area on a SPECT or positron emission tomography (PET) scan, respectively. The neuropsychological examination has been covered in great detail in [Chapter 6](#). Its use in a clinical examination is confirmatory rather than diagnostic, and the same is true in a forensic brain injury examination. However, in a person with an actual brain injury, there is no other single testing method capable of providing metrics of cognitive function other than appropriately administered neuropsychological tests. Historical information, mental status examination data, neurological examination results, and brain imaging studies cannot provide a measure of memory, attention, or other cognitive domains against a standardized database.

After the neuropsychiatric examiner has completed the historical, mental status, neurological, brain imaging, and neuropsychological portions of the examination, an analysis of the data then proceeds in a manner similar to that discussed in the cases below. This will enable the forensic examiner to provide causation and damage analyses.

## COLLATERAL HISTORY SOURCES

During a forensic neuropsychiatric examination for brain injury, collateral sources can be most helpful. This, of course, will be required if the examinee is a young child. Moreover, collateral

sources of information will be required if the examinee is so grievously injured that he or she cannot provide needed information. On the other hand, in cases where injury appears to be mild or nonexistent, the forensic examiner should be very cautious when using information from collateral sources, particularly if those persons have a vested interest in the outcome of the examination. A man claiming a brain injury with specious data to support his assertion may have as a collateral source his wife, who has a substantial vested interest in whether her husband receives a monetary award for the putative brain injury. The same can be said for the parents of a small child who may have limited evidence or an entire lack of evidence of a brain injury. The important issue of malingering by proxy has been discussed previously in this text and is worth reviewing at this time. A small child cannot competently be a witness at trial, but parents can, and the forensic examiner should be aware of this potential bias during examination and the taking of collateral history.

### **PREINJURY MEDICAL RECORDS**

It is very important to review these records in a forensic setting. They provide two important sets of databases. The first set will enable the forensic examiner to determine if prior medical issues are presently playing a role in the genesis of symptoms attributable to a brain injury. The opposite is clearly true as well. That is, the preinjury medical records may clearly establish that there was no evidence of any form of cognitive or psychiatric disorder present in the examinee prior to a brain injury discovered at the neuropsychiatric examination.

The preinjury medical records also may provide information for more subtle analyses. For instance, a preinjury MRI showing periventricular gliosis and ischemic changes in an examinee's brain prior to brain injury may be indicative of potential cognitive changes present prior to the alleged brain injury. They also may be markers for other brain-damaging diseases such as diabetes, hypertension, or collagen-vascular disorders. Thus, the preinjury medical records may prove very helpful in causation analysis and equally important in damages analysis.

### **ACADEMIC AND EMPLOYMENT RECORDS**

The use of academic records has been discussed previously in this text. Within a forensic context, academic records may prove much more useful than in a clinical setting. If the examinee is 16 years of age or older, ACT and SAT scores may be found within the academic records. These are very useful for assistance in establishing a preinjury cognitive baseline. Both ACT and SAT scores can enable the psychologist to provide the neuropsychiatric examiner with information regarding the examinee's preinjury cognitive function measured against a national database. On the other hand, one should not place too much reliance upon grades. Grades are subject to significant distortion from issues of motivation, substance abuse, learning disorder, and childhood psychiatric illnesses such as attention deficit disorder. Where possible, it is recommended that the forensic examiner make an attempt to obtain academic records on any person being examined for traumatic brain injury in a forensic context, where those records are available. There often is a wealth of other useful information in the records. School records contain significant demographic variables about the family of origin; they may provide the examiner with psychological reports; they often will contain data regarding learning disorders or other medical conditions; and they may contain behavioral descriptions made by teachers and other educational professionals. This information can be very helpful in the examiner's attempt to determine issues of causation. Again, these data cut both ways. As learned previously in this text, the learning-disabled child is especially vulnerable to a traumatic brain injury. On the other hand, in a person with little or no evidence of brain injury, who is claiming severe attentional deficits, the finding of an attention deficit disorder diagnosis within the school records can be dispositive for explaining the current attentional deficits in the examinee.

Employment records are more difficult to obtain than academic records, in most instances. However, in a lawsuit, if a forceful argument is made for their use, a lawyer often can obtain these. They are particularly helpful when the examinee who may have sustained a brain injury is a

professional person. In this case, examples of work product of the examinee may be used to establish cognitive skills present prior to the alleged brain injury. For instance, if the examinee functioned as a neuropsychologist prior to a severe motor vehicle accident and is now claiming a brain injury, the redacted neuropsychological reports produced by the examiner prior to the alleged brain injury can be most useful for determining preinjury cognitive baseline. As another example, a lawyer who has been involved in a significant motor vehicle accident wherein brain injury seems highly likely may have significantly useful information about preinjury function contained within legal briefs or other pleadings made before courts. This would enable the forensic neuropsychiatric examiner to determine thinking style, language usage, abstract thinking, and other issues that can be developed from work products for postinjury comparisons.

## **LEGAL RECORDS**

Legal records are generally not reviewed in a clinical examination of a brain-injured patient. However, it is mandatory to ask for and review such records in a forensic neuropsychiatric evaluation. A number of issues are raised in a forensic situation that do not apply to a clinical situation. For instance, legal records are often excellent markers to determine whether antisocial tendencies are present. This can pose a significant dilemma for the examiner of a brain-injured person when that individual had a substantial antisocial personality disorder and then sustained an infraorbital traumatic brain injury that in and of itself may have produced a component of “acquired sociopathy.” The issue then may be exacerbation rather than causation. Furthermore, for the examinee who has no evidence of a brain injury but makes assertions that he does in fact have a brain injury, the legal records may be important to establish whether the individual is merely a litigious person. Repeated lawsuits without merit and multiple workers’ compensation claims without merit may provide evidence of this. Thus, legal records are extremely important in providing the examiner with preinjury evidence of aberrant behavior that may play a role in the determination of cognitive and behavioral effects following traumatic brain injury.

When the examiner obtains legal records, it is also wise to obtain workers’ compensation records, if they exist. Evidence of work-related injury may substantially enlarge the database for the forensic neuropsychiatric examiner and help him more accurately delineate causation. Moreover, where issues of injury apportionment are raised, workers’ compensation records may be extremely helpful in assisting with that analysis.

## **MILITARY RECORDS**

Similar to legal records, military records are rarely, if ever, sought for an evaluation in a clinical situation, unless the individual is an active-duty military person. Within the context of a forensic neuropsychiatric examination for brain injury, military records can be extremely helpful. Recall from [Chapter 10](#) that the Forensic Medical History Questionnaire asked substantial information about one’s military record. If there is evidence of prior disciplinary actions or discharges from the military service that were other than honorable, the examiner should secure the records, where possible. Again, the primary information to be gleaned from these records will be behavioral. Similar to legal records, military records are often prominent markers for aberrant behavior prior to a traumatic brain injury.

## **DEPOSITIONS**

The power of a deposition used within the context of a forensic neuropsychiatric brain injury examination has two major components: (1) It provides the examiner with sworn testimony; a history taken during a medical examination is not necessarily used as evidence at court; a deposition, however, is in fact evidence at court and is taken within the context of the functions of a court,

and (2) The deposition can provide the forensic examiner with a wealth of mental status examination. The cross-examination portion of the deposition or a deposition for discovery is particularly helpful. It will enable the examiner to evaluate memory elements, thought, abstracting ability, and auditory attention. Moreover, a careful analysis of the examinee's deposition will enable the forensic examiner to detect any inconsistencies in the neuropsychiatric history and examination, if they exist.

If depositions have been taken of individuals who were interviewed by the examiner on a collateral basis, (e.g., spouse or parent), it is wise to review these as well. Again, the examiner can see if the deposition information comports with the history given at the time of the collateral interview. Other important depositions to review include those of eyewitnesses of the behavior of the examinee on or about the time of the trauma. The examiner may find that eyewitness information describes an individual walking around at the scene, conversing with police officers, and providing information to others. At the time of the examination, the same individual may claim to the forensic examiner that he or she has no memory of the accident, was dysfunctional, and was unable to provide information.

### CAUSATION ANALYSIS

As an expert witness, the forensic examining physician can be expected to provide a causation opinion. In other words, what caused the brain injury claimed by the plaintiff (or the contrary, what is the basis to say that the plaintiff did not sustain a brain injury)? At this point, it might be wise to review the "Causation" section of [Chapter 10](#). [Table 11.2](#) provides further guidance to the records most likely to yield causation or damages data. Traditional analysis should probably begin with the "but for" test (i.e., "But for the negligence of the defendant, the plaintiff would have suffered no harm," or "A defendant's conduct is not a cause of the event if the event would have occurred without it"<sup>8</sup>). As discussed in [Chapter 10](#), this is an extremely oversimplified test. However, a lawyer generally begins with this rudimentary test of causation, and a forensic physician can do the same. If the examination data passes the "but for" test, the examiner also should consider whether there is an intervening cause. This complicated issue arises more often than one would suspect. The plaintiff is in motor vehicle accident A and a few months later is in motor vehicle accident B. By the time the individual is examined neuropsychiatrically, the date of the forensic examination comes after motor vehicle accident B. In this case, the forensic examiner must clearly establish whether there is an intervening cause for the brain injury in question. While these factors

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**TABLE 11.2**  
**Records Useful for Determining Causation or Damages**

Record	Primary Forensics Uses
Police report	Causation
Injury report	Causation
Emergency medical services	Causation
Hospitalization	Causation, damages
Rehabilitation/outpatient	Causation, damages
Neuropsychological	Causation, damage, deception detection
Neuropsychiatric	Causation, damages, deception detection
Collateral sources	Causation, damages
Preinjury documents	Causation, deception detection
Academic/employment	Causation, damages
Legal records	Causation, deception detection
Military records	Causation, deception detection
Depositions	Damages, deception detection

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are primary issues of liability and must be determined by lawyers and judges, the opinion expert physician may be asked an apportionment question, and thus the forensic physician should be prepared for this eventuality.

When the forensic physician testifies, the plaintiff lawyer will be interested in establishing a proximate cause of the brain injury, if one exists. On the other hand, the defense lawyer will attempt to prove that there is no proximate cause. In the law, the use of the term *proximate cause* is a clear example of an attempt to limit liability, even in cases where causation is clearly established. The original concept of “proximate” cause is traced to Lord Chancellor Bacon and is found in *The Law of Torts*.<sup>9</sup> Dean William Prosser noted that the term *proximate cause* is confusing and suggests that it is best to use terms such as *legal cause* and perhaps even *responsible cause*. However, these legal arguments notwithstanding, the forensic physician will be asked to render an opinion as to the proximate cause of the brain injury should the issue be tried at court.

From a physician’s standpoint, causation is usually fairly simple to determine in a brain injury case. If the plaintiff was in an automobile accident wherein he sustained a right temporal depressed skull fracture with an underlying subdural hematoma and is then found at neuropsychiatric examination to have evidence of right hemisphere brain dysfunction by brain imaging and neuropsychological assessment, the issue of causation should be simple and straightforward. The primary conflict for the physician regarding causation occurs in those instances of mild head injury that make up the vast majority of traumatic brain injury claims in the U.S. In these instances, the plaintiff generally has had a concussive blow to the head with limited to no evidence of external abrasion, no loss of consciousness, but then an apparent production of postconcussive symptoms thereafter. Many months or even a year later, the neuropsychiatric examination will take place. Minimal neuropsychiatric findings may be evident and the difficult question of causation then arises. It is these cases that present substantial challenges to the skill and analytical capacity of the neuropsychiatric examiner in a forensic situation. It is also these very cases of minor head injury that generally require the forensic examiner to develop a much more extensive database than would be required in determining causation of a more severe injury. It is the minor head injury that forces the forensic physician to broaden the examination in order to include a larger number of “rule ins” and “rule outs.”

To make an effective causation analysis, the examining physician should always return to the scene of the accident. It is the records of police, eyewitnesses, emergency medical services, and the emergency department that will provide the most salient information to assist the physician examiner in determining causation. The other records noted previously, which are used in the analysis of data, have importance, but from a causation standpoint, in most cases, the early records will be the most important to establish causation. However, there is a caveat. A not insignificant number of head injuries are overlooked early on in the treatment process. In other instances, the examinee may have had such severe physical injuries that an adequate assessment for brain injury could not be made (see Case 1 in [Chapter 8](#)). Another important factor to consider is that many individuals may be left with residual symptomatology from a concussive blow to the head when in fact the GCS score at the scene was measured to be 15. Even with these caveats, the early records are the most important for determining causation unless it can be conclusively shown that a traumatic brain injury was overlooked initially. [Table 11.3](#) provides a structure for determining medical causation following traumatic brain injury.

## DAMAGES ANALYSIS

The analysis of damages is noted in more detail below within the context of the cases represented in this chapter. As has been described elsewhere in this text, the damages portion of a forensic neuropsychiatric brain injury examination is the most important contribution the neuropsychiatric examiner will provide to the trier of fact in a brain injury litigation. The reader may want to review the “Damages” section of [Chapter 10](#) at this point. Outcome analysis is important, and the analysis of measurements made during the neuropsychiatric examination is also important.

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**TABLE 11.3**  
**Analysis of Brain Injury Medical Causation**

<p>Early records: Police, emergency services, injury reports, and emergency department evaluation</p>	<p>Does the police report document an injury to the head or body? Do the EMS records document alterations of behavior or cognition and evidence of trauma or injury? Is an injury report consistent with trauma? Does the emergency department document mental, neurological, or brain imaging abnormalities? Are the medical deficiencies continuously and temporally related to the trauma?</p>
<p>Intermediate records: Hospital, outpatient, rehabilitation, and neuropsychological</p>	<p>Was hospitalization required as a direct result of trauma? Is outpatient or rehabilitation treatment a direct result of the trauma? Is neuropsychological assessment completed as a direct result of the trauma?</p>
<p>Is there an intervening cause?</p>	<p>Did another trauma or disease occur between the original trauma and the neuropsychiatric examination?</p>
<p>Is there a preinjury cause or contributing factor?</p>	<p>Is there a preinjury psychiatric or neurological disease which better accounts for the present psychiatric or cognitive complaints? Has a preinjury condition been exacerbated by the trauma?</p>
<p>Is the neuropsychiatric examination consistent with the injury reports and subsequent treatment data?</p>	<p>Is there evidence of symptom magnification or malingering?</p>

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The most important information regarding damages that can be provided by the forensic physician to the trier of fact is the effect of the residual brain injury deficits upon daily life functioning. Therefore, within a damages assessment, not only should the physician determine the level of brain injury, but also the physician should give significant attention to the impact of measured deficits upon daily functioning. As has been stressed earlier, this analysis should have ecological validity. A plaintiff lawyer wants to know, “How do the traumatic brain injury deficits in my client affect my client’s ability to function socially, personally, and occupationally?”

For example, an examinee who has sustained a substantial frontal brain injury, and who demonstrates significant executive dysfunction, may appear perfectly normal sitting in a courtroom in front of a jury. Even while he testifies, the average layman may note little wrong with the individual. It thus is incumbent upon the examining physician to communicate effectively to the trier of fact the components of an executive disorder and how that translates to impairment of daily functioning. The disturbances of goal setting, planning, future memory, response inhibition, modulation of behavior, self-monitoring, and other features of dysexecutive syndromes should be explained in a narrative fashion such that a layman can understand how these deficits preclude normal functioning. This requires much greater skill on the part of the examining physician than presenting a patient to a jury with an obvious left hemiparesis and structural deformities of the head as a result of skull fractures. [Table 11.4](#) describes a simple schema for analyzing damages following traumatic brain injury.

### **CASE 1: MALINGERING BRAIN INJURY ATTRIBUTED TO RAILROAD INJURY**

This case is a demonstration of traumatic brain injury malingering within the context of a civil litigation. The examinee was the plaintiff in this action. The forensic neuropsychiatric examination took place 39 months after the alleged head injury. It is a complex case containing multiple areas where testimony was inconsistent, a medical examination was suspiciously positive, and the examinee provided abnormal results on tests used for measurement of cognitive effort and psychological

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**TABLE 11.4**  
**Analysis of Brain Injury Damages**

What is the medical evidence of damage?	What symptoms does the examinee express? What is abnormal in the mental status examination? Are there focal neurological findings? Are there neuropsychological deficits consistent with brain injury? Do the medical records document neuropsychiatric injury? Is there brain imaging evidence of injury? Is malingering or symptom magnification absent?
Can the damage be quantified?	Can the deficits be measured and compared to a normative database? Can an accurate preinjury cognitive and behavioral baseline be established in the examinee?
How do deficits affect daily cognitive, behavioral, social, and occupational function?	Can the examinee attend, remember, use language, demonstrate executive function, and remain oriented? Can the examinee maintain normal mood, display appropriate behavior, communicate normally, and think rationally? Can the examinee relate to others, function in a social setting, and maintain relationships? Can the examinee maintain work pace, complete tasks, and maintain behavior in a work setting?

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state. The examinee was 32 years old at the time of his alleged injury. (Please note that, in contrast to the clinical examinations described in [Chapter 8](#), the focus is now upon forensic examination. Therefore, where legal cases have yet to be tried and no verdict has been reached by a jury or a judge, the injury is termed *alleged*. Moreover, statements made by examinees may also be *alleged*.)

### **HISTORY FROM THE INJURY RECORDS**

B.K. was working for a contract employer on a railroad site. A large trailer used for transporting heavy loads by truck, while being loaded onto a train, struck the examinee. He was knocked backward onto the ground. He was attended at the scene by an emergency medical service. No significant trauma to the body was noted. He was transported for emergency care to a midwestern university hospital trauma unit.

At arrival to the emergency department, his initial neurologic screening examination revealed him to be “alert.” The nursing record at this facility stated, “Has full recall of accident and events pre- and post-accident.” Head examination was described as “WNL.” No loss of consciousness was reported. The examinee complained of a temporal headache and right flank pain. He refused any form of invasive study, brain imaging, or rectal examination. He did provide a urine specimen, which revealed no blood. Neurological examination was completely nonfocal. He was placed in a trauma room for further observation. The GCS was assessed on three separate occasions over more than a 2-h period. The first determination revealed a GCS score of 15; the second determination, a GCS score of 15; and the third determination, a GCS score of 15. On all three occasions, the RTS was 12.

Please recall that the maximum obtainable score on the GCS is 15 and the maximum obtainable score on the RTS is 12. Three separate entries in the hospital record by nurses and a physician reported, “No LOC” (loss of consciousness). The examinee asked the nurses to give him “pain pills” and send him home. He was discharged from the hospital without a head injury sheet. (Most trauma departments, if a head injury is suspected, will discharge a patient from the emergency department to home with instructions to follow in the likelihood that a head injury has occurred.) A planar chest x-ray was within normal limits. He was discharged with a diagnosis of right chest abrasion.

B.K. presented himself to a community hospital in a state different than the site of the alleged injury on four separate occasions within an 11-month interval following the alleged brain injury. His complaint was that of pain, and he was specifically asking for acetaminophen with codeine. At no time during these hospital visits did he make any mention of head trauma or complaints

consistent with postconcussion syndrome or other signs or symptoms of traumatic head injury. On the fourth emergency department visit, 11 months after the alleged brain injury, he reported a recent motor vehicle accident wherein his face and chest struck the steering wheel. At this examination, he was noted to have facial swelling, bleeding from the mouth, right-sided chest pain, and an abrasion of the left knee. He was found to have tenderness at the malar eminence, and blurry vision was noted in the right eye with intact object discrimination. A fluorescein stain revealed a linear abrasion across the cornea. No focal neurological deficits were present. His examination revealed him to be conscious and oriented and without signs of neurological deficit. He left the hospital against medical advice when asked to stay for observation.

Approximately 7 weeks after the alleged brain injury, B.K. was examined by an orthopedic surgeon. His history to the surgeon had changed substantially from the history he had reported previously. He told the surgeon that when struck by the trailer, he was thrown 50 ft with sufficient force that his “boots were thrown off.” He further reported he had been rendered unconscious and awakened on the ground. He informed the orthopedic physician that he had not come to consciousness until he found himself in the emergency room. The orthopedic surgeon accepted his history as told by B.K. and did not review the original trauma records. He reported to the orthopedic surgeon at this time that he was walking 5 to 7 mi daily. An MRI of the cervical spine revealed a centrally located small, soft bulge of the C3-C4 disc. The cord was normal and there was no evidence of canal stenosis. The orthopedic surgeon recommended myelography, but this was never performed. The orthopedic physician referred B.K. to a physical therapist for functional capacity testing.

This testing was not completed until 10 months following the alleged brain injury. The functional capacity evaluation revealed a Waddell score in the moderate range consistent with some symptom magnification (Waddell signs are used by orthopedic physicians and neurosurgeons to determine symptom magnification or malingering of physical disorders). The functional testing revealed that B.K. was able to lift in the medium category of work, according to U.S. Department of Labor standards. He demonstrated tolerance of sitting, standing, climbing stairs, sustained trunk bending, sustained overhead reach, repetitive squatting, repetitive stooping, repetitive ladder climbing ability, trunk twisting, sustained forward reaching, repetitive forward reaching, and pushing and pulling, all on a frequent basis, and the ability to walk, handle, finger, and grip, all on a constant basis.

Approximately 2 years after the original alleged injury, B.K. was evaluated by a neurologist. The chief complaint he gave to the neurologist was “headaches, memory problems, and fingers, neck, and back.” The history in the neurologist’s office was taken by a layperson, and it was purported that this individual had been taught to take a medical history. The history given to that employee by B.K. was him checking hitches on railroad cars and “the next thing he remembers is being in an ambulance with something on his neck and paramedics were bending over him. B.K. has no memory of what happened to him, other than what was told to him.” He told this examiner that he would repeat sentences over and over and that he could not complete sentences. He further told the examiner that he could not remember where objects were placed in his house, and he reported poor concentration and that his “nerves were shot.” The neurologist examined B.K. and documented in his examination lateral gaze nystagmus, bilateral Babinski signs, normal deep tendon reflexes, and thenar atrophy. Moreover, the neurologist further documented that B.K. was dystaxic. Within the history obtained in the neurologist’s office, it was stated that B.K. required his girlfriend to help him up off the commode or he would fall. There is no documentation in any medical record, including that of this neurologist, of a medically observed fall or tendency to fall by B.K. The neurologist had performed and then interpreted an electroencephalogram (EEG) and reported theta activity found bilaterally on nasopharyngeal leads during EEG. No mention of infraorbital slowing was noted. No mention of slowing in any other portion of the EEG record was mentioned. The neurologist subsequently concluded that B.K. had sustained both a brain stem injury and bilateral injury to the anterior temporal lobes. This was in contrast to an MRI ordered by the neurologist that revealed no evidence of increased signal, atrophy, or encephalomalacia. Furthermore, the MRI



did not document structural changes in either temporal lobe or the brain stem. There was no evidence of infraorbital or frontal brain changes on the MRI.

### **MEDICAL HISTORY FROM RECORDS PRIOR TO THE ALLEGED ACCIDENT**

B.K. had his head struck by a brick 22 years prior to the alleged brain injury. He was evaluated in a midwest community hospital with a negative skull x-ray. A contusion of the right forehead was noted. Ten years prior to the alleged brain injury, he was seen again at the same emergency department as a result of a chemical burn of the right knee caused during an industrial accident. Also, 10 years prior to the alleged brain injury, he was evaluated in the emergency department of the same hospital following a fight in which he sustained a human bite to the right cheek. Five years prior to the alleged brain injury, following a motor vehicle accident, he was seen by a chiropractor. The chiropractor found evidence of lumbar spasms without structural damage. One and a half weeks prior to the alleged brain injury, B.K. was evaluated by an internal medicine physician at the request of a workers' compensation lawyer. This examination was occasioned by an alleged work-related injury occurring some 6 years prior to the alleged brain injury. The internal medicine doctor found B.K. "totally and permanently disabled." This finding was in direct contrast to the chiropractor's examination wherein B.K. had written in his own hand that his work-related injury was 90 to 95% better since the initiation of chiropractic treatment.

### **HISTORY OBTAINED FROM B.K.**

His chief complaint to the forensic neuropsychiatric examiner was of being nervous and that his fingers moved and jumped. He also reported headaches, neck and back pain, and leg and arm numbness. With regard to his mental state, he claimed depression secondary to pain, which he believed was caused by the subject accident. He complained of poor concentration and loss of memory. He reported that he could not remember the month or day that his children were born, and he could not remember the month or day his father was born, even though he asserted that he knew those prior to the accident. Moreover, he was present in the delivery room when both of his children were born, and he claimed that he could not remember the location of their births or their birth dates. He further reported that he could not remember prior employers. He admitted to occasional thoughts of suicide but denied any plan to harm himself. He was evasive when asked about harming others. He at first claimed that he was thinking of killing other people following his accident, but then abruptly changed his story to the examiner and said he could not remember thinking that. He complained of a sleep disorder associated with nightmares. However, the nightmares were not specific to the injury facts. He reported seeing giant snakes, particularly black, gray, or blue pythons or anacondas chasing him. They would chase him onto railroad tracks and then a trailer would come down on his head. He admitted that his head was not struck during the accident in question. He further reported nightmares of people knocking on his door, breaking into his home, and shooting at him or his family. He admitted that neither he nor his family had ever been shot at. Neurologically, he complained that his fingers and feet would get numb; he could not unscrew tops of jars, and he had to have his fiancée perform these maneuvers. He also claimed to be so off balance that he fell against doors and could not get himself off the commode without his fiancée's assistance.

### **PAST MEDICAL AND PSYCHIATRIC HISTORY**

He was a 10-lb baby who was not born prematurely. He did not have a birth injury and he had no growth difficulty. Developmental milestones were normal. He could sit still in school. He had no difficulty learning to read, and he could keep his mind on tasks in the classroom. As an adult, he complained mostly of joint and back-related problems, low sexual desire, and sleeping difficulty. He admitted to prior motor vehicle injuries, and in one, he had injured a patella. He denied any prior history of head injury, broken bones, or loss of consciousness. He was using the anti-

inflammatory medication tramadol at the neuropsychiatric examination. He admitted to smoking four cigarettes daily and occasionally drinking beer. He claimed his last drink of alcohol was 2 years previous to the examination. (In an examination performed 2 weeks prior to his injury, he was noted to be using large quantities of alcohol and taking his mother's narcotic pain pills.)

His psychiatric history was essentially negative. He had never been prescribed any form of psychiatric medicine and he had never had psychiatric hospitalizations. He had never received any form of office counseling. He denied any history of overdoses with medications or attempts to take his life. He had never intentionally cut, burned, or disfigured himself. He denied having tattoos.

## **FAMILY AND SOCIAL HISTORY**

His father died at age 62 of myocardial infarction. His mother was living and 63 years of age. She had a stroke 2 years prior to this examination and was partially paralyzed on the right side. He denied any nervous breakdowns, mental illness, or depression in relatives. There was no history of addiction or alcoholism in his family. There was no family history of suicides, homicides, violence toward others, child abuse, or spouse abuse. No one in his family had epilepsy, other neurological diseases, or Alzheimer's disease. (Records of his neurologist stated that he had a cerebral-palsied child.)

He was born one of seven children. His father was employed in a steel mill and his mother worked for a meat-packing company. He dropped out of high school in his senior year. He denied any history of abuse in his home of origin. He denied that he had ever been violent to others. He obtained a GED approximately a year after leaving high school. He attended a trade school and held a certificate in construction and a certificate in lead abatement. He had never been married. He had two biological children by the woman with whom he lived. His legal history was negative, and he had never been convicted of a felony or misdemeanor. He had filed prior workers' compensation claims. He had never tried to enter military service. He had worked for his employer about 1½ years at the time of his injury.

## **REVIEW OF SYSTEMS AND ACTIVITIES OF DAILY LIVING**

He reported difficulty seeing small print, even with corrective lenses. His glasses had been prescribed prior to the accident in question, more than 3 years prior to the present examination. He complained of chest pain in the middle of his chest that radiated to the left side unrelated to exercise. He occasionally awakened during the night and reported pains in his chest, but he denied chest pain with exertion. In his gastrointestinal review, he reported heartburn and indigestion. The remainder of his system review was negative.

He had lived with the mother of his children for about 14 years. His time of arising was normal. He claimed to retire at night at approximately 6:00 or 7:00 P.M., even though it remained light outside. He claimed he had not driven a vehicle in almost a year. His report was that he had been told by his neurologist not to drive, but that admonition was not contained anywhere in the neurologist's record or in the neurologist's deposition.

B.K. was able to fix his meals. He claimed to read junk mail only because he could not read small print. He was able to write. He denied performing any work around his home and denied renting movies, attending movies or ball games, or hunting. He claimed that he had not fished in 8 or 9 years, and he had not eaten in a restaurant in 6 years. He did report using the telephone once a week. He admitted to dressing and bathing himself independently. He reported he was sexually functional twice monthly.

## **MENTAL STATUS EXAMINATION**

He was a pleasant, cooperative man who followed directions easily and without confusion. He was able to report his current street address, the city in which he lived, his 5-digit zip code, his age,

his 10-digit telephone number, and his 9-digit social security number from memory and independent of any memory aids. In almost the same breath, he claimed not to remember the birth dates of his children, where they were born, or the birth date of his father.

There was no evidence of dysprosody of language. His ability for narrative discourse was normal. No paraphasias were noted. There was no evidence of delusions or hallucinations, and thought and motor speed were normal and consistent with his level of educational attainment.

When asked specific questions requiring factual responses, he would often state, "I don't remember." However, during the examination, he was able to recite his father's age at death, his mother's current age, the date his mother had a stroke, which side of her body was paralyzed, how many brothers and sisters he had, his father's prior occupation, and where his mother was currently employed. He denied suicidal ideation. He was evasive when asked about homicidal ideation. However, he did not specifically name any party whom he was intending to harm. He reported minor subjective mood changes, but on direct observation, his affective range was normal but guarded. He was never tearful. He did not demonstrate lapses of attention or concentration.

During administration of the *Serial 7s Test*, he claimed not to know what he was supposed to do. When asked to subtract 7 from 100, he stated "63." When given the instructions a second time, he continued with "53, 43, 37, and 30." When asked the season, he responded "August." When asked the month of the year, he responded "second" (he was examined during the eighth month).

## NEUROLOGICAL EXAMINATION

He weighed 240 lb and had a blood pressure of 152/100 in the left arm. There were no bruits in the neck or head, and his head revealed no signs of trauma. Facial expression was symmetric. There were no deficits found in the cranial nerve examination. On motor examination, bulk and tone were good. Outstretched arms revealed no pronator drift. He could easily raise both arms above his head. Deep tendon reflexes were symmetric. The Babinski sign was bilaterally negative. No clonus was present.

On sensory examination, he reported reduced light touch over fingertips. Vibratory and position sensation were intact. The Romberg sign was not present. Tinel's sign was not present. His cerebellar examination revealed no dystaxia or dysdiadochokinesia. Neither lateral nor vertical gaze nystagmus was present. His gait was normal in stride and there was no dystaxia on turns. He could rise on his toes and raise his toes without dystaxia. He squatted and rose without signs of weakness, and there was no dystaxia, imbalance, or swaying during squatting, rising, toe-standing, or toe-raising.

## BRAIN IMAGING

An MRI was obtained 2 years following his injury and about a year and a half prior to this examination. It was performed with and without contrast and ordered by his neurologist. There was no evidence of intracranial hemorrhage, fluid collection, masses, or mass effect. No encephalomalacia was present and no posttraumatic changes were present. There was no evidence of hemosiderin deposits. The radiologist interpreted the films as being within normal limits.

## STANDARDIZED MENTAL ASSESSMENT

### Measures of Cognitive and Psychological Effort

B.K.'s results on the *Test of Memory Malingering* (TOMM) revealed a score of 25 on Trial 2 and a score of 26 on the Retention Trial. This pattern of performance is in the abnormal range. On the *Portland Digit Recognition Test*, he produced a percentage score of 52, which is in the abnormal range. A score below 63% correctly classifies 75% of those providing poor effort. On the *Rey 15-Item Figure Memory Test*, he produced a raw score of 8, which is in the abnormal range.

On the *Minnesota Multiphasic Personality Inventory-2* (MMPI-2) validity indices, he produced the following profile:

	Cannot Say	VRIN	TRIN	F	Fb	Fp	F-K	L	K	S
Raw score	0	10	9	17	16	2	4	9	13	22
T-score		69	50	89	108	56		74	45	47

On the *Structured Interview of Reported Symptoms*, he produced two scores in the “probable feigning” classification. His scores are noted next:

### Structured Interview of Reported Symptoms

Primary Scale	Raw Score	Classification
Rare Symptoms (RS)	2	Honest
Symptom Combinations (SC)	6	Indeterminate
Improbable or Absurd Symptoms (IA)	4	Indeterminate
Blatant Symptoms (BL)	2	Honest
Subtle Symptoms (SU)	16	Probable feigning
Selectivity of Symptoms (SEL)	9	Indeterminate
Severity of Symptoms (SEV)	9	Indeterminate
Reported vs. Observed Symptoms (RO)	7	Probable feigning

### Assessment of Reading Skill

On the *Wide Range Achievement Test-III* (WRAT-III), Reading Subtest, B.K. produced the following results:

### Wide Range Achievement Test-III

	Raw Score	Standard Score	Percentile	Grade Equivalence
Reading	34	68	2	4

### Mini-Mental State Examination

Based on the 1998 age and education adjusted norms from Spreen and Strauss, B.K. produced the following results:

### Mini-Mental State Examination

Raw Score	T-Score	Percentile	Classification
22	17	< 1	Significant

### Assessment of Emotional Adjustment

B.K. produced the following profile on the MMPI-2:

### Minnesota Multiphasic Personality Inventory-2

Scale	VRIN	TRIN	F	Fb	Fp	L	K	S
T-score	69	50	89	108	56	74	45	47

## Minnesota Multiphasic Personality Inventory-2

Scale	1	2	3	4	5	6	7	8	9	0
T-score	99	93	99	64	46	101	9	101	45	76

### RECORDS REVIEWED

During the neuropsychiatric examination, multiple medical records were reviewed. The original injury record and a copy of the original films from brain imaging studies were reviewed, as were the radiology reports. Multiple records were reviewed from his neurologist, orthopedic examinations, community hospitals, a chiropractor, and a vocational specialist. No preinjury cognitive evaluations were available, and there were no ACT or SAT scores for review.

### DEPOSITION TRANSCRIPT OF B.K.

Within his deposition, the forensic examiner was particularly interested in evidences of B.K.'s memory functioning. Depositions are an excellent way to determine memory functioning and the intactness of learning following an alleged brain injury. B.K. reported in his deposition the name of the physician who examined him following his accident even though he alleged he could not remember the accident. His testimony further indicated a prior history of three workers' compensation claims filed before the subject accident.

With specific regard to his factual memory, he was able to give his union number, the street of the union hall, and the specific street addresses of at least three prior employers. He corrected the lawyer who was examining him when the lawyer erroneously stated the wrong city for an automobile assembly plant where he had been employed previously.

When asked when he was injured, he stated in his deposition the correct month, day of the week, and year, but yet told the neuropsychiatric examiner that he could not remember the birth dates of his children or his injury date. In his deposition, he gave a clear description of the physical locations of other coworkers at the time of his alleged injury, specific statements about when trains were to arrive, and the exact numbers of trailers that were loaded the day of his alleged injury. He was able to report in his deposition that he worked long enough on the day of the alleged injury to receive overtime. He gave information about the phone calls shortly before his alleged injury that came from another city reporting times trains would be arriving.

### DIAGNOSES

He was diagnosed with no evidence of a cognitive disorder as a result of an alleged traumatic brain injury. He was further diagnosed with brain injury malingering.

### FORENSIC NEUROBEHAVIORAL ANALYSIS

Please refer to [Table 11.1](#). There was no police report to review as a result of this alleged injury. There was available an employer's report of injury. However, the ambulance record indicated no evidence of injury to the head, no alterations of consciousness, and no loss of consciousness. At the receiving hospital, B.K. did not cooperate fully with the medical examination. He refused to submit blood for analysis. He refused to allow a rectal examination. His GCS score was 15, and his RTS was 12. His neurological examination was nonfocal. He repeatedly asked nursing personnel to give him pain pills and let him go home. The emergency department record indicated, "Full recall of the accident and events pre- and post-accident."

Following release from the emergency department, during the next year, he presented himself to a community hospital on four occasions. He was his own health advocate on each occasion and recited accurate histories of his complaints. Three of these visits were to secure prescriptions for

acetaminophen and codeine. The fourth and last visit was for a motor vehicle accident. Not only did he leave the hospital early on this occasion, but he left against medical advice. No evidence of a prior brain injury or focal neurological findings was made by any physician or health care personnel during these four hospital visits.

When seen at a chiropractic clinic 1 month following the subject accident, he claimed on his medical questionnaire that he was “unconscious for 6½ hours.” He subsequently told an orthopedic surgeon who evaluated him that he was rendered unconscious at the time of the subject accident. Moreover, he stated he was struck so hard that his clothes and shoes were knocked off. (There is no evidence in any record that his clothing was removed from him by force.)

The neurological physician claimed a cognitive and brain stem injury to be present in B.K. Interestingly, the neurologist claimed that B.K. had bilateral Babinski signs, but yet his deep tendon reflexes were normal. The neurologist claimed a brain stem injury marked by horizontal nystagmus. Moreover, he claimed B.K. had bilateral temporal tip contusions, and yet the MRI he obtained was within normal limits. The basis for concluding the temporal tips were contused was an opinion by the neurologist that there was theta slowing bilaterally on the EEG with nasopharyngeal leads 2 years following the injury. This finding was not reproduced by any other physician.

With respect to the neuropsychiatric assessment of B.K., of the three standardized cognitive tests administered — Test of Memory Malingering, Portland Digit Recognition Test, and Rey 15-Item Figure Memory Test — B.K. failed all three. Of even more significance was his score on the *Mini-Mental State Test* by the method of Folstein. B.K. produced a score below the first percentile for U.S. norms. This indicated on this test instrument that he was more cognitively impaired than 99% of the U.S. population who completes this same test. These responses are not medically believable due to his obvious normal functioning following the alleged brain injury. A person scoring below the first percentile on the Mini-Mental State Test would be incapable of almost any useful function. The MMPI-2 produced by B.K. was consistent with symptom magnification (F = 89, Fp = 56, VRIN = 69). However, when the gold standard psychological malingering test, the Structured Interview of Reported Symptoms, was administered, B.K. produced a profile on two subscales with a probability of 75% or higher of faking.

The most telling argument in favor of faking brain injury is B.K.’s violation of Ribot’s law. Dr. Ribot reported in 1882, and this has been subsequently verified many times since, that if a person sustains a traumatic brain injury, the most recent memories are lost first in a retrograde fashion and the most remote memories are the best preserved. B.K. stated that he could not remember where his children were born, their birth dates, or the birth date of his father, but yet he was able to relate facts and events on the day of his injury; thus, he clearly violated this neurological law. Furthermore, B.K. gave great factual detail in his sworn deposition, which was entirely inconsistent with a memory disorder and entirely inconsistent with the information he provided to the forensic examiner. He could recite from memory his address, zip code, phone number, and social security number. Furthermore, his neurological examination was entirely within normal limits and not consistent with a brain injury. The MRI examination obtained by his own neurologist was entirely normal and showed no evidence of structural changes in the temporal tips, brain stem, or other parts of brain tissue.

## **CASE 2: ADULT GUNSHOT WOUND OF HEAD**

### **INTRODUCTION**

This case demonstrates the complications of penetrating brain injury. S.T. was a 41-year-old male at the time of his neuropsychiatric evaluation. He was suing a colleague who shot him in the head while they were wild turkey hunting. This case represents the sequelae from a very large craniotomy that was required to remove the extensive hematoma and lead fragments within the cranial vault. This also is an example of open-head injury that can result in posttraumatic seizures.

## **HISTORY OF THE ACCIDENT**

This examination occurred 4 years following the gunshot injury. S.T. was shot in the head from a distance of about 50 to 60 yd by a hunting companion. He was struck multiple times about the head and face with number 5 lead pellets projected by a 12-gauge shotgun firing 3-in. number 5 shot-shell heavy turkey loads. The frontal skull was entered and the left eye was severely damaged. After the shooting, he was transported by an emergency medical squad from the hunting area. His initial GCS score was 11. He was transported to a regional hospital near the hunting area.

An initial CT scan of the brain revealed a large and extensive hematoma tracking from the right frontal lobe posteriorly to the right temporal lobe. It also involved portions of the anterior right parietal lobe. Number 5 lead pellets were noted adjacent to the inner table of the right temporal bone with a tract of hematoma within the brain and also pneumocephalus in the area of the hematoma. Bony fragments were noted within the frontal lobe hematoma, and subarachnoid blood was noted over the right hemisphere. The lead shot had penetrated into the region of the right posterior clinoid process.

He required a very large craniotomy with exploration of the frontal brain. During neurosurgical recovery, he developed complex partial seizures. He was loaded initially with phenytoin, but due to drowsiness, it had been changed to valproate. At the time of this neuropsychiatric examination, he was treated with 1000 mg of valproate daily. Approximately a year following the shooting, he required a second hospitalization as a result of posttraumatic seizures. He continued to suffer seizures periodically, with the last witnessed seizure present approximately 8 or 9 months before the current neuropsychiatric evaluation.

## **HISTORY FROM THE PATIENT**

He reported himself to be tired and weak. In the 4 years since the gunshot wound, he remained with chronic hypersomnia and lethargy. He required 8 to 10 h of sleep daily and yet required three or four naps a week due to sleepiness. He also had developed restless leg syndrome prior to sleep. He had developed total blindness of the left eye due to traumatic injury of the globe. With regard to his psychiatric state, he reported substantial mood changes associated with depression and loss of memory. On the other hand, he denied disorientation, word-finding difficulty, or any elements of psychosis. He denied any plans to harm himself or harm anyone else.

## **PAST MEDICAL AND PSYCHIATRIC HISTORY**

He was not born prematurely and he had not suffered a birth injury. He developed well and reported that he was happy as a child. He was able to sit still in school and keep his mind on tasks. He had no preinjury history of seizure disorder. He had never been injured in a motor vehicle accident, and he had never been in a coma or broken any bones. He had never had surgery until this accident. He had no known allergies to drugs or medicines. He smoked one and a half packs of cigarettes daily. He did not use alcohol. He did have a prior conviction for DUI with marijuana possession.

In his psychiatric history, he had never been treated with antidepressants, tranquilizers, or any other psychiatric medicines. He had never been hospitalized for psychiatric, drug abuse, alcohol, or mental difficulty. He had never undergone any form of counseling or psychotherapy. He had never intentionally overdosed himself on drugs or medicines, and he had never made an attempt to take his life. He had never cut, burned, or disfigured himself.

## **FAMILY AND SOCIAL HISTORY**

His father was living at the time of this examination and was quite elderly — more than 80 years of age. However, he had sustained two strokes. S.T. was not sure of his mother's age, but she was

elderly as well and her apparent health was good. There was no family history of mental illness or depression. There was no history of substance abuse in the family, and there had been no history of suicides, homicides, violence toward others, or any form of abuse in his family. No one in his family had epilepsy, other neurological diseases, or Alzheimer's disease.

In his social history, he was born in the western part of Kentucky. He was one of three children. His father was self-employed, and his mother was a homemaker. His father did not abuse his mother, and his home life was happy. He had no history of sexual or physical abuse to himself. He had no history of violence toward others. He was a high school graduate and had obtained a 2-year associate arts degree from a university. He had majored in building construction. He had never been married, and he had no children.

At the time of his shooting, he was employed at a coal-loading tipple. As a result of his injury, he remained off work for more than a year but had returned to his usual employment after approximately 14 months postinjury. Due to apparent cognitive difficulty, he had an accident at work and was suspended from his employment. After a hearing, his employment was returned to him and he continued working at a coal tipple until he was laid off 10 months prior to the neuropsychiatric examination.

## **REVIEW OF SYSTEMS AND ACTIVITIES OF DAILY LIVING**

In his general review, he complained of severe fatigue and tiredness. In the HEENT (head, eyes, ears, nose, and throat) review, he complained of chronic headaches since the gunshot wound and craniotomy. His chest and cardiovascular reviews were negative. His gastrointestinal review was positive for heartburn and diarrhea. His genitourinary review was positive for bed-wetting, which had not been an issue prior to the anterior brain injury. His psychiatric and neurological symptoms were noted previously.

In terms of his activities of daily living, he was not employed at the time of the examination. Due to financial difficulties, he had moved in with his elderly parents. He remained able to drive a vehicle and able to read a newspaper and write. He watched television two or three times daily and did some yard and garden work for his parents when the weather permitted. Interestingly, he continued to hunt and fish. He was able to eat outside his home about 15 times monthly. He was able to use the telephone and could dress and bathe himself independently. He denied any sexual dysfunction.

## **MENTAL STATUS EXAMINATION**

He was a thin man and looked older than his stated age. He had an obviously dysfunctional and anatomically distorted left eye. He had no useful vision in the left eye. He was oriented to person, place, and time and was a capable historian. He independently completed a 22-page forensic medical questionnaire. The questionnaire was then verified with him face-to-face by the examiner. He was given written warning that his information would not be held confidential; he was also warned that the examiner would not have a doctor-patient relationship with him and would provide no treatment or counseling.

Mood was diminished by subjective report. Objectively, his affective range was constricted. He became tearful when describing the outcome of the shooting. He also had significant flatness of affect and the affective components of language were reduced. He did not smile at any time during the examination and was devoid of facial expression, with extreme blandness of his affect and persona. He specifically denied suicidal ideas or plans. His thinking was logical and coherent without evidence of loose associations or circumstantial thinking. No delusions or hallucinations were present. As noted, there was an apparent alteration of prosody. There was no coloring to his language. The melodic line was constricted in amplitude accompanied by a shortening of phrase length. There were no obvious paraphasias or word-finding difficulties present.



## NEUROLOGICAL EXAMINATION

S.T.'s body mass index was 26. His blood pressure was 130/82 in the left arm sitting position. No bruits were detected about the head or neck. The face revealed substantial signs of trauma and was asymmetric with evidence of traumatic injury to the left globe. The right fundus was benign, but the left fundus was grossly scarred. Cranial nerve function was normal with the exception of an absent light reflex and pupillary response to light in the left eye. He demonstrated anosmia. The extraocular movements of the left eye remained intact.

On motor examination, strength was symmetric and there was no drift of outstretched arms. Cerebellar examination revealed no evidence of dystaxia, dysmetria, or dysdiadochokinesia. There was neither horizontal nor vertical nystagmus present. Deep tendon reflexes were symmetric and normal in amplitude. Sensory examination was within normal limits to light touch, vibration, and temperature sense. The Romberg sign was not present. The Babinski sign was absent bilaterally, and no clonus was present.

His gait and station were normal, and he had a normal stride while walking. Heel raising was performed strongly. He was able to rise on his toes strongly. He could squat and rise from the squatting position without dystaxia. Arm swing was normal in arc and amplitude. Motor speed was normal. There was no dystaxia present on turns and no evidence of dystaxia on heel-toe walking.

## BRAIN IMAGING AND SKULL X-RAY

An MRI of the head without contrast was obtained. One image is seen in [Figure 5.3](#). There was noted in multiple images to be extensive injury involving primarily the right frontal brain, right anterior basal ganglia, and right deep sylvian fissure. Mild right hippocampal atrophy was noted when compared to the left side. Associated white matter changes were prominent throughout the right hemisphere related to atrophy of white matter tracts.

A limited skull series was obtained. One view is noted in [Figure 5.2](#) as well. Numerous lead shot remain imbedded in the soft tissues over the face. There is evidence of previous right frontal craniotomy with titanium-retaining devices in place. An electroencephalogram also was obtained. The predominant background rhythm was 8 to 9 Hz of moderate amplitude activity. The background activity was seen best in the posterior head regions. Photic stimulation elicited no abnormalities. Light sleep was noted during the recording. No focal or generalized abnormalities were seen, and no distinct epileptiform discharges were noted.

## SKULL X-RAY

A skull x-ray was obtained to determine if ferromagnetic material was present prior to MRI examination. The x-ray revealed numerous titanium plates holding in place a bone flap removed during a right frontal craniotomy. Moreover, multiple lead shot remained imbedded in the superficial tissues of the skull. There was evidence of destruction of the anterior teeth as a result of the shooting.

## MAGNETIC RESONANCE IMAGING

An MRI of the brain was obtained. A large skull defect in the right frontal area was noted. A surgical tract was present running from the anterior right frontal lobe posteriorly into the temporal and parietal brain structures following the axis of penetrating lead shot. Please see [Figure 5.3](#) and [Figure 5.4](#).

## STANDARDIZED MENTAL ASSESSMENT

He was administered the *Test of Memory Malingering* (TOMM), the *Victoria Symptom Validity Test* (VSVT), and the *Letter Memory Test* to screen him for cognitive effort. On the TOMM, he produced

a perfect score of 50 on Trial 2, which is a normal pattern. On the VSVT, he produced a total score of 47 of 48, with a score of 23 of 24 on the difficult item section. These results were within the valid range. On the Letter Memory Test, he produced a percentage score of 100, which was within normal limits.

With regard to psychological malingering, he was administered the *Personality Assessment Inventory* (PAI). On it, he produced the following validity profile:

	ICN	INF	NIM	PIM
T-score	58	44	55	50

The results of these four validity indices are within normal limits. As a further check on psychological validity, the PAI Malingering Index was also calculated (see [Chapter 7](#)), and he produced the following valid profile with no evidence of malingering:

### PAI Malingering Index

Index Item	Item Weight	Score
1. NIM = 110T	1	0
2. NIM – INF = 20T	1	0
3. INF – ICN = 15T	1	0
4. PAR-P – PAR-H = 15T	1	0
5. PAR-P – PAR-R = 15T	1	0
6. MAN-I – MAN-G = 15T	1	1
7. DEP = 85T and RXR = 45T	1	0
8. ANT-E – ANT-A = 10T	1	1
<b>Total</b>		<b>2</b>

### Measures Providing Estimates of Preinjury Function

The *Wechsler Test of Adult Reading* (WTAR) was administered to S.T. He produced the following demographic predicted profile:

### Wechsler Test of Adult Reading

Raw Score	Standard Score
18	77

### Demographic Predicted WAIS-III Indices

	Standard Score	Percentile	Classification
WAIS-III VIQ	107	68	Average
WAIS-III PIQ	105	63	Average
WAIS-III FSIQ	107	68	Average
WAIS-III VCI	106	66	Average

Due to the large discrepancy between demographic predictions and WTAR predictions, the demographic predictions were used only to predict certain *Wechsler Adult Intelligence Scale-III* (WAIS-III) scores, and they were not used to predict *Wechsler Memory Scale-III* (WMS-III) scores. The examiner should review the WTAR manual if further information is needed.

## Attention and Concentration

The *Ruff 2 and 7 Selective Attention Test* was administered to S.T. to measure visual attention. As a measure of auditory attention, he had administered to him the *Brief Test of Attention (BTA)*. The following scores were determined:

### Ruff 2 and 7 Selective Attention Test

		Raw Score	T-Score	Percentile	Classification
Letters	Automatic detection speed	112	39	14	Mildly impaired
	Automatic detection errors	1			
	Automatic detection accuracy	99	55	70	Above average
Digits	Controlled search speed	97	36	8	Mildly impaired
	Controlled search errors	11			
	Controlled search accuracy	90	42	21	Below average

### Total Scores

Measure	Sum of T-Scores	T-Score	Percentile	Classification
Total speed	75	39	14	Mildly impaired
Total accuracy	97	48	42	Average

### Brief Test of Attention

	Raw Score	Percentile	Interpretation
Form N (numbers)	7		
Form L (letters)	9		
<b>BTA total score</b>	<b>16</b>	<b>25-74</b>	<b>Average</b>

As a further test of attention, S.T. was administered the Digit Span subtest of the Wechsler Adult Intelligence Scale-III. The results of that measure are noted below:

### WAIS-III Digit Span Subtest

	Raw Score	Standard Score	Percentile	Classification
Longest digit span forward	6	93	32	Average
Longest digit span backward	2	70	2	Borderline
Digit span scaled score	5	75	5	Borderline

## Language and Language-Related Skills

S.T. was administered the *Boston Naming Test* and produced the following scores:

### Boston Naming Test

Raw score	49
T-score	24
Classification	Moderately to severely impaired
Percentile	0.9

S.T. was also administered the *Controlled Oral Word Association Test (COWA)* as a measure of verbal fluency. He produced the following scores:

### Controlled Oral Word Association Test

Raw score	15
T-score	23
Percentile	0.8
Classification	Severe defect

In an effort to measure posterior brain language functions, he was administered the *Token Test* for comprehension and produced the following profile:

### Token Test

Total raw score	158
Percentile	30
Classification	Normal

### Visuospatial Abilities

S.T.'s skills in this domain were measured using the *Judgment of Line Orientation*. He produced the following mean corrected score:

### Judgment of Line Orientation

Raw score	27
Age-corrected raw score	27
Percentile	72
Classification	High average

### Memory

Memory was assessed using the WMS-III. S.T. produced the following memory scores and classifications:

### Wechsler Memory Scale-III

	Scale Score			Classification
	Sum	Index Score	Percentile	
Auditory immediate	17	92	30	Average
Visual immediate	13	78	7	Borderline
Immediate memory	30	82	12	Low average
Auditory delayed	20	99	47	Average
Visual delayed	14	81	10	Low average
Auditory recognition delayed	7	85	16	Low average
General memory	41	87	19	Low average
Working memory	12	79	8	Borderline

### Sensory Perceptual Skills

These were measured using the *Reitan-Kløve Sensory Perceptual Examination*. S.T.'s T-scores are reported next:

## Sensory Perceptual Examination

	Raw Score	T-Score	Classification	Percentile
Total right errors	28	21	Moderately to severely impaired	0.5
Total left errors	21	15	Severely impaired	0.05
<b>Sensory perceptual total</b>	<b>49</b>	<b>19</b>	<b>Severely impaired</b>	<b>0.2</b>

## Motor and Visual Motor Skills

S.T. had administered to him the *Grooved Pegboard Test*, *Grip Strength Test*, and *Finger Tapping Test*. On a measure of manipulative dexterity, he produced the following profile:

### Grooved Pegboard Test

	Raw Score	T-Score	Classification	Percentile
Dominant right hand	87	32	Mildly to moderately impaired	4
Nondominant left hand	99	29	Moderately impaired	2

Strength was measured using the Grip Strength Test, and S.T. produced the following strength scores:

### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant right hand strength	43	41	Below average	19
Nondominant left hand strength	35	36	Mildly impaired	8

Finger-tapping speed was assessed, and S.T. produced the following T-scores:

### Finger Tapping Test

	Mean Raw Score	T-Score	Classification	Percentile
Dominant right hand	47	41	Below average	19
Nondominant left hand	43	41	Below average	19

## Executive Function

S.T. was administered the *Wisconsin Card Sorting Test* to determine his ability to form, maintain, and shift cognitive sets and utilize feedback in modifying responses. He produced the following scores:

### Wisconsin Card Sorting Test

	Raw Score	Standard Score	T-Score	Percentile	Classification
Total errors	94	< 55	< 20	< 1	Severely impaired
Completed categories	0			< 1	Severely impaired
Perseverative responses	122	< 55	< 20	< 1	Severely impaired

As a measure of visuo-optical scanning ability and the ability to maintain concentration under time constraints, S.T. produced the following scores on *Trail-Making Tests A and B*:

### Trail-Making A

Raw Score	T-Score	Classification	Percentile
61	24	Moderately to severely impaired	0.9

### Trail-Making B

Raw Score	T-Score	Classification	Percentile
251	20	Moderately to severely impaired	0.3

### Test Intelligence

This was measured using the WAIS. S.T. produced the following profile:

### Wechsler Adult Intelligence Scale-III

Subtest	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Picture Completion		4		4		
Vocabulary	7		7			
Digit Symbol-Coding		5				5
Similarities	5		5			
Block Design		6		6		
Arithmetic	7				7	
Matrix Reasoning		8		8		
Digit Span	5				5	
Information	8		8			
Picture Arrangement		5				
Comprehension	7					
Symbol Search		(7)				7
Letter-Number Sequencing	(5)				5	
<b>Sum of scaled scores</b>	<b>39</b>	<b>28</b>	<b>20</b>	<b>18</b>	<b>17</b>	<b>12</b>

### Deviation IQs

	Standard Score	Classification	Percentile	Range
Verbal IQ	79	Borderline	8	75-85
Performance IQ	73	Borderline	4	68-81
Full-scale IQ	74	Borderline	4	70-79

### WAIS-III Index Scores

	Standard Score	Classification	Percentile	Range
Verbal comprehension	82	Low average	12	77-88
Performance organization	76	Borderline	5	70-85
Working memory	73	Borderline	4	68-81
Processing speed	79	Borderline	8	73-90

## Psychopathology

This was measured using the PAI. S.T. produced the following scores:

### Personality Assessment Inventory

	1	2	3	4	5	6	7	8	9	10	11
	SOM	ANX	ARD	DEP	MAN	PAR	SCZ	BOR	ANT	ALC	DRG
T-score	70	51	54	62	51	62	45	56	52	47	46
	Validity				A	B	C	D	E	Y	Z
	ICN	INF	NIM	PIM	AGG	SUI	STR	NON	RXR	DOM	WRM
T-score	58	44	55	50	51	45	57	61	51	40	47

## RECORDS REVIEWED

The records in this case consisted entirely of the records of a regional medical center near S.T.'s home. All his care was provided in his local area, including acute care and outpatient care.

## DIAGNOSES

The diagnoses in this case were cognitive disorder due to gunshot wound to brain, DSM-IV 294.9; dementia due to gunshot wound to brain, DSM-IV 294.1; and personality change due to gunshot wound to brain, DSM-IV 310.1. Axis II is not specific for a personality disorder, and personality changes due to an organic mental condition were placed in Axis I. Under Axis III, S.T. had a diagnosis of penetrating brain injury and visual loss in the left eye associated with posttraumatic seizure disorder. Under Axis IV, he had moderate difficulty due to unemployment as a result of his brain injury, and it was judged that his current Global Assessment of Function was approximately 50.

## FORENSIC NEUROBEHAVIORAL ANALYSIS

The reader should again review [Table 11.1](#). This schema for collecting the forensic neuropsychiatric database can prove useful at the time of the forensic neurobehavioral analysis. This step-wise approach offers the forensic examiner consistency as a forensic neuropsychiatric brain injury evaluation is analyzed. As was discussed in [Chapter 8](#), a portion of the forensic neurobehavioral analysis should be evaluated by the examiner within the context of what was learned in the clinical sections of this book. That is, the examiner should review the history, mental status examination, neurological examination, brain imaging, and results of standardized mental assessment in combination with records reviewed in order to determine the overall aspects of the case. These factors should be systematically analyzed individually.

Taking this case at hand, the history of the accident is fairly clear. It was incontrovertible that S.T. was shot in the head, causing number 5 lead pellets to penetrate the frontal brain and damage his left eye. Further evidence of damage to his brain was noted on the initial CT scan. At the time of the neuropsychiatric examination, numerous MRI views indicated extensive injury to the right frontal area and right hippocampal complex. The right anterior basal ganglia and the right deep sylvian fissure were noted to be injured on other MRI views. Moreover, it is noteworthy that right hippocampal atrophy is present and white matter changes are present throughout the right hemisphere related to posttraumatic atrophy of various white matter tracts. Furthermore, the medical records reveal the postsurgical onset of complex partial seizures. At the time of this neuropsychiatric evaluation, he required valproate for suppression of seizures. Moreover, the records further indicate that he had required one hospitalization as a result of posttraumatic seizures approximately a year after his injury.

He demonstrated posttraumatic hypersomnia (see [Chapter 2](#)). In association with his hypersomnia was the syndrome of restless legs that he developed. He chronically complained of headaches, primarily behind the left eye. He reported substantial mood changes of depression and further reported memory impairment. He denied language dysfunction or psychosis. His mental status examination was noteworthy for elements of dysprosody of language. The gestural aspects of spoken language were abnormal, and the affective components of language were suppressed. He had a constricted range of affect associated with the right hemisphere disorder. His neurological examination was focal for anosmia. Recall that he had damage to the right basal ganglia, but there was no evidence of abnormal involuntary movements or obsessive-compulsive features.

The skull x-ray noted in [Chapter 5](#) ([Figure 5.3](#)) demonstrated numerous lead shot imbedded in soft tissues over the face. This will become important in the causation analysis discussed below. Moreover, there is evidence of previous right frontal craniotomy, which correlates with a surgical track that can be seen on the MRI ([Figure 5.4](#)). This MRI is important in the damages analysis discussed below. The electroencephalogram showed no current evidence of epileptiform activity, but S.T. was actively treated with valproate at the time the EEG was obtained within the context of this neuropsychiatric examination.

After the examiner has reviewed the data just discussed, attention should be given to the forensic aspects of the neuropsychological assessment. First is the issue of whether S.T. produced optimal cognitive effort. On the TOMM, he produced a perfect score of 50. On the VSVT, he produced a score of 47 of 48, which is within the valid range. On the Letter Memory Test, he produced a perfect score of 100. Thus, the neuropsychiatric examiner can be confident with these cognitive effort values that the remainder of the neurocognitive examination was performed within the context of optimal effort. With regard to his psychological effort, the reader should review the validity data for the Personality Assessment Inventory noted above. In particular, it is noteworthy that on the NIM (negative impression) scale he produced a T-score of 55, and on the ICN (inconsistency) and INF (infrequency) scales he produced respective T-scores of 58 and 44. As the reader recalls from [Chapter 7](#), these are well within normal limits, and thus S.T. produced a PAI profile that is valid and interpretable. Moreover, on the PAI Malingering Index, S.T. produced a total score of 2, which is well within normal range and indicative of no evidence of malingering. These cognitive and psychological validity data enable the forensic examiner to testify confidently that S.T.'s cognitive and psychological effort was optimal within the neuropsychiatric evaluation. These data also enable the forensic examiner to testify within a *Daubert* challenge that tests with proven reliability and scientific validity are being used within the context of the neuropsychiatric evaluation.

The next item of analysis within the neuropsychological portion of the neuropsychiatric examination is an attempt to evaluate probable preinjury cognitive function. The reader should refer to the WTAR produced by S.T. Please note that he produced a standard score of 77 on the reading portion. Due to the discrepancy between that score and the demographic predictions of the WTAR, only the demographic predictions are used to predict preinjury function for S.T. Further details are beyond the scope of this text; for more information, consult with a psychologist or refer to the WTAR manual. However, clearly we would expect, based on the demographic predictive power of the WTAR, for S.T. to produce cognitive scores within the average range.

As we look specifically at data for S.T. on the Ruff 2 and 7 Selective Attention Test, S.T. has visual search speed below predicted levels; however, his accuracy remained in the average range. His auditory attention does not appear to be impaired as a result of the gunshot wound, and it remains in the average range. There is a significant discrepancy between digits forward and digits backward on the WAIS-III Digit Span subtest. Since digits forward primarily measures the efficiency of auditory attention; this is consistent with the BTA findings. On the other hand, digits backward uses working memory and involves mental double tracking. Thus, it is not unexpected that S.T. produces impaired levels on the digit backward test. If the reader reviews the working memory index score for the WAIS-III, it can be seen that S.T. also is within the borderline range on that test. This provides confidence that his performance on the WAIS-III Digit Span subtest is subnormal.



In a review of language and language-related skills, S.T. is significantly impaired on the Boston Naming Test and the COWA. Since these tests both measure anterior language functions, the results are not unexpected in light of the injury to the anterior brain parts. If the reader will review the Token Test, clearly S.T.'s performance is in the normal range, and since comprehension is a posterior brain function, the language findings comport very closely with the anatomical site of injury. Furthermore, visuospatial abilities, another posterior brain function primarily, are in the high average range on the Judgment of Line Orientation Test, again consistent with the anatomical location of injury.

On memory assessment, the primary area of injury appears to be in the visual domain, whereas the auditory domain is spared. Moreover, working memory on this test again is probably in the impaired range based on S.T.'s preinjury predictions. The impairment of visual memory correlates well with the impairment of visual attention. On the sensory perceptual portion of the neuropsychological assessment, S.T. performs poorly on both sides of the body but probably more so on the nondominant side, consistent with the primarily right hemisphere injury he sustained. The same pattern of lateralization is present on the Grooved Pegboard Test and the Grip Strength Test. The Finger Tapping Test reveals no lateralization.

S.T. demonstrates rather profound levels of impairment on tests of executive function. This is not unexpected in light of the severe structural injury to the anterior brain parts. On a measurement of test intelligence, there is evidence of a probable change from preinjury predictions. This allows for the diagnosis of dementia, whereas the overall test impairments allow for the diagnosis of cognitive disorder. With regard to psychopathology, while S.T. complains of mood alterations, these are not born out on the Personality Assessment Inventory. Scale 4, the Depression Scale, has a T-score of 62. However, it does not reach a level of clinical importance on this particular test instrument. The only elevation of clinical significance on the PAI is the Somatic Scale (scale 1), consistent with the perception of physical impairment he demonstrates.

Following this level of analysis, the examiner then should focus more specifically on the forensic issues. First of all, what is the evidence for medical causation? This is fairly simple to answer in that the medical records clearly document a gunshot wound. Number 5 lead pellets remain demonstrated in his facial structures. The MRI obtained during the neuropsychiatric evaluation clearly delineates evidence of frontal brain injury. His physical appearance demonstrates loss of vision in the left eye. Thus, the forensic neuropsychiatric examiner has no difficulty demonstrating causation between the gunshot to the head and the adverse neuropsychiatric outcome for S.T.

With regard to damage analysis, the reader should refer to [Table 11.3](#). The medical evidence of damage is clear. The history is consistent with expected symptoms following a gunshot wound to the frontal brain. The mental status examination is abnormal, particularly in nondominant cerebral hemisphere affective language components. While there are no focal neurological findings other than anosmia, clearly substantial neuropsychological findings are consistent with brain injury. There is brain imaging evidence of injury present. The remaining question is the level of injury. If the reader reviews [Table 11.2](#), the Clinical Dementia Rating (CDR) from the *Guides to the Evaluation of Permanent Impairment* can be used to assist with quantification. The left side of this table lists areas of inquiry to be memory, orientation, judgment and problem solving, community affairs, home and hobbies, and personal care. Within this schema, there is evidence of moderate memory loss. With regard to judgment and problem solving, based on the Wisconsin Card Sorting Test and the Trail-Making Tests, S.T. is severely impaired in his ability to provide executive function. His ability to function at home and engage in hobbies has been substantially truncated by his injury, and he is unable to be employed. Thus, using this table, a decline is evident from his previous functional level due to cognitive loss. His overall CDR lies between Category I (mild) and Category II (moderate). Overall, he is beyond the mild cutoff, and as noted in [Chapter 10](#), mild impairment ranges from 0 to 14% using Chapter 13 of the *Guides*. Thus, it was judged that his cognitive, intellectual, and personality changes accounted for a 25% impairment (moderate ranges from 15 to 29% using the *Guides*).

If asked to testify, the forensic neuropsychiatric examiner can state that from a damages standpoint, S.T. demonstrates alteration of right hemisphere language processing with evidence of dysprosody. Furthermore, he demonstrates impairment in the areas of verbal fluency, object naming, executive function, sensory-perceptual abilities, visual motor coordination, and left hand grip strength. In addition, nonverbal reasoning ability, mental processing speed, working memory, and immediate visual memory are within the borderline range of impairment.

## **CASE 3: INFANT MOTOR VEHICLE INJURY**

### **INTRODUCTION**

This case is interesting from two standpoints. First of all, it is that of a child, and second, it is a child injured at age 3 weeks while riding in an unrestrained car seat in the front seat of his parents' vehicle. It demonstrates the unique difficulties attendant to examining children injured shortly after birth. Moreover, it is instructive regarding secondary complications that may occur in a child injured at such a tender age.

### **HISTORY OF THE ACCIDENT**

At the time of the accident, L.G. was 3 weeks of age. At the time of the neuropsychiatric examination, L.G. was 4 years of age. An appropriate time interval had occurred between the date of the injury and the date of the neuropsychiatric examination in order to improve the quality of neuropsychological testing.

At age 3 weeks, L.G. was restrained in a car seat. However, the car seat was not restrained within the front seat of the vehicle in which he was riding. His vehicle was struck head-on by a large truck. He was attended at the scene by an emergency medical squad and transported immediately to a small community hospital. From there, he was intubated and transported by air to a large children's hospital in the Midwest. An initial CT scan of the head revealed multiple skull fractures. On day 2 of hospitalization, L.G. experienced a seizure and was treated with lorazepam and loaded with phenytoin. After phenytoin loading, he was placed on phenobarbital. Initial CT imaging revealed bilateral subdural hematomas, greater on the right side than the left. Physical examination initially revealed a right retinal hemorrhage, and there was also noted on CT scan to be a small intraventricular hemorrhage. Small foci of cortical contusions were noted over the right brain convexity. L.G. was discharged after approximately 2 weeks hospitalization.

Three weeks after the initial injury, L.G. was readmitted to the children's hospital because of the development of hydrocephalus secondary to meningitis. On admission, he was noted to have a bulging fontanelle and ventricular size was increased on CT scan. He was very irritable and had sundowning eyes. He required ventriculoperitoneal shunt placement.

### **HISTORY FROM THE PATIENT**

The mother was interviewed. She noted that L.G. was delayed in speech acquisition relative to his 6-year-old brother. He articulated poorly, and his mother had difficulty understanding him. If tasks were perceived to be difficult by L.G., he would not persist, and his mother reported a short attention span. He was irritable and would throw his preschool workbooks aside and ask to "do something else." The mother denied any evidence of mood changes, and she denied that L.G. was anxious. His sleep was reported to be relatively normal.

### **PAST MEDICAL AND PSYCHIATRIC HISTORY**

At 7 months of age, L.G. had a head circumference of 43.5 cm. He demonstrated a deceleration in head growth velocity to just below the 50th percentile. He was under active follow-up by a pediatric

neurosurgeon, and during an examination at age 28 months, L.G. was noted to use both hands equally. He was able to run well and jump in place. However, he was slow to use phrases and sentences. He was a shy, tentative child at that time. His head circumference had increased to 47.5 cm, which placed him only at the 10th percentile at that time. His shunt was intact and flowing well.

An MRI of the brain without contrast was obtained at age 35 months. This was compared to an examination that had been obtained at age 1 month. On the second MRI examination, the cerebellar tonsils were 11 to 12 mm below the foramen magnum. This was interpreted as a marked change since the prior examination almost 3 years earlier, when his cerebellar tonsils were in their normal position. The MRI revealed thinning and mild irregularity of the posterior aspect of the corpus callosum. There was a mild loss of central white matter noted. The findings were consistent with a Chiari I malformation, but there was no evidence of hydrocephalus on this examination. A cerebral spinal fluid flow study was obtained, and it revealed that the anterior subarachnoid space was narrowed at the level of the transverse ligament. The subarachnoid space posterior to the tonsils was narrowed. No significant subarachnoid fluid below the level of the cerebellar tonsils was noted. A ventricular shunt was noted extending from the right frontal region.

With regard to his continuing development, L.G.'s mother reported that he could sit alone at about age 6 to 7 months and he crawled at 10 months of age. He pulled himself up at 9 months. He stood alone at about 10 months of age and walked alone at 14 months. At the time of the neuropsychiatric examination, he was not yet potty trained. His seizure history did not persist. After 2 years, seizure medications were discontinued. At the time of the neuropsychiatric examination, he was being treated with 250 mg of amoxicillin three times daily for upper respiratory infection. He was using no over-the-counter medicines and no herbs or natural products. He had no known allergies to drugs or medicines. His caffeine use consisted of one Pepsi daily. He had required no psychiatric intervention since his injury.

## **FAMILY AND SOCIAL HISTORY**

The father was age 29 and the mother was 35. A half-sister died of heart disease at age 21 months as a result of multiple congenital heart defects. He and the half-sister shared the same mother. There was no family history of mental illness or psychiatric disorders. There was no history of substance abuse in the family. There was no family history of suicides, homicides, violence toward others, child abuse, or spouse abuse in his family. No one in the family had demonstrated neurological diseases or Alzheimer's disease.

L.G. was born in a midwest state other than the state wherein he resided at the time of the neuropsychiatric examination. His father worked in heavy construction, and his mother was employed as a cashier. Both parents were present in the home, and they had been married for 7 years. His home life was happy, and there was no abuse within the home. L.G. had never been sexually or physically abused by anyone, and his mother reported no history of violence toward other children. At the time of the neuropsychiatric examination, he was in a special-needs preschool. This was his second year in that facility, and he was scheduled to attend regular kindergarten in the year following this examination. He had not been a behavioral problem in the preschool.

## **REVIEW OF SYSTEMS AND ACTIVITIES OF DAILY LIVING**

L.G.'s general review was negative except for an upper respiratory infection present the day of the neuropsychiatric examination. His mother reported no difficulty with vision. His chest, cardiovascular, gastrointestinal, and genitourinary reviews were negative. His psychiatric review was negative for behavioral difficulties. His neurological and developmental review was positive for alteration of language development. His musculoskeletal and sleep reviews were negative. In his normal day, L.G. would arise about 9:00 A.M. and retire about 10:00 P.M. There were no unusual aspects to his daily activity, and he attended preschool five mornings weekly.

## MENTAL STATUS EXAMINATION

L.G. was a pleasant, cooperative youngster who was noted to be small for his age. He had a very intense focus when he talked to the examiner with an extreme fixed gaze. However, he could only hold this gaze for a short period, as his attention span was quite short and he was easily diverted by sounds or movements in the peripheral space. He was noted to have a very significant articulatory disturbance, and he slurred consonants. The melodic line and phrase length seemed to be reasonably normal. His mood was not depressed, and he was not anxious with the examiner, nor was he irritable.

## NEUROLOGICAL EXAMINATION

L.G. weighed 35 lb and had a blood pressure of 64/38 in the right arm sitting position. Examination of the head and face revealed a palpable V-P shunt tube traversing the right lateral neck. His head circumference was 49 cm and he was 105 cm in height. He had a slight pectus carinatum on chest examination. The cardiac impulse was in the normal position, and he had a widely split-second sound on cardiac exam. A benign functional flow murmur was appreciated. His abdomen was scaphoid and without organomegaly, and extremities revealed full range of motion without cyanosis, clubbing, or edema. Cranial nerves II to XII were intact with the exception of poor articulatory ability. L.G. could hop on either leg and ran without dystaxia. His strength and deep tendon reflexes were symmetric. The toes were downgoing, and there were no abnormal movements or spasticity noted.

## BRAIN IMAGING

A SPECT scan was obtained using 31.7 mCi of technetium 99-labeled Neurolite intravenously. Axial images revealed an area of diminished activity in the right posterior parietal lobe. There was slightly diminished activity in both frontal lobes.

An MRI was obtained. There was no evidence of atrophy or white matter developmental disturbance. There was no evidence of hydrocephalus or focal encephalomalacia. The hippocampal complexes were within normal limits and without evidence of mesial sclerosis or developmental anomaly. There was a minimal residual defect extending along the tract of the catheter shunt. The shunt was placed in the right hemisphere.

## STANDARDIZED MENTAL ASSESSMENT

L.G.'s overall neuropsychological assessment was performed using the *NEPSY* (see [Chapter 6](#)). L.G. produced the following profile:

### NEPSY Scaled Scores

Subtest	Raw Score	Attention/ Execution	Language	Sensorimotor	Visuospatial	Memory
Body Part Naming	14		10			
Design Copying	18				8	
Phonological Processing	10		11			
Visual Attention	14	10				
Comprehension of Instructions	12		8			
Imitating Hand Positions	5			6		
Visuomotor Precision	12			8		
Narrative Memory	7					8
Block Construction	4				4	
Sentence Repetition	11					8
Statue	23	11				
<b>Sum of scaled scores</b>		<b>21</b>	<b>29</b>	<b>14</b>	<b>12</b>	<b>16</b>

## NEPSY Scaled Scores

	Core Domain Score	Percentile	Classification
Attention/executive functions	103	58	At expected level
Language	98	45	At expected level
Sensorimotor functions	81	10	Below expected level
Visuospatial processing	73	4	Below expected level
Memory and learning	88	21	Borderline

Test intelligence was measured in L.G. using the *Wechsler Preschool and Primary Scale of Intelligence-Revised* (WPPSI-R). He produced the following profile and IQ scores:

## Wechsler Preschool and Primary Scale of Intelligence-Revised

Performance Subtest	Scaled Score	Verbal Subtest	Scaled Score
Object Assembly	10	Information	7
Geometric Design	3	Comprehension	7
Block Design	7	Arithmetic	9
Mazes	6	Vocabulary	4
Picture Completion	10	Similarities	4
(Animal Pegs)	6	(Sentences)	8
Performance Score	36	Verbal Score	31

IQ standard scores are:

Performance IQ = 81

Verbal IQ = 78

Full-scale IQ = 77

In an effort to determine his current development, L.G. was administered the *Denver Developmental Screening Test-II*. This was in an effort to screen L.G. for personal and social development, fine motor adaptation, language development, and gross motor development. L.G. produced the following results:

## Denver II

Areas of Function	Classification
Personal-social	Suspect
Fine motor adaptation	Suspect
Language	Suspect
Gross motor	Suspect

In an effort to determine L.G.'s personal and social sufficiency, the *Vineland Adaptive Behavior Scales* were administered to his mother. Based on mother's reporting, the following profile was developed:

## Vineland Adaptive Behavior Scales

	Raw Score	Standard Score	Percentile	Stanine	Adaptive Level
Communication domain	132	71	3.0	1	Moderately low
Daily living skills domain	97	61	0.5	1	Low

## Vineland Adaptive Behavior Scales (Continued)

	Raw Score	Standard Score	Percentile	Stanine	Adaptive Level
Socialization domain	110	70	2.0	1	Moderately low
Motor skills domain	80	49	< 0.1	1	Low
Adaptive behavior composite	251	58	0.3	1	Low

L.G.'s behavior was assessed using the Parent Rating Scales (PRSs) of the *Behavior Assessment System for Children* (BASC). His mother completed these scales with the assistance of a psychologist, and the following scores were produced:

### Behavior Assessment System for Children: Parent Rating Scales

Scale	Raw Score	T-Score
Hyperactivity	28	68
Aggression	17	66
<b>Externalizing problems composite</b>	<b>134</b>	<b>69</b>
Anxiety	11	60
Depression	11	54
Somatization	5	46
<b>Internalizing problems composite</b>	<b>160</b>	<b>54</b>
Atypicality	7	63
Withdrawal	11	53
Attention problems	13	76
<b>Behavioral Symptoms Index</b>	<b>387</b>	<b>71</b>

Scale	Raw Score	T-Score
Adaptability	13	31
Social skills	23	48
<b>Adaptive skills composite</b>	<b>79</b>	<b>38</b>

Validity Scale	Raw Score	Classification
F index	0	Acceptable
Response pattern	92	Acceptable
Consistency	5	Acceptable

## RECORDS REVIEWED

The original police report was available and reviewed. It documented the impact from the large truck, a substantial crush injury to L.G.'s vehicle, and L.G.'s injury. Since this youngster was injured at such an early age, his birth records were secured and reviewed. The hospital records wherein L.G. was treated were obtained, as was the flight record for his transport. There was a large body of follow-up neurosurgical and neurological records available covering more than 3 years of L.G.'s life. His family practice medical records were reviewed as well.

## DIAGNOSES

Using the DSM-IV classification system in a child who has had a brain injury is quite difficult. Phenomenologically, L.G. demonstrates an expressive language disorder, a phonological disorder, and an attention deficit hyperactivity disorder. However, the DSM-IV does not allow for conditions other than developmental to account for these dysfunctions in a child. This in turn may provide difficulty for the forensic neuropsychiatric examiner testifying at court. Use of these terms, since

they are based on developmental delays and not traumatic brain injury, could potentially precipitate a *Daubert* challenge. Thus, the best description one can give of the language disorder in this youngster is an expressive aphasia 784.3 based on ICD-9 (International Classification of Diseases, 9th ed.) classification systems. With regard to the attentional deficits, attention deficit disorder, 314.9, not otherwise specified, may be used from the DSM-IV classification or 314.01 from the ICD-9 classification system. The Axis III diagnoses in this case would include status-post multiple skull fractures from trauma, meningitis, hydrocephalus, and ventriculoperitoneal shunt. His Global Assessment of Functioning at the time of the neuropsychiatric examination was judged to be approximately 40.

## **FORENSIC NEUROBEHAVIORAL ANALYSIS**

From a causation standpoint, the police records are clear. The vehicular damage as a result of frontal impact with a large truck clearly accounted for the skull fractures occurring in L.G. If the forensic examiner is employed in a state where contributory negligence is an issue, the examiner would have to agree that having the child's car seat unrestrained in the front of the vehicle was in all likelihood a contributing factor to L.G.'s injury. The ambulance records and the flight records further document the subject accident as the proximate cause of L.G.'s brain injury.

The reader should recall that children brain-injured before age 5 years are more likely to sustain permanent brain injury from trauma than children injured after age 5 years. The acute imaging studies in this case reveal bilateral subdural hematomas and intraventricular hemorrhage as well. There was evidence of cortical contusion over the right brain convexity. However, on the MRI obtained during the neuropsychiatric evaluation, no significant evidence of structural abnormality could be determined. Moreover, there was no evidence of focal neurological findings on the neurological examination. Thus, from a forensic standpoint, the evidence of injury in L.G. turns primarily on functional matters rather than structural matters.

There is substantial evidence of behavioral dysfunction in this youngster based on the mother's history. With regard to the neuropsychological evaluation, it did not confirm or measure substantial attention and executive dysfunction. This is in contrast to the mother's report wherein she reported that L.G. had difficulty following instructions and listening attentively to a short story. He had difficulty persisting at task to put puzzles together in preschool work. Her report on the BASC-PRS is consistent with hyperactivity and aggression due to the moderately elevated scales in these areas. This evaluation further indicated that L.G. may have a tendency to be unresponsive to adult direction and a tendency to exhibit atypical behavior such as babbling to himself. This suggested that L.G. is at risk for the development of problem behaviors as he ages, and recall in this text that brain-injured children have very high rates of postinjury psychiatric disturbance as they age.

If the reader will review the NEPSY, it can be seen that the visuospatial processing core domain score of 73 is below the expected level for L.G.'s age. L.G. exhibited great difficulty on the Block Design subtest, and these findings are consistent with poor ability to understand and visualize spatial relationships. If the reader will review the WPPSI-R, it can be seen that on the Geometric Design subtest, L.G. demonstrated significant levels of impairment. This correlates with the visuospatial impairment noted on the NEPSY. Thus, there is a very high likelihood that L.G. has a substantial visuospatial injury.

With regard to L.G.'s development, behaviorally he was not yet toilet trained and he continued to use diapers. Thus, he was not at the expected level of development. Moreover, he was yet to develop sharing or cooperative relationships with other children. He demonstrated difficulty taking turns in a game or honoring a simple bargain. Thus, while he may develop these skills as he ages, he is likely to be behind his peers in their acquisition. This most likely will present difficulties as he is integrated into the school system.

Taking his neuropsychological assessment as a whole, L.G. demonstrates substantial cognitive deficits. He revealed impairment in the areas of articulation, oromotor coordination, verbal expres-

sion, verbal and visual concept formation, and the maintenance and formation of abstract cognitive sets. He was below the expected age level on measures of spatial-constructional skills, sensorimotor abilities, and gross motor and fine motor abilities. In all likelihood, these damages were the direct result of his traumatic brain injury at age 3 weeks. Moreover, taking his highest three scores from the WPPSI-R — object assembly, picture completion, and arithmetic — one can state within reasonable medical probability that L.G. would probably have functioned within the average range of cognitive ability had it not been for the traumatic brain injury at age 3 weeks. For the forensic neuropsychiatric examiner testifying at trial regarding L.G., within reasonable medical probability, it could be stated that L.G. will not obtain language at a rate he should have. Due to the very immature age of his brain at the time of injury, L.G.'s ability to express himself narratively will most likely always be impaired. Moreover, testimony can be given that he probably has a lower level of intelligence than he would have had without the brain injury. His behavioral difficulties present particular concerns for the future. The reader may wish to review the section in this text on emotional intelligence. There are substantial indicators that this mental domain may be impaired in L.G. Recall that his relating style is somewhat different than expected for a 4-year-old. Moreover, while the NEPSY contains a language domain, it does not measure the affective components of language significantly. These prosodic elements are far more important for the development and maintenance of emotional intelligence than the semantic portions of language. On the Denver-II, L.G. produced a suspect classification in terms of language. On the Vineland Adaptive Behavior Scale, his adaptive level for socialization skills was moderately low (second percentile).

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# 12 Forensic Report Writing and Testimony in Traumatic Brain Injury Cases

## INTRODUCTION

In forensic neuropsychiatric examinations, in most instances, the written report will be the most important work product following the examination. Virtually all cases referred from a court, and most cases referred from attorneys, will require the preparation of written reports. Reports of forensic evaluations differ in a number of important ways from reports prepared for use in traditional medical practice. The recipients of the forensic report will not be other physicians but legal persons and laypersons who may be unfamiliar generally with the language of neuropsychiatry and behavioral neurology. Moreover, the report content is much more likely to become part of the public domain, as part of a court record or through media coverage as a result of public court proceedings. Therefore, special care should be exercised when writing forensic medical reports.

The report should stand alone on its own merits. The clarity and conclusions in the report may be the final outcome of the neuropsychiatric examiner's opinions. In the vast majority of brain injury cases, the legal issues are mediated or concluded by settlement or agreement and never reach a public courtroom. Thus, the words, style, and analysis of a forensic neuropsychiatric report should be chosen and completed with great care. While testimony is uncommon relative to report writing, it presents a different challenge to the forensic neuropsychiatric examiner. Whereas one's ideas and opinions are conveyed in writing within the forensic report, testimony is provided orally by deposition or to a jury or judge (trier of fact). This poses special obstacles to the forensic physician. One has time to edit writing and time to formulate written ideas. Oral testimony, on the other hand, is taken as it is spoken, and responses to questions are expected immediately. A different skill set is required of the forensic examiner for oral testimony than for written reports.

## FORENSIC REPORT WRITING

### THE PURPOSE AND AUDIENCE

During a forensic neuropsychiatric examination, the examiner cannot be all things to all people. The scope of the examination must be focused, and therefore, the initial factor in the report is the purpose. That should be stated clearly early on in the report. Sometimes physicians producing forensic reports in the context of an independent medical examination omit the purpose and for whom the report is being generated. This is not wise and does not demonstrate objectivity. As noted earlier in this text, forensic examinations should strive for honesty and objectivity, and omitting the purpose and recipient of the report certainly does not demonstrate objectivity. Moreover, it may suggest to others a lack of candor.

If the purpose of the examination is to determine whether a neuropsychiatric impairment resulted from a brain injury, then that should be stated in the report. If, on the other hand, the

purpose is to determine fitness for work, capacity to engage in an act, or other functions, these should be stated clearly. As the report is developed, the examiner should keep in mind the audience. The language style and factual information may be presented differently, depending on the receiving audience. For instance, if the report is being written for the court (a judge), the style can be written scientifically and with less explanation. A judge will be aware of issues at a much higher level than a claims adjuster, for example. The same can be said for lawyers. However, lawyers will be more variable in their understanding of the medicine of traumatic brain injury. If the forensic examiner understands the legal client well, the report can be modified depending on the expertise of the lawyer with matters of brain injury. In any event, one of the main functions of a forensic neuropsychiatric brain injury report is to permit disposition of the case without a formal proceeding. Thus, a well-written, articulate report that satisfies both parties in a litigation or dispute may serve as a basis for negotiation, plea bargaining, or out-of-court settlements in civil cases.<sup>1</sup>

## THE STYLE OF THE REPORT

Regardless of the scientific orientation of the evaluator, or his literary skill, a clear writing of reports is required. The examiner's writing style for forensic reports should stress matching sentences to ideas, linking main ideas, and simplifying descriptions. It is important to develop writing consistency and to speak directly to the reader. Avoidance of run-on or lengthy paragraphs is suggested.<sup>2</sup> In most instances, it is best to write in the active voice. It is much more powerful to say, "The motor vehicle accident caused the brain injury" than it is to say, "The brain injury was caused by the motor vehicle accident." The subject should actively carry the action. For literary style, of course at times the examiner may wish to write in the passive voice, but it is much easier for the reader to follow the logic of the report and to understand it with clarity if the active voice is emphasized. Moreover, it shortens sentences and improves the fluency of the report. For instance, while the active voice is generally more forceful, and a procession of passive constructions is a sure way to cure insomnia, the passive voice is a perfectly legitimate alternative when used by the examiner for variety or to emphasize a key word in a sentence by making it the subject.<sup>3</sup>

While formulating the report, as noted next, the factual portion should be emphasized early in the writing. It is important to separate facts from inferences or conclusions. The factual information taken from the history, the medical records, or the mental status examination should be presented separately from the more theoretical or inferential formulations that may link the clinical data to the question asked by the referring attorney. This style of organization allows the forensic examiner to "build a case" by organizing the investigative data and measurements in a manner that invites the reader of the report to follow the logic along with the author.<sup>1</sup> Regardless of the forensic issue, the forensic brain injury report should contain, at a minimum, the following sections:

1. *Identification Data:* This should include the age of the person being examined, who referred the individual, and the purpose of the examination.
2. *History:* Depending on the clinical orientation of the forensic examination, this may change in style depending on whether the examination is performed by a psychiatrist, neurologist, physiatrist, or other physician. At a minimum, the history should contain a description of the accident or the putative cause of the brain trauma, the past medical and psychiatric history, the family and social history, and the review of systems. In a forensic situation, as noted previously in this text, a separate section listing the records reviewed also should be included.
3. *Mental and Physical Examination:* As noted in [Chapter 4](#), a clear description of the mental status examination and neurological examination should be included in most, if not all, neuropsychiatric examinations for traumatic brain injury. If the reader will note the case reports described next, they contain a short explanatory paragraph or two describing the nature of the mental status examination and the neurological examination.

Physicians often assume that laymen or other nonmedical professionals understand these terms, and often they do not. Thus, it may be useful for the forensic examiner to provide a brief explanation of the nature and scope of the mental status examination and of the neurological examination.

4. *Brain Imaging and Laboratory Data:* If these are performed within the scope of the forensic neuropsychiatric examination, they should be included. Again, if the reader will refer to the reports cited next, a short description of the nature of the brain imaging is included to assist the reader in understanding, for instance, the differences between a positron emission tomography (PET) scan and magnetic resonance imaging (MRI). Using these topical descriptions makes the report more user-friendly.
5. *Standardized Mental Assessment:* This section should clearly state who performed the neuropsychological or psychological testing if it was made part of the neuropsychiatric examination. Since this form of mental testing is not generally performed by physicians, in order to maintain clarity and honesty in the report, whoever provided these services to the physician should have his name within the body of the report, and his highest academic degree should follow his name. Thereafter, it is useful, and continues with the user-friendly nature of forensic reports, to describe each particular test and provide a short statement of the purpose of the test. Recall that the forensic report may well become a legal document. It is not unusual for forensic neuropsychiatric examination reports to be made part of the legal record during a deposition or, in some instances, introduced at trial. Therefore, the numerical data should be contained within the body of the report. It is not enough to merely make conclusory statements such as “The Halstead–Reitan Neuropsychological Battery demonstrated traumatic brain injury.” The numerical data consistent with brain injury should be included within the body of the report to convey honesty and transparency.
6. *Records Reviewed:* This section of the report should list all records used by the forensic examiner within the scope of the neuropsychiatric examination. Moreover, as a purely logistical matter, it is also helpful to have the records indexed by the same number as in the report. This will enable the forensic examiner to quickly find information when giving a deposition or when testifying at trial, especially if cross-examination questions are asked about particular medical records.
7. *Forensic Neurobehavioral Analysis:* This is a term of art, and other similar rubrics are clearly appropriate depending on the particular orientation of the physician. Some examiners may wish to call this section Bases for Conclusions, Diagnostic Formulation, or any other appropriate term.
8. *Diagnoses:* This section should include all the diagnostic conclusions made by the examining physician.
9. *Conclusions:* At this point, conclusory statements can be made. These should directly comport with the original referral question from the attorney, judge, or other party who requested the examination.

When writing the forensic neuropsychiatric report, it is important to stay within the scope of the referral question. It is best not to stray far from the referral question or to expand into areas wherein the examining physician is poorly qualified. The examiner should confine exploration of the examinee’s life to issues legitimately raised by the question of brain injury. In a neuropsychiatric examination, however, this may be a broader area than if the examination were performed merely for orthopedic reasons. For instance, as noted previously in this text, a person’s behavior prior to brain injury is a legitimate issue if the matter of brain injury has been raised by a plaintiff. Moreover, stressful family and social factors may play a role in a person’s mental state at the time of the neuropsychiatric examination. Thus, from a behavioral standpoint, the content of a neuropsychiatric

examination may be much more broad than if the examination is performed by a neurosurgeon, psychiatrist, or other physician.

It is important not to err by failing to address issues that have been raised in the referral question. Thus, a lawyer may ask multiple questions with regard to a traumatic brain injury that go beyond causation of brain injury. As noted previously in this text, issues of damages, outcome, impact upon family relationships and marriage, and other issues may also need to be addressed expertly by the examining physician. On the other hand, it is not recommended to offer gratuitous opinions on issues that have not been raised by the referral questions. This is particularly true if the examiner is providing an evaluation within a criminal matter. In those instances, the referral questions should be explicitly followed, and gratuitous information that may have an impact on criminal issues outside the scope of the questions asked by the court or lawyer should be avoided.

From the standpoint of clarity, it is important to explain clinical jargon where necessary and to avoid complex terms where possible. However, brain injury reports most often are being written for judges or attorneys, and a certain level of medical expertise can be expressed, but one wants to be careful and not provide overkill in this matter. Thus, it is important to revise, edit, and, where necessary, rewrite one's report with the constant goal of clarity in mind. Ernest Hemingway was asked about his writing style on one occasion. He noted that while writing *A Farewell to Arms*, he rewrote the last page 39 times in order to "get the words right."<sup>4</sup> It is not recommended to be this compulsive about writing one's forensic report, but clearly editing is important. The editing should focus upon clarity, style, and, where possible, reductionism. Where a few words can convey an idea, this is always preferable to large, run-on paragraphs that produce eyelid ptosis in the reader.

## USE OF WORD PROCESSING TEMPLATES

It is best not to "reinvent the wheel" each time the forensic evaluator writes a neuropsychiatric report following a brain injury examination. In order to enhance clarity and productivity, it is recommended that repetitive portions of a neuropsychiatric report be placed into word processing as templates. For instance, in the report examples that follow, the descriptors of various psychological tests, the descriptors of the mental status examination or neurological examination, and other similar entities can be inserted as templates, which are then available for insertion into the neuropsychiatric report where appropriate. This reduces cognitive strain upon the examiner and cognitive strain upon the clerical staff of the examiner. Moreover, it assists in standardization of the report and prevents the accidental oversight of information important to the report. The same can be said for subject headings. A forensic report written paragraph after paragraph with no explanatory headings to help direct the reader is extremely frustrating to the lawyer or judge. Not only is it polite to provide subject headings within the body of the report, it is also logical and provides a structural outline of the forensic report for the reader. Subject headings such as History, Mental Status Examination, Neurological Examination, etc., can be kept as templates as well.

## ANALYSIS AND CONCLUSIONS

This section may be the most important element of the forensic report. The intellectual elements of preparing this section have been discussed previously in [Chapters 9 to 11](#). It is recommended that the forensic neuropsychiatric examiner approach a forensic brain injury examination by formulating a hypothesis in the null. For instance, approach the neuropsychiatric examination of a plaintiff and the analysis of that examination as the following statement: "The subject motor vehicle accident did not produce a brain injury in the examinee." By examination and scientific testing, the examiner should attempt to prove this hypothesis regardless of who hired the physician. This assists in the removal of prejudice toward the examination. This approach enhances the likelihood of an honest and objective examination. A prejudiced examiner is one whose opinions are preconceived; he forms them without paying due attention to the evidence. The only reasonable ground

for holding a conclusion is that the facts require the examiner to do so. To be guilty of prejudice is to have jumped to unwarrantable conclusions, to believe what is comfortable to believe, or to let thinking be influenced by one's feelings.<sup>5</sup> Some examiners may fret that they will never get a complete certainty in inferring causes from their effects. In a brain injury examination, this may well be true; but in practice, we can never be completely certain in predicting effects from causes for any matter. This should not unduly worry or affect the forensic examiner, for the level of certainty required at court in a civil brain injury action is "reasonable medical probability," that is, more likely than not or a probability greater than 50%.

It is important that the Analysis and Conclusions sections of the forensic report follow the collection and analysis of the data in a linear fashion and to present the conclusions in a reader-friendly format that can be understood by almost any intelligent person using the examiner's report. We want the examiner, if the results of the examination indicate that brain injury occurred, to follow that A causes B. On the other hand, if no brain injury occurred and the examiner's data are consistent with lack of brain injury, we also want the reader of the report to understand that A did not cause B. In other words, our report should be clear enough that the reader can understand that an injury to the head does not always produce a brain injury, but in some circumstances, injury to the head does produce a brain injury. Depending on which outcome proceeded following the trauma to the head, the reader should be able to follow the logic of the report and understand the conclusions of the examiner.

## **DICTATING THE REPORT**

The last point about report writing concerns dictation. Most reports written by physicians are dictated. Oftentimes, however, reports appear to be "stream of consciousness" due to the disorganized dictating style of the physician. The dictation should take place in an area where it is quiet enough for the physician not to be distracted by extraneous noise. Moreover, it is not wise to produce reports that say "dictated but not read." Many physicians live by this motto, and for forensic report writing, this is unacceptable.

To assist with proper transcription of dictated forensic reports, it is necessary to speak more slowly than one would speak in normal conversation. This is to improve articulation, as audiotapes degrade spoken speech somewhat and the transcriptionist may not hear the word clearly if it is pronounced rapidly. Moreover, many physicians pride themselves upon how quickly they dictate, and they do so in a rapid, staccato-like fashion that leads to increased errors when transcribed. Another key issue is to think before dictating.<sup>6</sup> It is important to organize in the mind the schema for dictating the report. As noted previously, if templates are placed into dictation formats, this removes the onus from the physician and will improve the quality of the dictation and standardize the dictated report. If this is done repeatedly by the physician, it also permits the forensic examiner to reflect upon medical reasoning while the report is being dictated. The dictation of each report practices within the mind of the physician the reporting format and improves the likelihood that medical reasoning will remain logical and organized from report to report. Three reports of actual traumatic brain injury cases are presented next. While these reports reflect the literary and thinking style of the author, they may or may not apply to individual forensic examiners. Moreover, the reports more closely fit a neuropsychiatric format, and neurologists, psychiatrists, and other physicians performing forensic examinations may prefer a different style. Thus, these reports are submitted for educational purposes only, and the forensic physician should pick a style that fits his or her organizational needs.

### **REPORT 1: RIGHT DEPRESSED TEMPORAL BONE FRACTURE IN AN ADULT**

This report demonstrates a suggested disclosure of a neuropsychiatric examination following head trauma that resulted in a right depressed temporal bone fracture. This is a complex injury that

resulted in cognitive impairment, dementia, and personality changes in the victim. The examination was performed on behalf of a plaintiff attorney. This report demonstrates an actual case example of a traumatic brain injury. Names, locations, and other sensitive data have been changed to protect the identities of the examinee, physicians, hospitals, locations, and other recognizable data. The facts and test results remain unchanged.

September 10, 2002

Mr. Plaintiff Attorney  
Foxtrot, Oscar and Charlie, P.L.C.  
Somewhere, Illinois  
RE: A.Z. v. *John Defendant*

Dear Mr. Attorney:

I examined A.Z. at my offices on August 1 and 2, 2002. My complete examination and testing required 11 hours. Two hours were devoted further to reviewing medical records, reviewing brain scans, reviewing my test data, and preparing this report. This was a neuropsychiatric examination of five components. The first component consisted of taking Mr. Z.'s history of the accident and how it has affected him mentally. Further history was obtained regarding Mr. Z.'s past medical history, family history, and social history.

The second component consisted of a two-part mental examination. The first part consisted of a face-to-face qualitative mental status examination, and the second part consisted of the administration and interpretation of standardized mental test instruments. The third component was a neurological examination, while the fourth component consisted of MR brain imaging. The fifth component consisted of reviewing available medical and other records, analyzing my data, performing a neurobehavioral analysis, and reducing my findings to this report.

#### **IDENTIFICATION DATA**

A.Z. is a 42-year-old male plaintiff from Somewhere, Illinois. He is being examined at the request of his attorney to determine if he retains neuropsychiatric impairment as a result of a motor vehicle accident.

#### **HISTORY FROM THE RECORDS**

On or about March 2, 2001, Mr. Z. was in a motor vehicle accident. He was first evaluated at the Somewhere Methodist Hospital emergency department. The helicopter crew injected him with midazolam, vecuronium, and normal saline intravenously. He had loss of consciousness, but the duration was unknown. When attended, he was noted to be apparently an unrestrained driver of a van, and he was found lying on the passenger side of the vehicle. The Somewhere Methodist Hospital record reported that he had a fair amount of recollection. He was complaining of pain to the right side of his head. While he knew who he was, he "had to think a bit to know how old he was." His right ear was missing. CT scan revealed major injuries to the skull base and demonstrated a tripod fracture of the right orbit with a right temporal bone fracture depressed approximately 5 millimeters. He had a 5-millimeter subdural hematoma noted on CT scan. Urine drug abuse screen was positive for benzodiazepines, but this is accounted for by the prior administration of midazolam. However, urine drug abuse screen was also positive for cocaine, which was not administered to him by the emergency squad. A third agent, opiates, was positive as well.

At a University Hospital in Chicago, Illinois, Mr. Z. was noted to have had a closed fracture of the skull vault with subarachnoid and subdural hemorrhage. Pneumocephalus was noted on CT scan. Multiple facial fractures were present. The right temporal fracture required open reduction. The last CT scan available to me was obtained March 8, 2001. It revealed a left frontal extraaxial hematoma

with a left frontal hemorrhagic contusion. No midline shift or mass effect was seen. Blood was seen along the falx posteriorly.

### **HISTORY FROM MR. Z.**

Mr. Z. completed a 22-page medical questionnaire. I verified this document with him face-to-face. He was given written warning that his information might not be confidential. Warning was also given that we would not have a doctor–patient relationship, and I would provide no treatment or counseling.

At today’s examination, he reports psychiatric symptoms of depression, sadness, nervousness, poor concentration, and loss of memory. He has short-term memory loss, and his brothers remind him that he repeats himself. His balance is poor since the accident. He also notices that his visual field in the right eye is constricted and he has tunnel vision. He has difficulty seeing either to the right or the left with the right eye. He also reports hearing loss in the right ear and tinnitus in the right ear.

### **ACTIVITIES OF DAILY LIVING**

He lives with a girlfriend and a number of children. He arises about 8:00 A.M. and retires at 11:00 P.M. He is not employed currently. Prior to the accident, he was an automobile repossession agent. His hobbies are taking care of his chickens and repairing an automobile. He reads the local trade magazines. He can write and watch television. He doesn’t mow the yard. He rents about eight movies a month to watch in the home, but he doesn’t attend ball games or hunt or fish. He can’t eat outside the home socially. He doesn’t use the telephone. He can dress and bathe himself independently. He reports he’s sexually functional.

### **PAST MEDICAL HISTORY**

He was born a 7-pound, 10-ounce full-term baby. His developmental milestones were normal and he sustained no birth injury. He had difficulty learning in school and difficulty keeping his mind on tasks as a youngster. As an adult, his primary medical problem has been hypertension. He was injured in a motor vehicle accident in 1976 at age 16 and sustained some cuts on his head and neck, but he had no cognitive sequelae.

His present medications are:

1. Lisinopril, 20 mg daily
2. Metoprolol, 100 mg daily
3. Paroxetine, 10 mg daily
4. Hydrocodone, 7.5 mg once or twice daily
5. Amoxicillin, 1500 mg daily

He uses over-the-counter aspirin but no herbs or natural products. He doesn’t use tobacco products. He drinks two six-packs of beer weekly. He has a past history of using marijuana and cocaine. He drinks coffee on the weekends and colas during the week.

### **PAST PSYCHIATRIC HISTORY**

He’s never been treated formally for a psychiatric illness, but he was anxious in the early-1990s and he would receive alprazolam from friends. He’s never been hospitalized for psychiatric, drug abuse, alcohol, or mental problems. He’s never formally received counseling or psychotherapy. He’s never intentionally overdosed himself on drugs or medicines, and he’s never made an attempt to take his life. He’s never intentionally cut, burned, or disfigured himself.

## **FAMILY HISTORY**

His father died of lung cancer and his mother died of leukemia. A 12-year-old daughter was born with epilepsy and is treated with valproate. A brother is an alcoholic and also depressed. He denies any family history of suicides, homicides, violence toward others, child abuse, or spouse abuse. He denies any other neurological diseases, Alzheimer's disease, or strokes in his family.

## **SOCIAL HISTORY**

He was born in McLean County, Illinois, and he's one of five children; he has four brothers. His father was employed as a corn and soybean farmer and his mother was a homemaker. His father died in 1999 and his mother died in 2001, but both parents were present in the home when he was young. His father did not abuse his mother.

He denies he's ever been sexually or physically abused. He does have a history of violence toward others. He owns a .22-caliber rifle but denies plans to harm himself or anyone else. He's never been in difficulty due to his sexual behavior. He skipped school on occasion, and in the ninth grade, he dropped out of school when he was not allowed to return following an absence without receiving corporal punishment. He then chose not to return to school.

He married his first wife, Sue, in 1977. Their marriage produced three children who are now ages 19, 21, and 23 years. They couldn't get along and divorced in 1984. He married Angie in 1987, and this marriage produced four children who are now ages 10, 12, 13, and 15 years. He couldn't get along with Angie, and they divorced in 2000. He currently has a girlfriend and lives with her.

## **LEGAL HISTORY**

He was convicted of a DUI in McLean County in 1997. He's never been a party in a lawsuit. He's never been a party in a restraining order or emergency protective order. He's never been charged with spouse abuse, child abuse, or terroristic threatening. He does have a prior workers' compensation claim for a back injury. He's never declared bankruptcy. He has filed for Social Security Disability benefits and he currently receives a welfare check.

## **EMPLOYMENT/VOCATIONAL HISTORY**

He last worked as an auto recovery repossession agent from September 1997 until March 2001.

## **MILITARY HISTORY**

He served in the U.S. Army from October 1977 until September 1980. He received an honorable discharge, but he was reduced in rank for leaving a weapon unattended. He served in the mechanized infantry and operated an armored personnel carrier.

## **REVIEW OF SYSTEMS**

His general review is negative. In his HEENT review, he has tunnel vision in the right eye due to trauma to that eye. He also reports hearing loss in the right ear and tinnitus in the right ear. His chest review is negative. In his cardiovascular review, he has hypertension. His gastrointestinal review is negative, and the genitourinary review is negative.

His psychiatric review is noted above. Neurologically, he reports poor balance. His musculoskeletal review is positive for joint pain and difficulty with mobility. His sleep is disturbed due to a sleep continuity disturbance.



## MENTAL STATUS EXAMINATION

The mental status examination is a face-to-face examination between the psychiatric physician and the patient. The purpose of the examination is to determine the function of the elements of mental and brain activity. For instance, thought is examined to determine if the person can go from point A to point B logically in his thinking. Thought is also examined to determine the presence or absence of circumstantial thinking, loose associations, or other determinants of abnormal mental function. Content of the thinking is examined for delusional thoughts, morbid ideas, perceptual distortions, suicidal/homicidal ideas, or other signs of mental pathology. Language is examined for expressive and receptive function, repetition errors, and to determine if the person can take mental ideas and properly convert them to motor acts.

Orientation to person, place, and time is determined. Gross memory ability is determined. Evaluation of the mental stream of activity, mood, range of affect, and thought and motor speed is completed. The mental status examination is a qualitative examination, and quantified elements of the mental examination are determined by standardized mental assessment below.

He's a pleasant, cooperative man. He has a large tattoo over the right arm. He is a capable historian, and he independently completed a 22-page historical questionnaire. He is oriented to person, place, and time. Affective range is constricted. Mood is subjectively reported as depressed and anxious. He denies suicidal ideas or plans. There are no delusions or hallucinations present. There is no evidence of loose associations or circumstantial thinking. Articulatory agility is reasonably good. The melodic line and phrase length both appear to be within normal limits. There are no paraphasias or word-finding difficulty noted.

## NEUROLOGICAL EXAMINATION

The neurological examination is a physical examination performed by the physician. The purpose of the examination is to measure gross neurological functioning of the input and output nerves of the brain, as well as cerebral hemisphere functioning.

Weight is 228 pounds. Blood pressure is 140/92 in the left arm. There are no bruits in the neck or head. The face reveals no signs of trauma. The face is symmetric. Hand dominance is crossed.

### *Cranial Nerves*

In the optic group, there is a constricted right visual field. Ocular motility is full. The pupils are equal and reactive to light and accommodation. The fundoscopic examination is benign.

In the branchiomotor group, masseter and temporalis muscles are intact. The forehead rises equally, the eyelids close, and the lips purse. The gag reflex is present. *E* is well phonated. There is no nasal speech present. The SCM and trapezius muscles are intact. The tongue protrudes midline and is without atrophy or fasciculations.

In the cranial sensory group, anise and peppermint oils are poorly appreciated. Taste was not tested. Tuning fork sound is perceived normally in the left ear and reduced in the right ear. The Weber sign is lateralized right. In the somatic cranial sensory group, the corneal reflexes are intact;  $V_1$  is intact,  $V_2$  is intact, and  $V_3$  is intact.

### *Somatic Motor*

Bulk and tone are normal. Strength is symmetric. Right grip strength is 44 kg. Left grip strength is 49.5 kg. Outstretched arms reveal no pronator drift. Deep tendon reflexes are symmetric at the biceps, the brachioradialis, the triceps, the patellar tendons, and the Achilles tendons.

### *Somatic Sensory*

Light touch and pinprick are intact. Vibratory and position sensation are intact. The Romberg position reveals no sway.

### *Cerebellar/Vestibular*

Nystagmus is not present vertically or horizontally. Finger–nose function is performed well. Heel–shin function is normal. Dysdiadochokinesia is not present.

### *Gait and Station*

Gait analysis reveals normal stride. Heel rising is intact. Toe rising is intact. Squatting and rising are intact. Arm swing is normal. Motor speed is normal. Heel–toe walking is dystaxic.

## **MAGNETIC RESONANCE IMAGING**

MRI was obtained at the University Hospital on August 2, 2002. The intracranial study reveals findings consistent with encephalomalacia. This chiefly involves the left frontal lobe with loss of tissue and evidence of focal gyral atrophy over the left frontal pole. There is a separate focal area over the posterior left frontal convexity surface. The corpus callosum remains intact. The hippocampi are symmetrical. [Figure 12.1 demonstrates the left frontal MRI encephalomalacia. This figure was not displayed in the original report but was presented at deposition.]

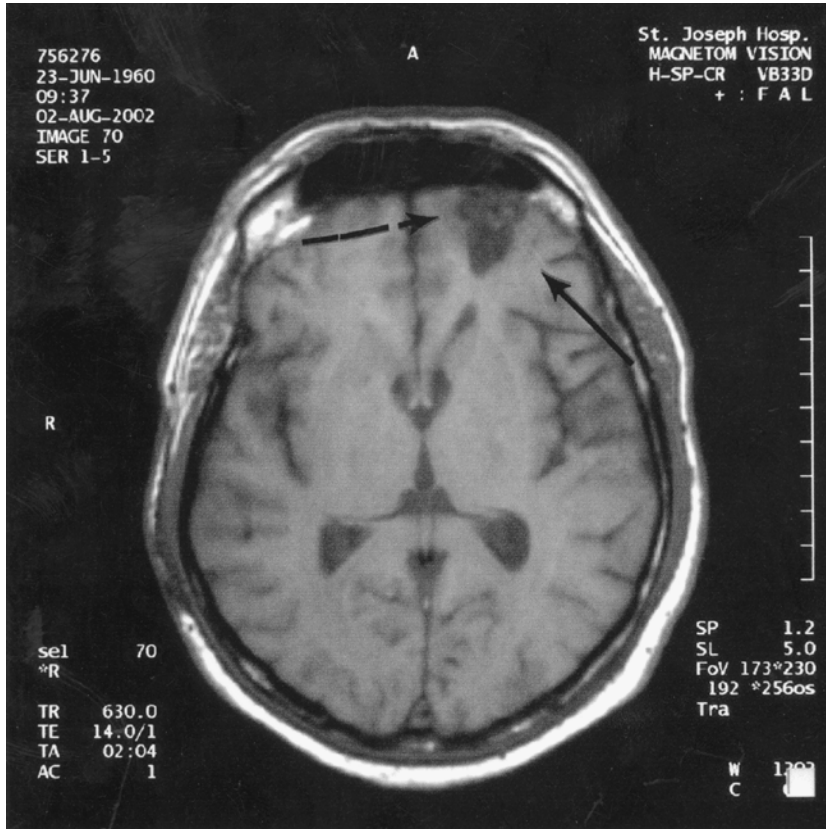
## **STANDARDIZED MENTAL ASSESSMENT**

Forensic Doctor, M.D., requested formal neuropsychological testing. These tests were performed by Jane Psychologist, M.A. Alice Psychologist, Ph.D., supervised the administration, scoring, summarization, and analysis of the test data. These persons are agents or employees of Forensic Doctor, M.D., P.S.C. Medical conclusions about these data were made by Forensic Doctor, M.D.

*Standardized* means that the administered test instruments had exact rules for test administration and exact rules for test scoring. These rules have been standardized and published and are expected to be utilized by any person performing or interpreting the following test instruments.

The following test instruments were administered:

1. Test of Memory Malinger
2. Letter Memory Test
3. Victoria Symptom Validity Test
4. Wide Range Achievement Test-III, Reading subtest
5. Wechsler Test of Adult Reading
6. Ruff 2 and 7 Selective Attention Test
7. Brief Test of Attention
8. Boston Naming Test
9. Controlled Oral Word Association Test
10. Reitan–Kløve Sensory Perceptual Examination
11. Wechsler Memory Scale-III
12. Grooved Pegboard Test
13. Grip Strength Test
14. Finger Tapping Test
15. Wisconsin Card Sorting Test
16. Trail-Making Tests A and B



**FIGURE 12.1** This is a T1-weighted MRI taken 17 months after a motor vehicle accident causing a right temporal bone fracture. The encephalomalacic lesion is noted in the left temporal pole consistent with contrecoup injury.

17. Wechsler Adult Intelligence Scale-III
18. Minnesota Multiphasic Personality Inventory-2

### **BEHAVIORAL OBSERVATIONS DURING PSYCHOLOGICAL TESTING**

Mr. Z. is a 42-year-old male who has completed 9 years of education; he is currently not employed. On March 2, 2001, Mr. Z. was involved in a single-vehicle accident. He suffered head trauma, predominantly on the right side. His right ear was also severed at the time of the accident. According to medical records, Mr. Z. admits to driving under the influence of alcohol. A CT scan of the head at the time of the injury reveals a depressed skull fracture of the parietal lobe with free air in the cranium. Mr. Z. was kept medically paralyzed for nearly 3 weeks following the injury. Currently, he reports difficulty with tunnel vision of the right eye, right-side hearing loss, high blood pressure, sleep disturbance, depression, sadness, nervousness, concentration difficulties, and loss of memory.

Mr. Z., accompanied by his girlfriend and his girlfriend's child, arrived on time for the purpose of a neuropsychological evaluation. Mr. Z. was appropriately and casually dressed in blue jeans and a pullover shirt. He was appropriately groomed and appeared to be his stated age of 42. Time was spent reviewing the evaluation process, and Mr. Z. was given the opportunity to ask questions. Mr. Z. was pleasant and friendly, and he displayed appropriate affect and mood. His speech was clear and coherent, with no apparent receptive or expressive language difficulties. He remained cooperative throughout the evaluation, answering all questions and attempting all requested tasks. Rapport was easily established.

Mr. Z. seemed to understand and follow directions without difficulty, although he sometimes seemed reluctant to attempt more difficult items. Mr. Z.'s responses to questions in general, and to items on the WAIS-III verbal subtests especially, were rather brief and often required the examiner to query incomplete or unclear responses. Mr. Z. also seemed to have difficulty completing timed tests in a rapid manner. This was evidenced by rather slow performance on test measures where the test directions explicitly instructed him to "work as quickly as you can." It was obvious that Mr. Z. was concerned about his performance in this evaluation. Several times throughout the assessment, Mr. Z. would shake his head and attribute his perceived deficits to his injury. He further appeared frustrated with difficult items.

Mr. Z. is left-hand dominant and appeared to favor this hand when writing. However, he appeared to favor both hands during tasks involving manipulation of objects, such as Block Design. Overall, Mr. Z. seemed to provide a good effort on the measures administered in this evaluation.

## FORENSIC DISTORTION ANALYSIS

With any standardized mental assessment the recruitment of optimal effort is imperative. The instruments used to assess brain functioning cannot be "faked good." It is impossible for a brain to perform better than its capacity. However, it is possible for individuals to attempt to present themselves in a negative manner, to "fake bad" or present a "worst-foot forward." The extreme example of this presentation results in a response pattern referred to as malingering.

The following instruments were administered to determine the level of effort and validity of responses:

### A. Measures of Cognitive Distortion

#### 1. *Test of Memory Malingering*

The Test of Memory Malingering (TOMM) is a 50-item test for adults published in 1996 that is designed to discriminate between malingered and authentic memory impairments. Although the TOMM is sensitive to malingering, it is insensitive to neurological impairment. The TOMM has been extensively validated with groups of cognitively intact adults and with groups of adults diagnosed with cognitive impairment, aphasia, or traumatic brain injury. The TOMM consists of two learning trials and a retention trial. A low score on the TOMM suggests that memory impairment symptoms are false or exaggerated. Mr. Z. produced a score of 50 on Trial 2. This pattern of performance is within normal limits.

#### 2. *Letter Memory Test*

The Letter Memory Test (LMT) is a motivational test developed in 1998 for the assessment of effort. The LMT is a computer-administered test that requires the recall of increasingly difficult sets of letters for a 5-second time interval. Poor performance is indicative of a motivational deficit. Mr. Z. produced a percentage score of 100, which is within normal limits.

#### 3. *Victoria Symptom Validity Test*

The Victoria Symptom Validity Test (VSVT) is based on a forced-choice paradigm that requires digit recognition after increasing periods of delay following stimulus presentation. Recognition items vary in degree of similarity and appear to be easy or difficult. Although difficult items are designed to appear significantly more challenging than easy items, actual differences in performance across these two types of items are minimal in normal individuals. Results are provided for the number of easy, difficult, and total items correct, as well as the number of items correct and response latencies in each block. VSVT classifications are determined on the basis of binomial probability theory and are specifically designed to eliminate false positives.

The results of the VSVT for Mr. Z. are summarized as follows:

### Victoria Symptom Validity Test

	Raw Score	Classification
Easy items correct	23/24	Valid
Difficult items correct	23/24	Valid
<b>Total items correct</b>	<b>46/48</b>	<b>Valid</b>

Overall, the results of this measure are within the valid range.

**B. Measures of Psychological Distortion**

*MMPI-2 Validity Indices*

MMPI-2 validity indices are derived from the MMPI-2 responses. The MMPI-2 is the most recent (1989) revision of the original 1943 MMPI. In addition to the four previous validity scales — Cannot Say, L, K, and F scales — five additional scales are provided: back-page Fb, TRIN, VRIN, Fp, and S.

Mr. Z. produced the following profile:

Scale	Cannot Say	VRIN	TRIN	F	Fb	Fp	F-K	L	K	S
Raw	0	6	12	11	7	1	1	3	10	21
T-score		54	72	70	71	48		48	39	45

Results of the MMPI-2 validity measures are consistent with a psychological profile that is most likely valid, although characterized by an inconsistent response style in which Mr. Z. tends to respond true in an inconsistent manner. This may have contributed to the elevation of the F scale, although a problem-oriented approach to items cannot be ruled out. Hence, caution is warranted interpreting the clinical scales. The elevation of the Fb scale precludes interpretation of the supplementary and content scales.

**MEASURES PROVIDING ESTIMATES OF PREINJURY FUNCTION**

1. *Wide Range Achievement Test-III, Reading subtest*

The Wide Range Achievement Test-Revised (WRAT-III) is a screening measure for academic achievement. It assesses reading recognition, spelling, and arithmetic. Raw scores are determined in each of these areas, and grade equivalence and percentiles are assigned. Standard scores are also obtained, in which the mean (average) is represented by a standard score of 100, and the standard deviation is 15. Standard scores are used for interpretation. Reading scores tend to resist dementing processes.

Mr. Z. produced the following raw and derived scores:

**Wide Range Achievement Test-III**

	Raw Score	Standard Score	Percentile	Grade Equivalence
Reading	40	81	10	8

Mr. Z.'s performance on this measure is in the low average range.

2. *Wechsler Test of Adult Reading*

The Wechsler Test of Adult Reading (WTAR) is a reading recognition test developed in 2001 to provide an assessment tool for estimating premorbid intellectual ability of adults ages 16 to 89. Reading recognition ability is relatively stable in the presence of cognitive declines associated with normal aging or brain insult. The WTAR was developed in conjunction with the WAIS-III and WMS-III. WAIS-III and WMS-III test scores are predicted from a combination of WTAR performance and demographic variables.

The WTAR has been validated for use with individuals diagnosed with disorders associated with cognitive decline, such as Alzheimer's dementia, Parkinson's disease, Huntington's chorea, and Korsakoff's syndrome. In addition, the WTAR has been validated with individuals who have been diagnosed with acute brain damage due to traumatic brain injury. The WTAR has also been validated with individuals diagnosed with neuropsychiatric and developmental disorders.

Mr. Z. produced the following WTAR scores:

**Wechsler Test of Adult Reading**

Raw Score	Standard Score
18	77

## WTAR Demographic-Predicted WAIS-III and WMS-III Indices

	Standard Score	Percentile	Classification
WAIS-III VIQ	86	18	Low average
WAIS-III PIQ	88	21	Low average
WAIS-III FSIQ	86	18	Low average
WAIS-III VCI	85	16	Low average
WAIS-III POI	90	25	Average
WAIS-III WMI	89	23	Low average
WAIS-III PSI	87	19	Low average
WMS-III Immediate Memory Index	86	18	Low average
WMS-III General Memory Index	87	19	Low average
WMS-III Working Memory Index	90	25	Average

Given the difference of 16 points between Mr. Z.'s standard score of 77 and the demographics-predicted score of 93, caution is warranted interpreting the predicted intellectual and memory functioning scores above.

### 3. *Vocabulary subtest of the WAIS-III*

This subtest tends to resist dementing brain disorders. Mr. Z. produced the following scaled scores on the Vocabulary subtest of the WAIS-III:

#### WAIS-III Subtest

	Scaled Score	Standard Score	Percentile	Classification
Vocabulary	6	80	9	Low average

## ATTENTION AND CONCENTRATION

### 1. *Ruff 2 and 7 Selective Attention Test*

The Ruff 2 and 7 Selective Attention Test measures two overlapping aspects of visual attention:

A. Sustained attention

B. Selective attention

Sustained attention refers to the ability to maintain a consistent level of visual performance over an extended period. Selective attention refers to the ability to select relevant visual stimuli (targets) while ignoring salient but irrelevant stimuli (distracters).

Mr. Z.'s scores on the 2 and 7 Selective Attention Test are:

#### Ruff 2 and 7 Selective Attention Test

	Measure	Raw Score	T-Score	Percentile	Classification
Letters	Automatic detection speed	103	40	16	Below average
	Automatic detection errors	5			
	Automatic detection accuracy	95.37	46	34	Average
Digits	Controlled search speed	103	42	21	Below average
	Controlled search errors	6			
	Controlled search accuracy	94.50	51	55	Average

#### Total Scores

	Measure	Sum of T-Scores	T-score	Percentile	Classification
	Total speed	82	43	25	Below average
	Total accuracy	97	48	42	Average

2. *Brief Test of Attention*

The Brief Test of Attention (BTA) is an auditory perception task developed for use with adults ages 17 to 84 who are able to distinguish between spoken numbers and spoken letters of the alphabet. Designed to measure an individual's ability to divide auditory attention, the BTA consists of a series of numbers and letters of increasing length that are presented to the respondent via audiocassette. The BTA consists of two subtests. During the Form N subtest, the respondent is required to disregard the letters presented and report how many numbers were read aloud for each series of numbers and letters presented. During the Form L subtest the respondent is required to disregard the numbers presented and count how many letters are read aloud. The number of correctly monitored series is computed for each subtest, with total raw scores ranging from 0 to 20. The total raw score is then converted to a percentile value according to the respondent's age.

Mr. Z. produced the following profile:

**Brief Test of Attention**

	<b>Raw Score</b>	<b>Percentile</b>	<b>Interpretation</b>
Form N (numbers)	6		
Form L (letters)	7		
<b>BTA total score</b>	<b>13</b>	<b>24</b>	<b>Low average</b>

3. *Digit Span Subtest of the Wechsler Adult Intelligence Scale-III*

The Digit Span subtest in the Wechsler Adult Intelligence Scales measures span of immediate verbal recall. It is comprised of two different tests, digits forward and digits backward. These involve different mental activities and are affected differently by brain damage. Both measure short-term storage capacity for auditory stimuli and require auditory attention as well as intact short-term auditory retention capacity.

Digits forward primarily measures the efficiency of auditory attention. Digits backward calls upon working memory and involves mental double-tracking in that both memory and the reversing operations must proceed simultaneously.

Mr. Z. produced the following results on the Digit Span subtest:

**WAIS-III Digit Span Subtest**

	<b>Raw Score</b>	<b>Standard Score</b>	<b>Percentile</b>	<b>Classification</b>
Longest digit span forward	5	81	10	Low average
Longest digit span backward	4	91	27	Average
Digit span scaled score	7	85	16	Low average

**LANGUAGE AND LANGUAGE-RELATED SKILLS**

1. *Boston Naming Test*

The Boston Naming Test is a 60-item test containing drawn pictures of objects ranging in difficulty from a bed to an abacus. The pictures have been selected so as to eliminate items that have alternative acceptable names. The Boston Naming Test is particularly useful for detecting relatively mild word-retrieval problems. Raw scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation.

Mr. Z. produced the following profile:

**Boston Naming Test**

Raw score	37
T-score	16
Classification	Severely impaired
Percentile	<1

2. *Controlled Oral Word Association Test*

In the Controlled Oral Word Association Test (COWA), the test taker is asked to produce as many words as possible beginning with a given letter in a specified period of time. This test measures word production. *F*, *A*, and *S* are the most commonly used letters for this test. The score is the sum of all admissible words for the three letters. This score is adjusted for age, sex, and education. The adjusted scores are converted to percentiles.

Mr. Z. produced the following results:

**Controlled Oral Word Association Test**

Raw score	27
T-score	37
Percentile	10
Classification	Severe defect

**MEMORY**

*Wechsler Memory Scale-III*

The Wechsler Memory Scale-III (WMS-III) is the most recent revision of the Wechsler Memory Scale. There are 11 different subtests that are combined to form eight different memory indices. These indices have a mean of 100 and a standard deviation of 15.

Mr. Z. produced the following profile:

**Wechsler Memory Scale-III**

	Scale Score			
	Sum	Index Score	Percentile	Classification
Auditory immediate	15	86	18	Low average
Visual immediate	13	78	7	Borderline
Immediate memory	28	78	7	Borderline
Auditory delayed	13	80	9	Low average
Visual delayed	18	94	34	Average
Auditory recognition delayed	8	90	25	Average
General memory	39	84	14	Low average
Working memory	18	93	32	Average

**SENSORY PERCEPTUAL SKILLS**

*Reitan-Kløve Sensory Perceptual Examination*

The Reitan-Kløve Sensory Perceptual Examination consists of three measures: bilateral simultaneous sensory stimulation, fingertip writing, and tactile finger recognition.

A. *Bilateral Simultaneous Sensory Stimulation*

This group of tests assesses how accurately the test taker can perceive bilateral simultaneous sensory stimulation after it has been established that perception of unilateral stimulation of each side is essentially intact. Examination is done for touch perception, auditory perception, and visual perception.

B. *Fingertip Writing*

Fingertip writing requires the test taker to report numbers written on the fingertips of each hand without the use of vision. The score represents the number of errors for each hand.

C. *Tactile Finger Recognition*

Tactile finger recognition requires the test taker to identify which finger was touched following tactile stimulation. A blindfold is used for this procedure, or it is done with eyes closed. Twenty trials are performed on each hand.

Reitan-Kløve Sensory Perceptual Examination error scores are totaled and converted to T-scores in which 50 is the mean and 10 is the standard deviation.



Mr. Z. produced the following profile:

### Sensory Perceptual Examination

	Raw Score	T-Score	Classification	Percentile
Total right errors	6	36	Mildly impaired	8
Total left errors	3	42	Below average	21
Sensory perceptual total	9	41	Below average	19

### MOTOR AND VISUAL MOTOR SKILLS

#### 1. Grooved Pegboard Test

The Grooved Pegboard Test is a manipulative dexterity test. It consists of 25 holes with randomly positioned slots. Pegs that have a raised edge along one side must be rotated to fit the slot before they can be inserted. This test adds a dimension of complex coordination to a pegboard task. It is part of the Wisconsin Neuropsychological Test Battery. It is sensitive to general slowing due to medication effects, brain injury, or neurological disease such as Parkinsonism or cerebral HIV infection. Normative data exist for dominant and nondominant hand speed, and for ages 5 to above 60.

Grooved Pegboard Test scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation.

Mr. Z. produced the following profile:

### Grooved Pegboard Test

	Raw Score	T-Score	Classification	Percentile
Dominant left hand	78.2	40	Below average	16
Nondominant right hand	85.8	38	Mildly impaired	13

#### 2. Grip Strength Test

This technique assesses differences in hand strength on the assumption that lateralized brain damage affects the strength of the contralateral hand. Two trials for each hand are given, alternating between hands. The score is the force exerted in kilograms for each hand, averaged across the two trials.

Grip Strength Test scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation.

Mr. Z. produced the following average scores:

### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant left-hand strength	49.5	46	Average	37
Nondominant right-hand strength	44.0	46	Average	37

#### 3. Finger Tapping Test

This is one of the tests contained in the Halstead–Reitan Battery. It assesses finger-tapping speed. Each hand is given five 10-second trials with brief rest periods between trials. The score for each hand is the average for each set of five trials.

Finger Tapping Test average scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation.

Mr. Z. produced the following results:

### Finger Tapping Test

	Mean Raw Score	T-Score	Classification	Percentile
Dominant left hand	51.4	47	Average	39
Nondominant right hand	40.0	38	Mildly impaired	13

## EXECUTIVE FUNCTIONS

### 1. *Wisconsin Card Sorting Test*

The Wisconsin Card Sorting Test (WCST) is a recent standardization of Berg's 1948 measure. It provides a measure of the test taker's ability to form, maintain, and shift cognitive sets, and to utilize feedback in modifying responses. Standard scores are determined with a mean of 100 and a standard deviation of 15. T-scores may also be derived, with a mean of 50 and a standard deviation of 10.

Mr. Z. produced the following scores, corrected for age and education:

### Wisconsin Card Sorting Test

	Raw Score	Standard Score	T-Scores	Percentile	Classification
Total errors	55	77	35	6	Mildly impaired
Completed categories	3				
Perseverative responses	24	87	41	19	Below average

### 2. *Trail-Making Tests A and B*

The Trail-Making Test consists of two parts, Trail-Making Test Part A (Trails A) and Trail-Making Test Part B (Trails B). Trail-Making Test Part A requires visuo-optical scanning ability, immediate number identification ability, basic motor skills of the dominant hand, and the ability to maintain concentration under time constraints. Raw scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation.

Mr. Z. produced the following profile:

### Trail-Making A

Raw Score	T-Score	Classification	Percentile
56.4	32	Mildly to moderately impaired	4

Trail-Making Test Part B (Trails B) requires all the skills of Trails A, but it also demands immediate letter identification ability and the mental flexibility to switch sets under time constraints. Raw scores are converted to T-scores with a mean of 50 and a standard deviation of 10.

Mr. Z. produced the following profile:

### Trail-Making B

Raw Score	T-Score	Classification	Percentile
169.4	33	Mildly to moderately impaired	5

## TEST INTELLIGENCE

### 1. *Wechsler Adult Intelligence Scale-III*

“Intelligence is what we use when we don’t know what to do” (Piaget, 1929). The Wechsler Adult Intelligence Scale-III (WAIS-III) is the 1997 revision of the Wechsler Adult Intelligence Scale-Revised (WAIS-R). Like the WAIS-R, the WAIS-III is an objective estimate of adult intelligence. The WAIS-III is comprised of 14 different subtests, yielding a verbal intellectual quotient (VIQ), a performance intellectual quotient (PIQ), and a full-scale IQ (FSIQ). The FSIQ is a combination of the VIQ and PIQ. In addition, four index scores are computed, measuring the following areas: verbal comprehension (VC), perceptual organization (PO), working memory (WM), and processing speed (PS). The three IQ scores and the four index scores are standardized to have a mean of 100 and a standard deviation of 15.

Mr. Z. produced the following profile:

### Wechsler Adult Intelligence Scale-III

Subtest	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Picture Completion		8		8		
Vocabulary	6		6			
Digit Symbol-Coding		4				4
Similarities	5		5			
Block Design		4		4		
Arithmetic	5				5	
Matrix Reasoning		8		8		
Digit Span	7				7	
Information	5		5			
Picture Arrangement		6				
Comprehension	6					
Symbol Search		(7)				7
Letter-Number Sequencing	(8)				8	
<b>Sum of Scaled Scores</b>	<b>34</b>	<b>30</b>	<b>16</b>	<b>20</b>	<b>20</b>	<b>11</b>

Mr. Z.’s IQ scores are the following:

	Standard Score	Classification	Percentile	Range
Verbal IQ	74	Borderline	4	70–80
Performance IQ	75	Borderline	5	70–83
Full-Scale IQ	72	Borderline	3	68–77

Mr. Z.’s index scores are the following:

	Standard Score	Classification	Percentile	Range
Verbal comprehension	74	Borderline	4	69–81
Perceptual organization	80	Low average	9	74–89
Working memory	80	Low average	9	74–88
Processing speed	76	Borderline	5	70–88

### ASSESSMENT OF EMOTIONAL ADJUSTMENT

The MMPI-2 was administered to provide hypotheses regarding the psychological functioning of Mr. Z. The validity of this test with individuals who have experienced a traumatic brain injury has not been verified. The standard interpretations of clinical scales may not apply to individuals with suspected brain injury. The interpretations presented in this report need to be verified by other sources of clinical information.

### Minnesota Multiphasic Personality Inventory-2

The Minnesota Multiphasic Personality Inventory-2 (MMPI-2) is the 1989 revision of the original 1943 MMPI. The MMPI is an objective personality test composed of 567 statements to which the test taker responds true or false. It is a widely used, well-accepted measure of personality, providing information about a variety of psychiatric symptoms and problems. Four previous validity scales (L, F, K, and Cannot Say) along with five new scales (Fb, TRIN, VRIN, Fp, and S) provide an assessment of response biases and test validity. Ten basic clinical scales, plus the supplementary and content scales, may be scored. Raw scores are converted to uniform T-scores to allow easy comparison. Normal T-scores are generally within the range of 50 to 64.

Profile data for Mr. Z. are as follows:

Minnesota Multiphasic Personality Inventory-2										
Scale	VRIN	TRIN	F	Fb	Fp	L	K	S		
T-score	54	72	70	71	48	48	39	45		
Scale	1	2	3	4	5	6	7	8	9	0
T-score	68	72	61	77	30	72	64	81	53	58

### RECORDS REVIEWED

1. Records of Somewhere Methodist Hospital
2. Records of University Hospital
3. Records of Saint Benedict's Hospital
4. Kentucky State Police Toxicology Analysis Report
5. Winek's Drug and Chemical Blood Level Data 2000
6. Follow-up medical reports: A. Mazurek, M.D., and M. Jason Creech, M.D.

### NEUROBEHAVIORAL ANALYSIS

*Neurobehavioral analysis* is a term for the analytical evaluation of the neuropsychiatric examination data producing a comprehensive statement about the person's brain-behavior capacity. It is based upon historical, neurological, psychiatric, brain imaging, laboratory, prior records, and neuropsychological data.

Our neuropsychological assessment found that Mr. Z. is exhibiting impairment currently in the areas of verbal comprehension, processing speed, naming ability, verbal fluency, immediate memory, and executive functioning. He also exhibits impairment of manual dexterity and complex coordination. There is evidence to suggest that his intellectual capacity may have declined from the previous level.

The MRI examination reveals a significant area of loss of brain tissue over the anterior left frontal lobe. There also is evidence of brain atrophy and probable evidence of brain laceration. When Dr. Radiology reviewed the MRI, he felt that not only was there encephalomalacia over the left frontal cortical surface and the posterior frontal anterior parietal surface on the left, but that some of this represented a laceration of the brain, which could have occurred during the trauma as the bone elevation was to the right skull.

Mr. Z.'s predicted full-scale IQ is 86. His obtained full-scale IQ is 72. His cognitive effort during testing was optimal. This finding is consistent with dementia. He displays multiple neuropsychological deficits below expected levels based upon estimates of preinjury function. These findings are consistent with a cognitive disorder. He presently is capable of some, but not all, useful cognitive function. This finding is consistent with a moderate cognitive impairment.

## DIAGNOSES (DSM-IV-TR)

### Axis I:

A. Cognitive disorder due to traumatic brain injury and subsequent neurosurgical procedure as a result of brain trauma on March 2, 2001

B. Dementia due to traumatic brain injury on March 2, 2001

Axis II: Personality change due to traumatic brain injury on March 2, 2001

Axis III: Status-post subarachnoid and subdural hemorrhage, pneumocephalus, and right temporal bone open elevation

Axis IV: No evidence that environmental factors in his life account for his current mental state

Axis V: Current GAF = 55

## CONCLUSIONS

1. In my opinion, within reasonable medical probability, A.Z. has a 25% neuropsychiatric impairment due to traumatic brain injury. This is based on *Guides to the Evaluation of Permanent Impairment*, (American Medical Association, Chicago, 2000), page 320, Tables 13-5 and 13-6 (CDR score = 1.0).
2. In my opinion, within reasonable medical probability, A.Z. presently lacks the mental capacity to engage in any work he is trained, educated, or experienced to perform.

Respectfully submitted,  
Forensic Doctor, M.D.

## REPORT 2: CLOSED-HEAD INJURY IN A TEENAGER

This report demonstrates the reporting of information regarding a teenager who sustained a moderate closed-head injury. It represents a complicated issue of a youngster who may have had poor academic motivation or a learning disorder prior to head injury and how that is addressed within the body of a forensic neuropsychiatric report. It also represents an issue that may be presented to the neuropsychiatric examiner by the lawyer who hires the physician. Lawyers often will pose specific questions to the forensic examiner. Furthermore, it demonstrates the issue of organic brain impairment producing cognitive injury and psychiatric injury. Thus, the *Guides to the Evaluation of Permanent Impairment*, fifth edition, are used to report both impairment of a cognitive nature and impairment of a more psychiatric nature. This report utilizes both Chapters 13 and 14 of the *Guides*. No facts have been changed. However, specific locations and identities are altered.

September 17, 2002

Mr. Plaintiff Attorney  
Delta, Echo and Hotel, L.L.C.  
Someplace, Indiana  
RE: *J.S. v. Joan Defendant*

Dear Mr. Attorney:

I examined J.S. at my offices on August 13, 2002. My complete examination and testing required 9 hours. Two hours were devoted further to reviewing medical records, reviewing brain scans and laboratory reports, reviewing my test data, and preparing this report. This was a neuropsychiatric examination of five components. The first component consisted of taking J.S.'s history of the accident and how it has affected her mentally. Further history was obtained regarding J.S.'s past medical history, family history, and social history.

The second component consisted of a two-part mental examination. The first part consisted of a face-to-face qualitative mental status examination, and the second part consisted of the administration and

interpretation of standardized mental test instruments. The third component was a neurological examination, while the fourth component consisted of laboratory studies and positron emission tomography (PET) brain imaging. The fifth component consisted of reviewing available medical and other records, analyzing my data, performing a neurobehavioral analysis, and reducing my findings to this report.

### **IDENTIFICATION DATA**

J.S. is a 16-year-old female from Nowhere, Indiana. She is being examined at the request of her attorney to determine if she retains neuropsychiatric impairment as a result of a motor vehicle accident.

### **HISTORY FROM THE RECORDS**

On September 26, 2001, J.S. was an unrestrained front-seat passenger in a vehicle driven by her brother. Her vehicle sustained a head-on collision with an 18-wheeled tractor trailer truck. J.S. was attended by Emergency Medical Services at the accident site. She had a Glasgow Coma Scale obtained on seven different occasions between 1515 and 1630. On each occasion, the Glasgow Coma Scale score was 12, with E = 3, V = 4, and M = 5. At the accident scene, she complained that her head hurt. She was trapped and required physical extrication from the vehicle. Major front-end damage to her vehicle was noted by the emergency squad. She was in and out of consciousness to verbal stimuli. She was transported to the Regional Medical Center in Nowhere, Indiana. She was diagnosed with a closed-head injury. She was then transferred to Our Lady of Mercy Hospital in Kirbyville, Indiana, because of a left parietal cerebral hemorrhage detected by computerized tomography (CT). On receipt at this hospital, she was noted to be alert and oriented to person, place, and time, but she was amnesic to the accident events and factual details regarding the accident. She was placed in the intensive care unit and evaluated by the neurosurgical department. She was subsequently transferred to the pediatrics division where she was later discharged after three hospital days. She was given a follow-up appointment with Dr. Gomez.

Dr. Gomez evaluated her on October 9, 2001, and reviewed with her a small punctuate left parietal contusion of her brain. He noted that she was sleepy during the day and had to take a nap almost on a daily basis. He found her to be oriented and that she'd had no seizures. He allowed her to return to school. On December 3, 2001, he noted that she would awaken after 12 hours and still be tired. She had frequent mood swings and occasional headaches. Dr. Gomez found these symptoms consistent with postconcussion syndrome. No cognitive testing was undertaken.

### **HISTORY FROM THE EXAMINEE**

J.S. and her mother completed a 22-page medical questionnaire. I verified this document with her face-to-face. She was given written warning that her information might not be confidential. Warning also was given that we would not have a doctor-patient relationship and I would provide no treatment or counseling.

J.S. tells me that her last memory prior to the accident was sitting in a car talking to an ex-boyfriend at approximately 2:45 P.M. She remembers her brother's car leaving the school parking lot and turning left. She has no memory thereafter. She does not remember the impact. She has no memory of being in the ambulance or how she was extricated from the vehicle. She has no memory of her hospitalization. She does recall attending a pageant after her discharge from the hospital, but she has a poor memory for this event as well.

She reports substantial mood changes following the trauma. Her emotions are very labile and she will cry for no reason. She complains of poor concentration, loss of memory, word-finding difficulty, excessive arguing, crying for no reason, and difficulty with thinking. She denies any plans to harm herself or harm anyone else. Her mother confirms these symptoms and reports that J.S. will cry easily and anger very quickly. Her mother is concerned about absence spells where she will be unresponsive for 5 to 10 seconds. Her mother believes these spells are becoming greater in frequency. Moreover, J.S.

has poor sleep and cannot maintain sleep. She tosses and turns throughout the night and feels tired in the morning.

### **ACTIVITIES OF DAILY LIVING**

She lives at home and is presently a junior in high school. She arises about 6:00 A.M. and retires about 11:00 P.M. She is able to drive a vehicle and fix her own breakfast. Her hobby is dancing. She's able to read magazines and short stories. She occasionally rents movies to watch in the home and attends movies outside the home. She attends high school ball games. She doesn't hunt or fish. She has friends or family visitors over daily and uses the telephone quite frequently. She can dress and bathe herself independently.

### **PAST MEDICAL HISTORY**

She had severe iron deficiency anemia as an infant and required a transfusion at 18 months. She also developed exercise-induced asthma, which has been present since she was a youngster. She weighed about 6 pounds, 8 ounces at birth and was not born prematurely. She had no growth problems or brain injury at birth. She's had some difficulty sitting still in school and keeping her mind on tasks prior to this injury, and that has dramatically worsened since the brain injury. She had no difficulty learning to read, but teachers complained that she was too active.

She's never been injured in other motor vehicle accidents. She's never been in a coma. She has fractured her right arm previously. She underwent a tonsillectomy at age 10 because of frequent streptococcus infections. She occasionally uses an albuterol inhaler for exercise-induced asthma, and she requires that about once or twice every 2 weeks. She uses over-the-counter anti-inflammatory or pain medicines. She's using no herbs or natural products, and she has no known allergies. She smoked cigarettes a bit last year but discontinued these 6 months ago. She occasionally will drink beer with friends. She denies abusing illicit substances. She drinks one cup of coffee daily and five soft drinks daily.

### **PAST PSYCHIATRIC HISTORY**

She's never been hospitalized for psychiatric, drug abuse, alcohol, or mental problems. She's never been prescribed any form of nerve medicine, antidepressant, or tranquilizer. She's never received any kind of counseling or psychotherapy. She's never intentionally overdosed herself on drugs or medicines, and she's never made an attempt to take her life. She's never intentionally cut, burned, or disfigured herself.

### **FAMILY HISTORY**

Her father is 38 years old and has hypertension. Her mother is 39 years old and in good health. There is no family history of mental illness or depression. There are no alcoholics or drug addicts in the family. There is no history of suicides, homicides, violence toward others, child abuse, or spouse abuse. No one in the family has had epilepsy, neurological diseases, or Alzheimer's disease.

### **SOCIAL HISTORY**

She was born in Bush Community Hospital, and she has one brother. She's the last child in the birth order. Her father is employed in construction, and her mother is a registered nurse. Both parents are present in the home, and her father does not abuse her mother. J.S. has never been sexually or physically abused. She has no history of violence toward others. There are no firearms in the home.

She has completed the 10th grade. Her grades are poor. At the time of this examination, her grade point average is 1.16 on a 4.0 scale. During freshman year, her grade point average was less than 1.0. She has completed 12½ credits. Teachers have noted that she is more easily distracted since her brain injury.

## **LEGAL HISTORY**

She has no juvenile legal history.

## **EMPLOYMENT/VOCATIONAL HISTORY**

She obtained employment at a restaurant near her home on November 1, 2001. This was shortly after her accident. She was unable to keep orders and couldn't remember prices. Her employer was going to dismiss her, so she resigned after 1 month of employment.

## **REVIEW OF SYSTEMS**

Her general review is negative. In her HEENT review, she has chronic headaches that worsen with lack of sleep and her concentration is poor. Her chest, cardiovascular, gastrointestinal, genitourinary, and gynecological reviews are negative except as noted previously. Her psychiatric and sleep reviews are noted before. In her neurological review, her primary complaint is headache. She denies poor balance, abnormal movements, poor coordination, or paralysis.

## **MENTAL STATUS EXAMINATION**

The mental status examination is a face-to-face examination between the psychiatric physician and the patient. The purpose of the examination is to determine the function of the elements of mental and brain activity. For instance, thought is examined to determine if the person can go from point A to point B logically in his thinking. Thought is also examined to determine the presence or absence of circumstantial thinking, loose associations, or other determinants of abnormal mental function. Content of the thinking is examined for delusional thoughts, morbid ideas, perceptual distortions, suicidal/homicidal ideas, or other signs of mental pathology. Language is examined for expressive and receptive function, repetition errors, and to determine if the person can take mental ideas and properly convert them to motor acts.

Orientation to person, place, and time is determined. Gross memory ability is determined. Evaluation of the mental stream of activity, mood, range of affect, and thought and motor speed is completed. The mental status examination is a qualitative examination, and quantified elements of the mental examination are determined by standardized mental assessment below.

J.S. is a pleasant, cooperative teenage girl who is oriented to person, place, and time. Her mother assisted her with completion of the 22-page medical questionnaire, but she was examined independent of her mother. She is a capable historian with the exception of the amnesia for the trauma itself.

Affect is constricted. She is excessively anxious. She denies suicidal ideas or plans. She does not smile. There is no evidence of delusions or hallucinations. She's logical and coherent in her thinking without evidence of loose associations or circumstantiality. She denies suicidal ideas or plans.

Articulatory agility is within normal limits. The melodic line and phrase length are both reduced. She has a reduced ability for narrative discourse. However, there are no paraphasias noted or word-finding difficulty.

## **NEUROLOGICAL EXAMINATION**

The neurological examination is a physical examination performed by the physician. The purpose of the examination is to measure gross neurological functioning of the input and output nerves of the brain, as well as cerebral hemisphere functioning.



J.S.'s weight is 116 pounds. Her blood pressure is 118/74 in the left arm. There are no bruits heard in the neck or head. The face reveals no signs of trauma. The face is symmetric. Hand dominance is right.

### *Cranial Nerves*

In the optic group, there are no evident visual field cuts. Ocular motility is full. The pupils are equal and reactive to light and accommodation. The fundoscopic examination is benign.

In the branchiomotor group, masseter and temporalis muscles are intact. The forehead rises equally, the eyelids close tightly, and the lips purse. The gag reflex is brisk. *E* is well phonated. There is no nasal speech present. The SCM and trapezius muscles are strong. The tongue protrudes midline and is without atrophy or fasciculations.

In the cranial sensory group, anise and peppermint oils are not appreciated. Taste was not tested. Tuning fork sound is heard normally in the left ear but poorly in the right ear. The Weber sign lateralizes right.

In the somatic cranial sensory group, the corneal reflexes are brisk.  $V_1$  is normal in sensation,  $V_2$  is normal, and  $V_3$  is normal as well.

### *Somatic Motor*

Bulk and tone are good. Strength is symmetrical. Right grip strength is 37 kg. Left grip strength is 32 kg. Outstretched arms reveal no pronator drift.

Deep tendon reflexes are symmetric at the biceps, the brachioradialis, the triceps, the patellar tendons, and the ankle jerks.

### *Somatic Sensory*

Light touch and pinprick are perceived normally. Vibratory and position sensation are perceived normally. The Romberg position reveals no sway or loss of position.

### *Cerebellar*

Nystagmus is not present horizontally or vertically. Finger–nose function is performed smoothly. Heel–shin function is performed smoothly. Dysdiadochokinesia is not present.

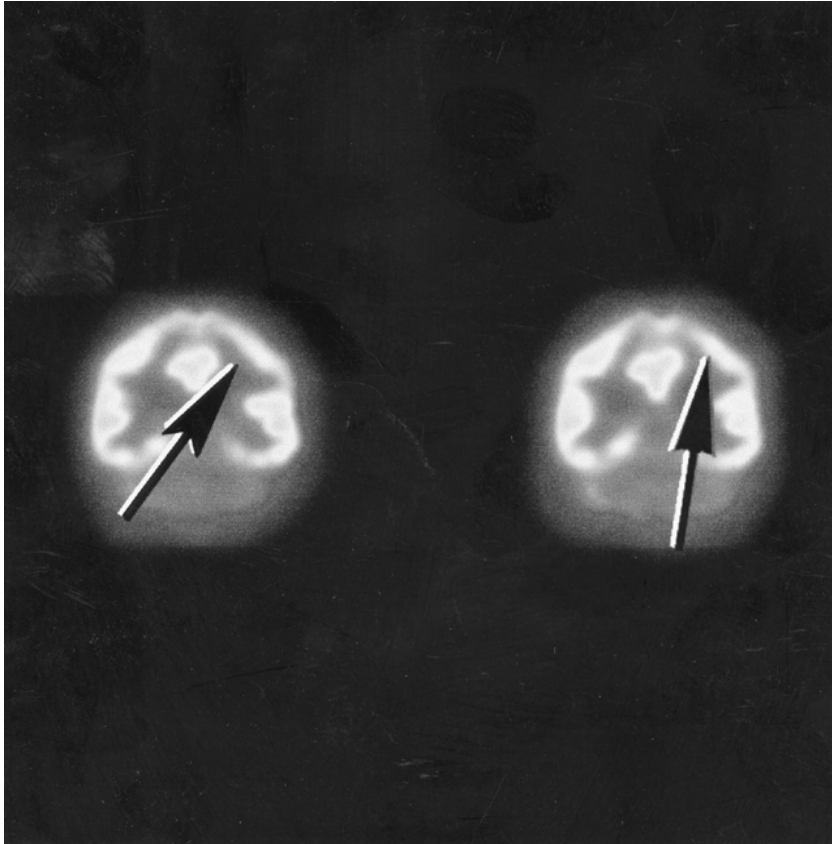
### *Gait and Station*

Gait analysis reveals normal stride. Heel rising is performed strongly. Toe rising is performed strongly. Squatting and rising are performed well. Arm swing is normal in arc and speed. Motor speed is normal. There is no dystaxia present on turns. Heel–toe walking is nondystaxic.

## **POSITRON EMISSION TOMOGRAPHY**

PET utilizes F-18-deoxyglucose (FDG) as a radioactive tracer. The brain treats this compound as if it were serum glucose. Therefore, the patient fasts for at least 4 hours prior to the administration of the FDG, as FDG competes with serum glucose for brain accumulation.

FDG-PET scans produce brain images of metabolic activity in brain tissue. Thus, this is a functional, rather than a structural, scan of the brain. Static images are obtained that are converted to single-slice displays in the transverse, sagittal, and coronal views. The tomograms are attenuation corrected.



**FIGURE 12.2** PET scan of brain obtained 11 months following a closed-head injury causing left parietal contusion. Note the relative hypometabolism of the left parietal cortex when contrasted to the right parietal cortex.

A PET scan was obtained at Jones Metabolic Imaging on August 13, 2002. J.S. received 8.9 millicuries of 18-FDG, administered intravenously with a concurrent blood glucose of 80 mg/dl. Brain imaging was performed 45 minutes after tracer injection, and the images were reconstructed in three planes.

There is minimal hypometabolism noted in the left frontal cortex and the left temporal pole. The left parietal cortex is also hypometabolic relative to the right parietal cortex, as is the head of the right caudate nucleus when compared with the corresponding contralateral structure. [Figure 12.2 demonstrates the left parietal cortex PET findings. This figure was not displayed in the original report but was presented at a deposition taken in the matter.]

#### **LABORATORY STUDIES**

A blood sample was obtained and sent to Medical Diagnostics Laboratory. J.S.'s blood was genotyped for apolipoprotein E. She has one copy of epsilon 3 and one copy of epsilon 4.

#### **STANDARDIZED MENTAL ASSESSMENT**

Forensic Doctor, M.D., requested formal neuropsychological testing. These tests were performed by Martha Psychologist, M.S. Alice Psychologist, Ph.D., supervised the administration, scoring, summarization, and analysis of the test data. These persons are agents or employees of Forensic Doctor, M.D., P.S.C. Medical conclusions about these data were made by Forensic Doctor, M.D.

*Standardized* means that the administered test instruments had exact rules for test administration and exact rules for test scoring. These rules have been standardized and published and are expected to be utilized by any person performing or interpreting the following test instruments.

The following test instruments were administered:

1. Test of Memory Malinger
2. Wide Range Achievement Test-III
3. Ruff 2 and 7 Selective Attention Test
4. Conners' Continuous Performance Test-II
5. Controlled Oral Word Association Test
6. Judgment of Line Orientation
7. Wechsler Memory Scale-III
8. Grooved Pegboard Test
9. Grip Strength Test
10. Finger Tapping Test
11. Wisconsin Card Sorting Test
12. Wechsler Adult Intelligence Scale-III
13. Minnesota Multiphasic Personality Inventory-2
14. Wechsler Test of Adult Reading

#### **BEHAVIORAL OBSERVATIONS DURING PSYCHOLOGICAL TESTING**

J.S. is a 16-year-old girl who is currently enrolled in her junior year of high school. On September 26, 2001, J.S. was involved in an automobile accident. J.S.'s mother reports that she currently experiences difficulty with mood swings, emotional instability, headaches, and fatigue.

During this assessment, J.S. was pleasant and cooperative. She appeared to apply her best effort to all tasks presented. J.S. exhibited a neutral facial expression during much of the assessment, but occasionally she would smile and laugh. She appeared to be mildly anxious regarding the assessment, but was not overly anxious.

J.S. rarely initiated conversation or volunteered personal information. During the assessment, she tended to use few words in her responses to test questions. Otherwise, no impairment of expressive or receptive language ability was noted. J.S. readily comprehended all test instructions.

She approached tasks in a rapid, and often impulsive, manner. She frequently would initiate her responses before instructions had been completed. J.S. also would often begin to respond to a test question and then subsequently change her response.

Late in the morning, J.S. complained of a headache. After lunch, however, she stated she had slept during the PET scan and was feeling better. Because of her rapid approach to the tests, J.S. completed all tests needed within 9 hours and did not return for the second day of the evaluation.

#### **FORENSIC DISTORTION ANALYSIS**

With any standardized mental assessment the recruitment of optimal effort is imperative. The instruments used to assess brain functioning cannot be "faked good." It is impossible for a brain to perform better than its capacity. However, it is possible for individuals to attempt to present themselves in a negative manner, to "fake bad" or present a "worst-foot forward." The extreme example of this presentation results in a response pattern referred to as malingering.

The following instruments were administered to determine the level of effort and validity of responses.

A. Measures of Cognitive Distortion

*Test of Memory Malingering*

The Test of Memory Malingering (TOMM) is a 50-item test for adults published in 1996 that is designed to discriminate between malingered and authentic memory impairments. Although the TOMM is sensitive to malingering, it is insensitive to neurological impairment. The TOMM has been extensively validated with groups of cognitively intact adults and with groups of adults diagnosed with cognitive impairment, aphasia, or traumatic brain injury. The TOMM consists of two learning trials and a retention trial. A low score on the TOMM suggests that memory impairment symptoms are false or exaggerated. This individual produced a score of 50 on Trial 2. This pattern of performance is within normal limits.

B. Measures of Psychological Distortion

*MMPI-A Validity Indices*

Minnesota Multiphasic Personality Inventory-Adolescent (MMPI-A) validity indices are derived from the MMPI-A responses. The MMPI-A is the 1992 adolescent revision of the original 1943 MMPI. In addition to the four previous validity scales — Cannot Say, L, K, and F scales — four additional scales are provided: F1, F2, TRIN, and VRIN.

When the profile is valid, MMPI-A validity indices usually fall into the T-score range of 50 to 65.

This individual produced the following profile:

Scale	Cannot Say	L Scale	F Scale	K Scale	F-K Index	F1	F2	VRIN	TRIN
Raw	0	1	5	9	-4	3	2	3	9
T-score		43	46	44		50	44	47	53

The above MMPI-A validity scores are consistent with a valid psychological profile.

**MEASURES PROVIDING ESTIMATES OF PREINJURY FUNCTION**

1. *Wechsler Test of Adult Reading*

The Wechsler Test of Adult Reading (WTAR) is a reading recognition test developed in 2001 to provide an assessment tool for estimating premorbid intellectual ability of adults ages 16 to 89. Reading recognition ability is relatively stable in the presence of cognitive declines associated with normal aging or brain insult. The WTAR was developed in conjunction with the WAIS-III and WMS-III. WAIS-III and WMS-III test scores are predicted from a combination of WTAR performance and demographic variables.

The WTAR has been validated for use with individuals diagnosed with disorders associated with cognitive decline, such as Alzheimer’s dementia, Parkinson’s disease, Huntington’s chorea, and Korsakoff’s syndrome. In addition, the WTAR has been validated with individuals who have been diagnosed with acute brain damage due to traumatic brain injury. The WTAR has also been validated with individuals diagnosed with neuropsychiatric and developmental disorders. J.S. produced the following WTAR scores:

**Wechsler Test of Adult Reading**

Raw Score	Standard Score
16	74

**WTAR Demographic-Predicted WAIS-III and WMS-III Indices**

	Standard Score	Percentile	Classification
WAIS-III VIQ	83	13	Low average
WAIS-III PIQ	89	23	Low average
WAIS-III FSIQ	83	13	Low average

## WTAR Demographic-Predicted WAIS-III and WMS-III Indices

	Standard Score	Percentile	Classification
WAIS-III VCI	82	12	Low average
WAIS-III POI	91	27	Average
WAIS-III WMI	85	16	Low average
WAIS-III PSI	92	30	Average
WMS-III Immediate Memory Index	89	23	Low average
WMS-III General Memory Index	89	23	Low average
WMS-III Working Memory Index	100	50	Average

### 2. *Wide Range Achievement Test-III, Reading subtest*

The Wide Range Achievement Test-Revised (WRAT-III) is a screening measure for academic achievement. The WRAT-III assesses reading recognition, spelling, and arithmetic skills. Raw scores, grade equivalence, and percentiles are assigned. Standard scores are also obtained, in which the mean (average) is represented by a standard score of 100 and the standard deviation is 15. Standard scores are used for interpretation. Reading scores tend to resist dementing processes. J.S. produced the following raw scores and derived scores:

#### Wide Range Achievement Test-III

	Raw Score	Standard Score	Percentile	Grade Equivalence	Classification
Reading	40	90	25	8	Average
Spelling	36	92	30	7	Average
Arithmetic	38	93	32	7	Average

### 3. *Vocabulary subtest of the WAIS-III*

This subtest tends to resist dementing brain disorders. J.S. produced the following scaled scores on the Vocabulary subtest of the WAIS-III:

#### WAIS-III Subtest

	Scaled Score	Standard Score	Percentile	Classification
Vocabulary	5	75	5	Borderline

## ATTENTION AND CONCENTRATION

### 1. *Ruff 2 and 7 Selective Attention Test*

The Ruff 2 and 7 Selective Attention Test measures two overlapping aspects of visual attention:

- A. Sustained attention
- B. Selective attention

Sustained attention refers to the ability to maintain a consistent level of visual performance over an extended period. Selective attention refers to the ability to select relevant visual stimuli (targets) while ignoring salient but irrelevant stimuli (distracters).

J.S.'s scores on the 2 and 7 Selective Attention Test are:

#### Ruff 2 and 7 Selective Attention Test

	Measure	Raw Score	T-Score	Percentile	Classification
Letters	Automatic detection speed	164	54	66	Average
	Automatic detection errors	27			
	Automatic detection accuracy	85.86	23	<1	Moderately to severely impaired

## Ruff 2 and 7 Selective Attention Test (*Continued*)

	Measure	Raw Score	T-Score	Percentile	Classification
Digits	Controlled search speed	155	58	79	Above average
	Controlled search errors	32			
	Controlled search accuracy	82.89	28	1	Moderately impaired

### Total Scores

Measure	Sum of T-Scores	T-Score	Percentile	Classification
Total speed	112	58	79	Above average
Total accuracy	51	25	1	Moderately impaired

### 2. *Conners' Continuous Performance Test-II*

The Conners' Continuous Performance Test-II (CPT-II) is the most recent (2000) version of the original CPT, which was developed in 1957. The computerized version of the CPT was first developed in the 1970s and has since been normed on over 2600 people, ranging from 6 to 70 years of age. The CPT provides a useful measure of attention and learning abilities and allows for comparison of responses to those of the general population, attention deficit hyperactivity disorder subjects, and neurologically impaired groups. Results are presented in the form of T-scores and percentiles. This individual produced the following profile:

### Conners' Continuous Performance Test-II

Measure	T-Score	Percentile	Guideline
# Omissions	44	71	Average
# Commissions	44	72	Average
Hit RT	41	80	Average
Hit RT standard error	48	58	Average
Variability	45	69	Average
Detectability (d')	41	82	Average
Response style (B)	43	75	Average
Perseverations	46	65	Average
Hit RT block change	47	58	Average
Hit SE block change	47	62	Average
Hit RT ISI change	50	47	Average
Hit SE ISI change	51	45	Average

*Note:* RT = reaction time; SE = standard error; ISI = inter-stimulus interval.

Please note that in order to be consistent with other test results, CPT-II percentile scores have been inverted from the values presented in the CPT-II computer-based score report. Thus, lower percentile scores reflect lower levels of performance in the table above. T-scores have not been inverted.

### 3. *Digit Span subtest of the Wechsler Adult Intelligence Scale-III*

The Digit Span subtest in the Wechsler Adult Intelligence Scales measures span of immediate verbal recall. It is comprised of two different tests, digits forward and digits backward. These involve different mental activities and are affected differently by brain damage. Both measure short-term storage capacity for auditory stimuli and require auditory attention as well as intact short-term auditory retention capacity.

Digits forward primarily measures the efficiency of auditory attention. Digits backward calls upon working memory and involves mental double-tracking in that both memory and the reversing operations must proceed simultaneously.

J.S. produced the following results on the Digit Span subtest:

## WAIS-III Digit Span Subtest

	Raw Score	Standard Score	Percentile	Classification
Longest digit span forward	7	103	58	Average
Longest digit span backward	5	101	53	Average
Digit span scaled score	9	95	37	Average

## LANGUAGE AND LANGUAGE-RELATED SKILLS

### *Controlled Oral Word Association Test*

In the Controlled Oral Word Association Test (COWA), the test taker is asked to produce as many words as possible beginning with a given letter in a specified period. This test measures word production. *F*, *A*, and *S* are the most commonly used letters for this test. The score is the sum of all admissible words for the three letters. This score is adjusted for age, sex, and education. The adjusted scores are converted to T-scores and percentiles.

J.S. produced the following results:

### Controlled Oral Word Association Test

Raw score	34
T-score	44
Percentile	27
Classification	Normal

## VISUOSPATIAL AND CONSTRUCTIONAL SKILLS

### *Judgment of Line Orientation*

This test examines the ability to estimate angular relationships between line segments by visually matching angled line pairs to 11 numbered radii forming a semicircle. While performing this test, cerebral blood flow in temporal–occipital areas increases bilaterally, with the greatest increase in the right hemisphere. Women’s raw scores tend to run about 2 points below those of men.

J.S. produced the following mean corrected score:

### Judgment of Line Orientation

Raw score	13
Age-corrected raw score	15
Percentile	1
Classification	Severely impaired

## MEMORY

### *Wechsler Memory Scale-III*

The Wechsler Memory Scale-III (WMS-III) is the most recent revision of the Wechsler Memory Scale. There are 11 different subtests that are combined to form eight different memory indices. These indices have a mean of 100 and a standard score deviation of 15. This individual produced the following profile:

### Wechsler Memory Scale-III

	Scale Score Sum	Index Score	Percentile	Classification
Auditory immediate	16	89	23	Low average
Visual immediate	25	115	84	High average

## Wechsler Memory Scale-III (Continued)

	Scale Score	Sum	Index Score	Percentile	Classification
Immediate memory	41		102	55	Average
Auditory delayed	13		80	9	Low average
Visual delayed	25		115	84	High average
Auditory recognition delayed	4		70	2	Borderline
General memory	42		88	21	Low average
Working memory	22		105	63	Average

## MOTOR AND VISUAL MOTOR SKILLS

### 1. Grooved Pegboard Test

The Grooved Pegboard Test is a manipulative dexterity test. It consists of 25 holes with randomly positioned slots. Pegs that have a raised edge along one side must be rotated to fit the slot before they can be inserted. This test adds a dimension of complex coordination to a pegboard task. It is part of the Wisconsin Neuropsychological Test Battery. It is sensitive to general slowing due to medication effects, brain injury, or neurological disease such as Parkinsonism or cerebral HIV infection. Normative data exist for dominant and nondominant hand speed, and for ages 5 to above 60.

Grooved Pegboard Test scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation. J.S. produced the following profile:

### Grooved Pegboard Test

	Raw Score	T-Score	Classification	Percentile
Dominant right hand	74	37	Mildly impaired	10
Nondominant left hand	80	37	Mildly impaired	10

### 2. Grip Strength Test

This technique assesses differences in hand strength, on the assumption that lateralized brain damage affects the strength of the contralateral hand. Two trials for each hand are given, alternating between hands. The score is the force exerted in kilograms for each hand, averaged across the two trials.

Grip Strength Test scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation. J.S. produced the following average scores:

### Grip Strength Test

	Kilograms	T-Score	Classification	Percentile
Dominant right-hand strength	34	57	Above average	77
Nondominant left-hand strength	29	51	Average	55

### 3. Finger Tapping Test

This is one of the tests contained in the Halstead-Reitan Battery. It assesses finger-tapping speed. Each hand is given five 10-second trials with brief rest periods between trials. The score for each hand is the average for each set of five trials.

Finger Tapping Test average scores are converted to T-scores in which 50 is the mean and 10 is the standard deviation. J.S. produced the following results:



## Finger Tapping Test

	Mean Raw Score	T-Score	Classification	Percentile
Dominant right hand	49	58	Above average	81
Nondominant left hand	48	61	Above average	87

## EXECUTIVE FUNCTIONS

### *Wisconsin Card Sorting Test*

The Wisconsin Card Sorting Test (WCST) is a recent standardization of Berg's 1948 measure. It provides a measure of the test taker's ability to form, maintain, and shift cognitive sets, and to utilize feedback in modifying responses. Standard scores are determined with a mean score of 100 and a standard deviation of 15. T-scores may also be derived, with a mean of 50 and a standard deviation of 10. This test taker produced the following scores, corrected for age and education:

### Wisconsin Card Sorting Test

	Raw Score	Standard Score	T-Score	Percentile	Classification
Total errors	29	97	48	42	Average
Completed categories	6				
Perseverative responses	11	105	53	63	Average

## TEST INTELLIGENCE

### *Wechsler Adult Intelligence Scale-III*

"Intelligence is what we use when we don't know what to do" (Piaget, 1929). The Wechsler Adult Intelligence Scale-III (WAIS-III) is the 1997 revision of the Wechsler Adult Intelligence Scale-Revised (WAIS-R). Like the WAIS-R, the WAIS-III is an objective estimate of adult intelligence. The WAIS-III is comprised of 14 different subtests, yielding a verbal intellectual quotient (VIQ), a performance intellectual quotient (PIQ), and a full-scale IQ (FSIQ). The FSIQ is a combination of the VIQ and PIQ. In addition, four index scores are computed, measuring the following areas: verbal comprehension (VC), perceptual organization (PO), working memory (WM), and processing speed (PS). The three IQ scores and the four index scores are standardized to have a mean of 100 and a standard deviation of 15.

J.S. produced the following profile:

### Wechsler Adult Intelligence Scale-III

Subtest	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Picture Completion		7		7		
Vocabulary	5		5			
Digit Symbol-Coding		13				13
Similarities	9		9			
Block Design		8		8		
Arithmetic	7				7	
Matrix Reasoning		12		12		
Digit Span	9				9	
Information	5		5			
Picture Arrangement		8				
Comprehension	4					

## Wechsler Adult Intelligence Scale-III (Continued)

Subtest	Age-Adjusted Scaled Scores					
	Verbal	Performance	VC	PO	WM	PS
Symbol Search		(12)				12
Letter-Number Sequencing	(11)				11	
<b>Sum of scaled scores</b>	<b>39</b>	<b>48</b>	<b>19</b>	<b>27</b>	<b>27</b>	<b>25</b>

J.S.'s IQ scores are the following:

	Standard Score	Classification	Percentile	Range
Verbal IQ	79	Borderline	8	75-85
Performance IQ	97	Average	42	90-104
Full-scale IQ	86	Low average	18	82-90

J.S.'s index scores are the following:

	Standard Score	Classification	Percentile	Range
Verbal comprehension	80	Low average	9	76-86
Perceptual organization	93	Average	32	86-101
Working memory	94	Average	34	88-101
Processing speed	114	High average	82	103-121

### PSYCHOPATHOLOGY

Measures of psychopathology were administered to provide hypotheses regarding the psychological functioning of this individual. The validity of this test with individuals who have experienced a traumatic brain injury has not been verified. The standard interpretations of clinical scales may not apply to individuals with suspected brain injury. The interpretations presented in this report need to be verified by other sources of clinical information.

#### *Minnesota Multiphasic Personality Inventory-Adolescent*

The Minnesota Multiphasic Personality Inventory-Adolescent (MMPI-A) is the 1992 revision of the original 1943 MMPI designed for use by children ages 14 through 18 years. The MMPI-A is an objective personality test composed of 478 statements to which the test taker responds true or false. It is a widely used, well-accepted measure of personality, providing information about a variety of psychiatric symptoms and problems. Four previously existing validity scales (L, F, K, and Cannot Say) along with four new validity scales (F1, F2, TRIN, and VRIN) provide an assessment of response biases and test validity. Ten basic clinical scales plus the supplementary and content scales may be scored. Raw scores are converted to uniform T-scores to allow easy comparison. Normal T-scores are generally in the range of 50 to 65. Profile data are as follows:

#### Minnesota Multiphasic Personality Inventory-A

Scale	L	F	K	1	2	3	4	5	
T-score	43	46	44	56	53	55	48	56	
Scale	6	7	8	9	0	F1	F2	TRIN	VRIN
T-score	47	58	48	68	38	50	44	53	47

## RECORDS REVIEWED

1. EMS ambulance run report — September 26, 2001
2. Records of Regional Medical Center, Nowhere, Indiana
3. Records of Our Lady of Mercy Hospital, Kirbyville, Indiana
4. Records of A. Gomez, M.D.
5. Transcripts, Johnson County High School

## NEUROBEHAVIORAL ANALYSIS

*Neurobehavioral analysis* is a term for the analytical evaluation of the neuropsychiatric examination data producing a comprehensive statement about the person's brain-behavior capacity. It is based upon historical, neurological, psychiatric, brain imaging, laboratory, prior records, and neuropsychological data.

The Glasgow Coma Scale (GCS) finding on September 26, 2001, is consistent with a moderate head injury. J.S. had a GCS score of 12 documented on seven different occasions. Moreover, since she had a CT finding of left parietal hemorrhage, by definition she has had at least a moderate brain injury. Following the accident, she had posttraumatic hypersomnia.

She demonstrates retrograde amnesia, amnesia for impact, and anterograde amnesia. These findings are consistent with significant brain trauma. PET scanning obtained at this examination reveals a very significant functional injury in the left cerebral hemisphere, as the level of metabolic abnormality far exceeds the original focus of hemorrhage. Moreover, the left parietal hypometabolism correlates with the acute CT imaging of a left parietal hemorrhage.

During measurement of neuropsychological domains, J.S. revealed impairment in bilateral visual motor coordination and the ability to estimate angular relationships. Delayed auditory recognition is within the borderline range of impairment. She has areas of significant weakness in verbal reasoning, verbal concept formation, verbal expression, fund of information, social judgment, and the ability to evaluate figural stimuli to distinguish essential details. On the other hand, she demonstrates neuropsychological strengths in the areas of immediate and delayed visual memory, mental processing speed, sequencing ability, and cognitive flexibility. On a measure of cognitive effort, she produced a 100% score. Her performance on the validity scales of the MMPI-A was within acceptable limits. The language-based deficits are consistent with the left cerebral hemisphere hypoperfusion on the PET scan; however, in light of her preinjury academic transcripts, there is probably evidence of reduced verbal skills prior to injury. However, preinjury learning disability predisposes her to a poorer outcome from brain trauma. Her MMPI-A results are consistent with an irritable and dysphoric mood and a significant denial of illness.

As a result of this brain injury, J.S. has an increased risk for developing Alzheimer's-like neurodegeneration when older. Persons who have one copy of epsilon 4 and sustain a traumatic brain injury have at least a twofold greater risk of developing Alzheimer's-like pathology than those without brain injuries.

## ANSWERS TO LAWYER'S QUESTIONS

**Question 1:** From your review of the records and the history you have taken, how would you describe the injury that was sustained by J.S.?

J.S. sustained a moderately severe traumatic brain injury on September 26, 2001. This is based on the Glasgow Coma Scale, left parietal hemorrhage, and postinjury alteration of mental function.

**Question 2:** Is J.S. still experiencing complications and problems as a result of the head injury she sustained in the wreck? If so, what?

The deficits noted in J.S. are described more fully in my preceding report.

**Question 3:** Do you think a head strike caused the closed-head injury, or do you feel it was based upon the rapid acceleration–deceleration of the brain within the skull that caused the injury, or both?

I am unable to determine, based on the presentation of her injury, whether her head struck an object or she sustained an acceleration–deceleration injury. Actually, the differential of this is irrelevant since we have the Glasgow Coma Scale and left parietal hemorrhage as markers of brain injury.

**Question 4:** Based upon everything you’ve reviewed and the testing you have performed, do you believe J.S. has sustained a permanent closed-head injury that will affect her in the future? If so, to what extent will she be limited by the closed-head injury and what evidence is there supporting those restrictions?

Please see my report for the evidence supporting her deficits. Those deficits will, in fact, reduce the effectiveness of her mental function during life. Her mood disorder should be immediately treated with antidepressants. She has an increased risk of late-appearing neurodegeneration.

### **DIAGNOSES (DSM-IV-TR)**

Axis I:

A. Cognitive disorder due to traumatic brain injury, September 26, 2001

B. Mood disorder due to traumatic brain injury, September 26, 2001

Axis II: No personality disorder identified

Axis III: Moderately severe traumatic brain injury (GCS = 12) with left parietal hemorrhage; one copy of apolipoprotein E4

Axis IV: No evidence that other external stressors account for the cognitive deficits found at the time of my examination

Axis V: Current GAF = 55 to 60

### **CONCLUSIONS**

1. In my opinion, within reasonable medical probability, J.S. has a 20% neuropsychiatric impairment from cognitive impairment due to traumatic brain injury. This is based on *Guides to the Evaluation of Permanent Impairment* (American Medical Association, Chicago, 2000), page 320, Tables 13-5 and 13-6 (CDR score = 1.0).
2. In my opinion, within reasonable medical probability, J.S. has a Class II psychiatric impairment due to traumatic brain injury-induced depression (mood disorder). This is based on *Guides to the Evaluation of Permanent Impairment* (American Medical Association, Chicago, 2000), page 363, Table 14-1.

Respectfully submitted,  
Forensic Doctor, M.D.

### **REPORT 3: MALINGERING APHASIA AND HEMIPARESIS**

This report can be difficult for a forensic examiner to write since it asserts that the examinee was faking his mental state. This is not a difficult report to write when the forensic examiner has been hired by the defendant, but it can be quite difficult to write if hired by the plaintiff lawyer, who sees his or her legal case evaporating as his client is called a malingerer. Thus, it is important if one suspects malingering to be extra careful in the documentation of historical and test data to support the issue. Juries are uncomfortable when a physician presents medical evidence in court and diagnoses an examinee as a malingerer unless there is substantial, believable evidence that can be understood by an ordinary person. During a preexamination review of records, inconsistencies were noted in the examinee’s histories. Thus, his mental and neurological examination and history were videotaped during this examination.

March 12, 2002

Mr. Defense Lawyer  
Able, Baker and Charlie, P.L.C.  
123 Main Street  
Anytown, Kentucky 40202  
RE: *S.T. v. Charles Defendant*

Dear Mr. Lawyer:

I examined S.T. at my offices on February 7 and 8, 2002. My complete examination and testing required 10½ hours. Eight and a half hours were required to abstract records, and two hours were devoted further to reviewing these medical records, reviewing brain scans, reviewing my test data, and preparing this report. This was a neuropsychiatric examination of four components. The first component consisted of taking his history of the accident and how it has affected him mentally. Further history was obtained regarding S.T.'s past medical history, family history, and social history.

The second component consisted of a two-part mental examination. The first part consisted of a face-to-face qualitative mental status examination, and the second part consisted of the administration and interpretation of standardized mental test instruments. The third component consisted of MR brain imaging. The fourth component consisted of reviewing available medical and other records, analyzing my data, performing a neurobehavioral analysis, and reducing my findings to a report.

#### **IDENTIFICATION DATA**

S.T. is a 46-year-old male from Anytown, Kentucky. He is being examined at the request of the defendant to determine if he retains neuropsychiatric impairment as a result of a motor vehicle accident.

#### **HISTORY**

S.T. completed a 22-page medical questionnaire. I verified this with him face-to-face. He was given written warning that his information might not be confidential. Warning was also given that we would not have a doctor-patient relationship and I would provide no treatment or counseling. His examination was video- and audiotaped.

S.T. is alleging that on December 21, 1999, an unknown and unwitnessed person ran him off the road, and he subsequently drove his truck over an embankment and struck his head. He claims severe brain injury with loss of ability to read or write and a memory impairment as a result. I reviewed the Uniform Police Traffic Accident Report, and Officer Amy Policeman wrote that on January 13, 2000, after S.T. was released from the hospital, she interviewed him. He stated to Officer Policeman "that there was another vehicle involved. The other unit was traveling west on 712 and came over to unit 1's lane, causing him to hit his brakes and slow down trying to get out of the other unit's way. The unknown unit still struck unit 1 on the left side of his vehicle. Unit 1 stated that all he remembered was that the unknown unit was gray in color and an older model vehicle. The officer could find no other evidence of another unit."

In an effort to check his historical accuracy, I had him describe for me the accident. He told me that he was traveling to meet a builder, as he had his own remodeling business at the time. Somebody entered his lane and struck his truck. He said the person was in a dark car. He recalls hearing people talk for a minute. This occurred in Anytown, and he claims to not know what he hit.

When asked if he'd been experiencing any mental or nervous problems in the last month prior to my examination, he reported "anxiety." When asked if he'd been experiencing any physical problems in the last month, he reported that he was recovering from knee surgery. With regard to his specific mental symptoms, he denies any plans to harm himself or harm anyone else, but he reports depression,

nervousness, loss of memory, confusion, inability to know the month or year, hearing voices, seeing things, irritability, crying for no reason, and trouble thinking. He says all of these symptoms are a result of the automobile accident.

Neurologically, he reports that he “blacks out a lot.” He reports poor balance, abnormal movements, and poor coordination, and he falls when he blacks out. In his musculoskeletal review, he’s had left knee surgery and he showed me the scars on that knee; they are quite extensive. He denies that this knee gives away with him. In his sleep review, he admits that he’s been diagnosed with obstructive sleep apnea, and he told me that he’d had these symptoms for quite some time. I advised him that one of the EEGs he recently had taken was positive for loss of breathing. He admits that others have observed him to stop breathing during sleep. He is supposed to use CPAP at night, but he claims to not know if he does use this or not.

After his accident, S.T. was admitted to the University of Anytown Hospital on December 22, 1999, and discharged on December 31, 1999. His admitting diagnosis was a mental status change, and his final diagnosis was “mental status change with postconcussion syndrome and resolving amnesia/aphasia syndrome.” However, a CT scan of his brain taken at Anytown Emergency Department was normal on the day of his injury, and an MRI/MRA done at the University of Anytown Hospital was also normal. A EEG was completed and was normal. He was reported to be somnolent and arouseable by verbal stimulation and was “unable to follow any instructions appropriately and he was aphasic.” Interestingly, on December 30, 1999, “the patient started spontaneously talking to the nurse and the residents and he stated that he wanted to go home and he also wanted psychiatry to evaluate his brain function and he said he didn’t think he had a neurological problem and basically, he had a psychiatric problem.” He had no observable neurological deficit. On December 31, 1999, the date of discharge, an EEG was obtained revealing normal background rhythms. However, the report states, “Note that the technologist observed respiratory features during sleep suggestive of obstructive sleep apnea.”

### **ACTIVITIES OF DAILY LIVING**

S.T. lives with his third wife, whom he married in 1998. He says she “has to take care” of him. He is not working currently, and he has not qualified for social security benefits, as he reports he’s receiving SSI benefits. His time of arising and going to bed is variable. His wife fixes breakfast. He says he’s not allowed to drive, but that is not listed as a restriction on his recently issued driver’s license or his physician records. His wife performs the household activities. He attends church once a month.

He denies having hobbies. He claims he can’t read or write. However, he watches television 10 hours daily. He does take out the garbage, but he denies renting movies or attending movies, and he denies attending ball games or hunting or fishing. He eats outside the home socially two or three times a month. He has friends or family visitors over four to eight times monthly. He says he “never” uses the telephone. He can dress himself “with help” and he can bathe himself “with help.” He claims to be entirely sexually dysfunctional, and his wife confirmed that on his questionnaire.

### **PAST MEDICAL HISTORY**

He reports a birth weight of 13 pounds, and he was born with spina bifida. He denies that he had any developmental difficulties. He says he was happy as a child, and he denies difficulty learning in school; he could keep his mind on tasks and had no problems learning to read. Teachers did not describe him as hyperactive.

He denies any other motor vehicle accidents causing injury. He’s never been in a coma. He did fracture his back when he fell from a ladder in 1998, and he underwent a lumbar discectomy and fusion thereafter. His surgeries include:

1. Lumbar surgery — age 41
2. Shoulder surgery — age 42
3. Left elbow surgery — age 44
4. Left knee surgery — age 46
5. Septoplasty and nose surgery — sometime in the past, but exact date unknown

His present medications include:

1. Lisinopril/HCTZ, 20/12.5 mg daily for hypertension
2. Atenolol, 50 mg daily for hypertension
3. Venlafaxine, 150 mg daily for depression
4. Quetiapine, 200 mg daily for depression
5. Hydrocodone, 10 mg as needed for pain
6. Trazodone, 100 mg at night for sleep
7. Vitamins

He denies using other over-the-counter medicines. He's allergic to codeine and IV contrast dye. He denies using tobacco products. He denies using alcohol. He claims that he used cocaine once when "some friend let me try it." He drinks five cups of coffee daily and two diet colas daily.

### **PAST PSYCHIATRIC HISTORY**

The first time he reports using psychiatric medicines is 1997. It apparently was before he fractured his back but after his back surgery; he also took either an antidepressant or anti-anxiety agent. The University of Anytown Hospital records indicate that he was taking venlafaxine when admitted to that hospital, which indicates that he was under treatment for depression prior to the alleged head injury.

In 1997 or 1998, he underwent counseling because of depression as a result of a back injury and was placed on venlafaxine at that time. He saw Jane Therapist for counseling. He denies he's ever overdosed himself on drugs or medicines or that he's ever made an attempt to take his life. He denies he's ever intentionally cut, burned, or disfigured himself. He denies setting fires or harming animals as a child.

### **FAMILY HISTORY**

His father died at age 50 and his mother died at age 52. He doesn't know the causes of death. A brother died at age 36 or 37. He denies any family history of mental illness or depression. He denies alcoholism or drug addiction in the family and denies any history of suicides, homicides, violence toward others, child abuse, or spouse abuse in the family. He further denies epilepsy, neurological diseases, Alzheimer's disease, or strokes in his family.

### **SOCIAL HISTORY**

He was born in Anytown, Kentucky, and he's the fourth child of five children. He had three sisters and one brother. He reportedly can't tell what his father did for a living, but apparently his mother was a homemaker. His father died in 1980 and his mother died in 1987. However, his parents divorced when he was 1 year old, and his mother subsequently raised him. He claims his father did not abuse his mother.

He denies he's ever been sexually or physically abused. He made good grades in high school. After he was released from prison, he attended Pleasantown State University for a while and eventually received an AA degree in business from St. John College. He completed a bachelor degree in psychology and sociology at Joy City College, and he told me that he "wanted to be a counselor" so he could prevent other youngsters from "ending up the way" he did.

He married his wife Joan in 1972 but divorced her in 1988. Two children, now 23 and 24 years of age, resulted from that marriage. He married Susanna in 1992 and divorced her in 1997, and he married Cleopatra in 1998. He describes his current marital relationship as good.

## **LEGAL HISTORY**

He admitted that he is a convicted felon. He claims to not remember when he was released from prison. Thus, I had to reconstruct his legal history from the documents supplied to me. The earliest legal record that summarizes his background is a U.S. District Court presentence report dated December 1, 1987. He was charged with aiding and abetting interstate transportation of a stolen boat and receiving stolen goods. It appears from reading the prosecution's version of the record that he stole many boats. His adult record indicates that at age 18, he was convicted of transportation of a stolen vehicle across state lines. He pled guilty to five counts of forgery. He pled guilty to escape. He pled guilty to second-degree forgery. He pled guilty to receiving stolen property. He pled guilty to felony theft and deceptive practices. He pled guilty to second-degree burglary and to being a persistent felony offender.

His personal data within this file indicate that at 5 years of age, his mother requested the Catholic charities to take care of her children. His father was in the state penitentiary at that time, and his mother was also facing criminal charges. He was placed in the St. Joseph Orphanage in Anytown, Kentucky, and remained there until August 1965. Throughout his adolescence, he was periodically recommitted to different institutions for various infractions of the law. At approximately 17 years of age, he went to live with his grandparents.

In contrast to what he told me about high school, school transcripts reviewed by the federal government indicate that S.T. withdrew from Excel Trade School in Anytown on October 20, 1969, while attending the tenth grade. While incarcerated in the Federal Bureau of Prisons, he obtained a GED at the Federal Youth Center in Hawaii in 1974.

Of direct relevance to this case is information that while within the Federal Bureau of Prisons, he was administered an intelligence test and scored a full-scale IQ of 110. The date and nature of the tests administered were not identified. The other records indicate that he enlisted in the U.S. Army and was medically discharged due to complications from spina bifida occulta.

A review of the FBI file on S.T. indicates that his criminal career did not end after December 1987. He was arrested subsequently for interstate transportation of stolen goods. He was sentenced to the Federal Correctional Institute for ten years. After later parole, he was rearrested and charged with possession of stolen property and again arrested and charged with possession of a handgun by a convicted felon. The observer of my files will note that he specifically denied that charge on my videotaped interview.

He was a party in a lawsuit when he allegedly fractured his back and shoulder while working for Superior Fence Company. He filed a workers' compensation claim in that alleged accident as well. He denies he's ever been a party in a restraining order or emergency protective order. He denies he's ever been charged with spouse abuse, child abuse, or terroristic threatening. He denies he's ever declared bankruptcy.

## **EMPLOYMENT/VOCATIONAL HISTORY**

S.T. worked a short time for Superior Fence Company as an installer, and then he owned and operated his own remodeling company. He currently receives SSI benefits, as he's never worked publicly long enough in his life to fully fund his Social Security Disability program.

## **MILITARY HISTORY**

He enlisted in the U.S. Army as noted previously and received a medical discharge in 1972.



## REVIEW OF SYSTEMS

In his general review, S.T. says he's lost 40 pounds, but today he reports a weight of 280 pounds. He has difficulty sleeping at night and is fatigued. In his HEENT review, he complains of headaches "at times." He also claims that he can't see out of his left eye and he's lost 40% of his hearing in his right ear and has ringing in his ears.

His chest and cardiovascular reviews are negative. In his gastrointestinal review, he complains of heartburn and indigestion. His genitourinary review is negative. His psychiatric, neurological, orthopedic, and sleep reviews are noted above.

## MENTAL STATUS EXAMINATION

S.T. appeared in my waiting room, and he was found sitting next to his wife with his head on her left shoulder, holding his small dog with both hands in his lap. I advised him and his wife that they would have to take the dog from the waiting room, as this was a medical facility and only seeing-eye dogs were allowed in our building. They complied with my request and took their dog and my questionnaire to their automobile, which was parked directly below my office window. I observed S.T. sitting in the passenger seat holding his dog and pointing to items on the information sheet as his wife completed it. After they completed the 22-page questionnaire, they returned to my office.

I took S.T. from the area where he was being psychologically tested to my library for the face-to-face examination. He ambulated without assistance, and interestingly, he walks with a left limp rather than a right limp, even though he has previously alleged having a right hemiparesis. While performing drawing activities for me, he uses his left hand, claiming that he is left-handed. His wife reported in his questionnaire that he was previously right-handed and is now left-handed. When he stands up to leave the room, he extends his right arm in an unsolicited fashion to shake my hand. He has a very strong grip. One can observe this on the videotape of my examination.

He does allege that he cannot read and write. The medical records suggest that this is due to aphasia, which he claimed or demonstrated at the time of his alleged traumatic brain injury. I let him speak considerably during the examination, which was videotaped and audio-recorded. Actually, he produces quite a bit of complex language, and on open-ended questions, he is able to generate language without difficulty, indicating that there is no anterior brain dysphasia. He produces excellent language for me with some hesitancy and occasional stuttering. He claims to forget facts but then is able to tell me that he has a "herniated disc" from a back fall, and that he had "spina bifida" as a youngster. He further states that he attended college at "Pleasantown University" and that he received a degree from "St. John College" and also received a degree from "Joy City College." He told me that he intended to be a "counselor" to help youngsters not to turn out the way he did, and that he had obtained his degree in psychology and sociology in order to do so.

I asked him to count from 20 to 1 backward, and he said he couldn't. I then asked him to count from 1 to 20 forward, and he stated he can't do that either, as he is "poor with numbers." I asked him if he can subtract in his head, and he claimed to not be able to do so. When asked who is the president of the U.S., he stated, "I don't know." On the other hand, he is able to name Japan as the country that bombed Pearl Harbor in World War II, and he is able to name New York as the city in which the World Trade Center was located.

He has no difficulty with repetition, and he is able to repeat "Methodist Episcopal," "the little boy went home," and "the fat, short boy dropped the china vase." When asked to name common items in his visual space, he was able to name *glasses*, *pencil*, and *cup*. He was able to draw intersecting pentagons and did so with his left hand, but had a tremor from using the left hand. The form of the pentagons is intact and each has five sides. The intersection is completed appropriately with the stimulus picture.

He subjectively claims to be depressed, but he has a good range of affect and no depressive facial expression. He is not tearful at any time. He tells me that he sees his dead mother and grandmother. I asked him how they came to be dead, and while complaining of a memory disorder, he was able to tell me that his brother and nephew were killed in a motor vehicle accident and his grandmother was killed in a separate motor vehicle accident. He then says that they talk to him and instruct him to get into a car.

Articulatory agility is reasonably good, particularly when he repeats words. He does stutter occasionally, and he has some slight hesitancy and latency of response. The latency of response grows less and less the longer we talk. With open-ended questions, his language generator is fluent, the syntax is intact, and grammar is appropriate. The melodic line is mildly constricted, but the phrase length is excellent. There were no paraphasias or word-finding difficulty noted. S.T.'s comprehension is excellent and his expressive language is excellent. Whenever he chose, he said, "I don't know." He inconsistently said "I don't know" to the question of who is the president of the U.S., but then responded that Japan bombed Pearl Harbor and New York held the location of the World Trade Center.

On the right side, he has no hyperreflexia, and the right and left reflexes are symmetric in the biceps, triceps, brachioradialis, patellar tendon, and Achilles reflex. He shows no loss of vibratory sensation in the feet or hands. He does claim that the Weber sign lateralizes to the left. He denies an ability to appreciate peppermint or anise oils. His nose response and facial expression indicate that he does smell what I ask him to smell, but if he says "I don't know" to a smell test, there is no way I can determine further his olfactory capability. He fixes his eyes on me well. Pupillary size is normal, and there is a variability of pupillary diameter depending on the light that shone on his face, the direction of his face toward the camera, and his level of excitement.

When I first met him the morning of his examination, he walked down the hall dragging his right foot and holding his right arm in a flexed posture. As I observed him leave my building with his dog, he was holding his dog with both arms and walking without a limp. As the observer will be able to see easily on the videotape, he gestures significantly with both the right and left hands, and there is no hand preference or observable impairment of the hands or arms.

## **MAGNETIC RESONANCE IMAGING**

An MRI of the brain was obtained at the St. John Hospital on February 8, 2002. I asked George Radiology, M.D., to interpret the images for me, and I advised Dr. Radiology that S.T. was alleging a right hemiparesis. This focused Dr. Radiology onto the left hemisphere of S.T.'s brain.

As is customary in my practice, Dr. Radiology issued me a report. The intracranial study was normal. No focal encephalomalacia was present. The temporal lobes were symmetrical, including the hippocampal complexes. The MRI of the brain was interpreted to be within normal limits.

## **STANDARDIZED MENTAL ASSESSMENT**

Forensic Doctor, M.D., requested formal neuropsychological testing. These tests were performed by Mary Psychologist, M.S. Alice Psychologist, Ph.D., supervised the administration, scoring, summarization, and analysis of the test data. These persons are agents or employees of Forensic Doctor, M.D., P.S.C. Medical conclusions about these data were made by Forensic Doctor, M.D.

*Standardized* means that the administered test instruments had exact rules for test administration and exact rules for test scoring. These rules have been standardized and published and are expected to be utilized by any person performing or interpreting the following test instruments.

The following test instruments were administered:

1. Test of Memory Malinger
2. Portland Digit Recognition Test, 27-item short form
3. Victoria Symptom Validity Test
4. Rey's 15-Item Figure Memory Test
5. Structured Interview of Reported Symptoms
6. Wide Range Achievement Test-III, Reading subtest
7. Minnesota Multiphasic Personality Inventory-2

## FORENSIC DISTORTION ANALYSIS

With any standardized mental assessment the recruitment of optimal effort is imperative. The instruments used to assess brain functioning cannot be “faked good.” It is impossible for a brain to perform better than its capacity. However, it is possible for individuals to attempt to present themselves in a negative manner, to “fake bad” or present a “worst-foot forward.” The extreme example of this presentation results in a response pattern referred to as malingering.

The following instruments were administered to determine the level of effort and validity of responses.

### A. Measures of Cognitive Distortion

#### 1. *Test of Memory Malinger*

The Test of Memory Malinger (TOMM) is a 50-item test for adults published in 1996 that is designed to discriminate between malingered and authentic memory impairments. Although the TOMM is sensitive to malingering, it is insensitive to neurological impairment. The TOMM has been extensively validated with groups of cognitively intact adults and with groups of adults diagnosed with cognitive impairment, aphasia, or traumatic brain injury. The TOMM consists of two learning trials and a retention trial. A low score on the TOMM suggests that memory impairment symptoms are false or exaggerated. S.T. produced a score of 28 on Trial 2 and a score of 29 on the Retention Trial. This pattern of performance is in the abnormal range.

#### 2. *Portland Digit Recognition Test, 27-item short form*

The Portland Digit Recognition Test (PDRT) in its 27-item abbreviated short form is a test that employs digit recognition to assess malingering. This is a forced-choice test that requires digit recognition after increasing periods of delay with intervening distraction. A score below 63% correctly classifies 75% of those providing poor effort. S.T. produced a percentage score of 48, which is in the abnormal range.

#### 3. *Victoria Symptom Validity Test*

The Victoria Symptom Validity Test (VSVT) is based on a forced-choice paradigm that requires digit recognition after increasing periods of delay following stimulus presentation. Recognition items vary in degree of similarity and appear to be easy or difficult. Although difficult items are designed to appear significantly more challenging than easy items, actual differences in performance across these two types of items are minimal in normal individuals. Results are provided for the number of easy, difficult, and total items correct, as well as the number of items correct and response latencies in each block. VSVT classifications are determined on the basis of binomial probability theory and are specifically designed to eliminate false positives.

The results of the VSVT for S.T. are summarized as follows:

<b>Victoria Symptom Validity Test</b>		
	<b>Raw Score</b>	<b>Classification</b>
Easy items correct	24/24	Valid
Difficult items correct	13/24	Questionable
Total items correct	37/48	Valid

Overall, the results of this measure are within the questionable range.

4. *Rey's 15-Item Figure Memory Test*

Rey's 15-Item Figure Memory Test (Rey-15) is a standardized assessment instrument used to determine the validity of memory complaints. The underlying principle is that the individual who either consciously or unconsciously attempts to appear impaired will fail at a task that all but the most severely brain damaged or retarded patients can easily perform. The individual is presented the task of memorizing 15 different items. While the number 15 is stressed in order to make the task appear difficult, in reality, the individual need only remember a few of the ideas to recall these items. After presentation of 10 seconds, the individual is asked to copy the 15 items. A score below 10 will correctly classify 93% of those providing poor effort. Psychiatric inpatients average a score of 13.5; mildly mentally retarded persons average a score of 9.9. S.T. produced a raw score of 4, which is in the abnormal range.

B. Measures of Psychological Distortion

1. *MMPI-2 Validity Indices*

MMPI-2 validity indices are derived from the MMPI-2 responses. The MMPI-2 is the most recent (1989) revision of the original 1943 MMPI. In addition to the four previous validity scales — Cannot Say, L, K, and F scales — five additional scales are provided: back-page Fb, TRIN, VRIN, Fp, and S.

S.T. produced the following profile:

Scale	Cannot Say	VRIN	TRIN	F	Fb	Fp	F-K	L	K	S
Raw	0	6	7	17	7	4	-4	5	21	33
T-score		54	64	89	71	70		56	62	59

The above MMPI-2 validity measures indicate high elevations of the scales measuring infrequent responding. Although the elevations of both the F and Fp scales may reflect psychopathology, they most likely contain elements of symptom exaggeration.

2. *Structured Interview of Reported Symptoms*

The Structured Interview of Reported Symptoms (SIRS) by Richard Rogers, Ph.D., was developed to assess systematically deliberate distortions in the self-report of symptoms. The scales of the SIRS provide useful information regarding how an individual may fabricate or distort his or her symptom picture. The SIRS consists of eight primary scales and five supplementary scales. The primary scales provide descriptions of response styles that classify individuals as honest or feigning. Supplementary scales are used for the interpretation of response styles. Scores are determined based on responses that can receive either a 0 score for no or nondeviant responses or a score of 1 or 2 for increasing levels of endorsement. Two classifications are utilized — probable and definite — to determine the level of symptom distortion for the primary scales. Scores in the probable range accurately differentiate at least 75% of the criterion group, while scores in the definite range accurately classify 90% or more of subjects.

S.T. produced the following profile:

**Structured Interview of Reported Symptoms**

Primary Scales	Raw Score	Classification
Rare Symptoms (RS)	0	Honest
Symptom Combinations (SC)	0	Honest
Improbable or Absurd Symptoms (IA)	2	Honest
Blatant Symptoms (BL)	3	Honest
Subtle Symptoms (SU)	4	Honest
Selectivity of Symptoms (SEL)	5	Honest
Severity of Symptoms (SEV)	2	Honest
Reported vs. Observed Symptoms (RO)	0	Honest

No primary scales of the SIRS are elevated.

The following supplementary scale of the SIRS is low: Defensive Symptoms (DS)

## MEASURES PROVIDING ESTIMATE OF PREINJURY FUNCTION

### *Wide Range Achievement Test-III, Reading subtest*

The Wide Range Achievement Test-Revised (WRAT-III) is a screening measure for academic achievement. It assesses reading recognition, spelling, and arithmetic. Raw scores are determined in each of these areas, and grade equivalence and percentiles are assigned. Standard scores are also obtained, in which the mean (average) is represented by a standard score of 100 and the standard deviation is 15. Standard scores are used for interpretation. Reading scores tend to resist dementing processes. Spelling and arithmetic scores may be used for vocational assessment.

S.T. produced the following raw scores and derived scores:

### Wide Range Achievement Test-III

	Raw Score	Standard Score	Percentile	Grade Equivalence
Reading	14	< 45	< 1	K

S.T.'s performance on this measure is in the extremely low range.

## ASSESSMENT OF EMOTIONAL ADJUSTMENT

The MMPI-2 was administered to provide hypotheses regarding the psychological function of S.T. The validity of this test with individuals who have experienced a traumatic brain injury has not been verified. The standard interpretations of clinical scales may not apply to individuals with suspected brain injury. The interpretations presented in this report need to be verified by other sources of clinical information.

### *Minnesota Multiphasic Personality Inventory-2*

The Minnesota Multiphasic Personality Inventory-2 (MMPI-2) is the 1989 revision of the original 1943 MMPI. The MMPI is an objective personality test composed of 567 statements to which the test taker responds true or false. It is a widely used, well-accepted measure of personality, providing information about a variety of psychiatric symptoms and problems. Four previous validity scales (L, F, K, and Cannot Say) along with five new scales (Fb, TRIN, VRIN, Fp, and S) provide an assessment of response biases and test validity. Ten basic clinical scales, plus the supplementary and content scales, may be scored. Raw scores are converted to uniform T-scores to allow easy comparison. Normal T-scores are generally in the range of 50 to 64.

Profile data for S.T. are as follows:

### Minnesota Multiphasic Personality Inventory-2

Scale	VRIN	TRIN	F	Fb	Fp	L	K	S		
T-score	54	64	89	71	70	56	62	59		
Scale	1	2	3	4	5	6	7	8	9	0
T-score	94	83	96	74	46	64	81	89	49	60

## RECORDS REVIEWED

### *Accident and Postaccident Records*

- Uniform Police Traffic Accident Report
- Emergency Medical Services Ambulance Run Report
- Records of Tri-County Baptist Hospital/ER
- Emergency Medical Services Ambulance Run Report
- Records of University of Anytown Hospital

Records of June West, M.D., and Mofi Safir, M.D., University Psychiatric Services, P.S.C.  
Records of Kentucky Medical Imaging  
Records of Ann Guest, Ph.D.  
Photographs of S.T. vehicle damage  
Plaintiff's Answers to Interrogatories  
Surveillance videotapes made by defendant lawyer  
Deposition of John Wash

*Preaccident Records*

U.S. District Court/Western District of Kentucky-Louisville Division/Presentence Report  
U.S. Department of Justice/Federal Bureau of Investigation/Identification Record  
Records of Janice Best, M.Ed., CPC  
Criminal Records/Department of Corrections Records  
Deposition transcript of S.T.  
Deposition transcript of Mrs. S.T.  
Anytown counseling records  
Expert Witness Pretrial Disclosure  
Dr. Bill Post  
Dr. Sam Smith  
Jasmine Lock, R.N.  
Dr. Janet Jackson  
Dr. Jake Post  
Dr. Ari Sidiq

## **NEUROBEHAVIORAL ANALYSIS**

*Neurobehavioral analysis* is a term for the analytical evaluation of the neuropsychiatric examination data producing a comprehensive statement about the person's brain-behavior capacity. It is based upon historical, neurological, psychiatric, brain imaging, prior records, and neuropsychological data.

S.T. failed four of the four cognitive effort tests. Thus, I can confidently state that he's faking his cognitive effort. Moreover, he produced a reading recognition score below the lowest level of mental retardation. This is impossible in a person who graduated from college and then completes my 22-page questionnaire, unless that individual currently has a severe receptive (not expressive) aphasia. There is no evidence whatsoever that S.T. has an aphasia. His MMPI-2 is consistent with symptom magnification.

His behavior in my waiting room and in the parking lot is consistent with a man faking his impairment. For a few minutes, he will hold the right side of his body as if he has a hemiparesis and then shake hands with his right hand. On other occasions, he has a left limp and then switches to a right limp. His apparent language dysfunction and stuttering is inconsistent with any known aphasic syndrome.

Lest the reader of this report forget, S.T. has had many years to learn dissimulation in prison. Moreover, he received a degree in psychology. Thus, he has had the laboratory learning experience of the federal and state penitentiary systems coupled with a degree in psychology. This enables him to develop an amateurish fake of a brain disorder.

In my opinion, the reason he was diagnosed with an aphasia at the time of his accident is because he did not speak. However, his "aphasia" remitted spontaneously and in a markedly rapid fashion inconsistent with actual traumatic aphasia. He's perfectly capable of reading and learning what behavior is required to produce a picture of aphasia. Moreover, it's just as likely as not that he either staged his accident to accrue to his benefit or fell asleep at the wheel from obstructive sleep apnea and then faked a brain injury. The police officer could not verify that anyone struck his vehicle.

His motor vehicle accident was December 21, 1999. If one reviews the Anytown counseling records, S.T. admitted to "hearing voices" or "seeing things" on July 27, 1999. Moreover, he admitted psychotic symptomatology to Dr. Smith in a May 13, 1999, report. Thus, we have incontrovertible evidence that

he knew well how to fake a psychotic illness before the alleged motor vehicle accident on December 21, 1999. This means that either he was psychotic before the accident and now remains psychotic or he was already faking a mental disorder before the alleged accident, again to gain some type of benefit to him. He is not presently psychotic.

I reviewed color pictures of his vehicle. The damage is insufficient to cause a brain injury and is merely cosmetic. The surveillance videos obtained outside my office indicate that his observable function is not consistent with a brain injury or psychosis. No hemiparesis is noted. S.T. speaks to others without difficulty on this tape.

### **DIAGNOSES (DSM-IV-TR)**

Axis I:

A. Malingering a mental disorder for primary and secondary gain

B. No evidence of a traumatic brain injury

Axis II: Severe antisocial personality disorder with a long history of federal and state penal incarceration

Axis III: No evidence of a medical disorder affecting his mental state

Axis IV: Profoundly and severely dysfunctional lifestyle present since childhood

Axis V: Unable to accurately determine due to the faking he introduced into the testing situation

### **CONCLUSIONS**

1. In my opinion, within reasonable medical probability, S.T. has a 0% neuropsychiatric impairment due to a motor vehicle accident on December 21, 1999. This is based on *Guides to the Evaluation of Permanent Impairment* (American Medical Association, Chicago, 2000), page 320, Tables 13-5 and 13-6 (CDR score = 0).
2. In my opinion, within reasonable medical probability, S.T. has the mental capacity to engage in any work he is trained, educated, or experienced to perform.

Respectfully submitted,

Forensic Doctor, M.D.

### **EXPERT TESTIMONY**

Most physicians do not enjoy providing expert testimony, avoiding it at all costs. However, if a physician chooses to examine a traumatically brain-injured person within a forensic setting, it is expected that the physician may testify at court or by deposition. Recall discussions elsewhere in this text: the forensic examiner is to strive always for honesty and objectivity within the examination, report writing, and testimony, regardless of who hires the physician. The methods whereby the physician functioning as an expert witness gives data regarding the neuropsychiatric examination and the opinions derived therefrom can help or hurt the perceptions of the physician by the trier of fact. Thus, it is important that physicians attempting to be expert witnesses gain skill in teaching their opinions to triers of fact. The less skillfully the expert witness teaches, the more likely the message will not be received by the trier of fact.

The physician should clearly understand the differences in training, philosophy, and objectives between lawyers and physicians. The physician as scientist-practitioner is accustomed to the pursuit of truth through dispassionate examination of data, whereas the plaintiff and defense attorneys are instead committed primarily to persuasion.<sup>1</sup> Moreover, attorneys are more interested in credibility than in truth per se, and many of the questions posed in court by attorneys are aimed at enhancing or undermining the expert witness's credibility. The physician examiner needs to understand these differences in philosophy well and adjust to them. It should be remembered that the lawyer is far more skilled at the presentation of information in court than the average physician expert witness.

On the other hand, the physician expert usually knows far more about the subject matter being presented to the court than the lawyer.

## **THE NATURE OF TESTIMONY**

There are many ways that a forensic physician can testify or provide evidence to a court in a matter regarding brain injury. One method is by *affidavit*. This is a sworn written statement attested to by the physician and generally presented to a court over the notarized signature of the physician. One manner in which it is often used relates to the plaintiff's attempts to avoid examination by a physician hired by the defendant in a brain injury action. If the plaintiff lawyer dislikes the known examination techniques of the physician, or if he is concerned that the physician is highly skillful, the plaintiff may avoid the examination by notifying the court that the physician practices too far from the plaintiff's home, or will place an undue burden upon the plaintiff during the examination. In turn, the defense lawyer may ask the prospective examining physician to provide an affidavit to the court outlining the nature of the examination, what tests are expected to be performed, and the duration of the examination. On other occasions, the examining physician's skill in the area of brain injury may be questioned, and it may be necessary for the physician to supplement information about training and experience in brain injury by way of affidavit. Thus, affidavits can be used in any manner to present the court a sworn statement in order to answer an objection by the opposite party.

Another method whereby the court receives information is through an *interrogatory*. While this is not presented as formally as testimony in a deposition, it does provide answers to specific questions for the court's review and forms a record during the litigation to be considered by the court. For instance, if the physician is hired by the plaintiff attorney, the defense attorney may ask the plaintiff attorney to provide answers to interrogatories about the expert physician. The most common question posed is: "What will the physician testify at trial about the alleged brain injury in the plaintiff?" The physician is urged to be careful regarding interrogatories. It is not unusual for lawyers to write interrogatories in answer to each party's questions without including the physician in the loop. Thus, the physician can be hired by a plaintiff attorney, an interrogatory of the defense attorney can be answered regarding what the physician is expected to testify at trial, and the physician may then be presented with the interrogatory during a deposition and realize that it is a document the physician has never seen. This is not to imply any form of trickery in this regard; interrogatories are standard methods of information transfer between parties at trial. It is important that the physician be aware of these and ask the retaining lawyer if specific answers regarding the physician's opinions have been supplied by interrogatory.

With regard to oral testimony, expert witness physicians generally present this one of two ways: by deposition or live at trial. Depositions usually are given for discovery, evidence, or unusual matters beyond the scope of this text, such as spoliation of evidence (the intentional destruction of evidence) or other procedural or evidentiary matters that may be argued to the court by either the plaintiff or defense lawyer. In most instances, regardless of whether the expert physician is hired by the plaintiff or the defendant, a deposition is usually taken prior to the expert appearing at trial. There are some instances, however, when no depositions may be taken and the first testimony given by the expert witness physician will be directly to the jury. This is more likely to occur in criminal matters rather than civil actions.

## **DEPOSITION FOR DISCOVERY**

If the physician has provided an examination to a person alleging brain injury, a discovery deposition may be triggered by the process. If the plaintiff has hired the physician, the defendant may take the deposition of the physician to determine the nature of the examination, the diagnoses made by the physician, and the conclusions expected to be given by the physician at court. If the examination has been made at the request of the defendant, the plaintiff in all likelihood will take a discovery



deposition of the physician to determine the same information. Simply put, the lawyer on the side opposite the examining physician wants to know what the physician will say if called to trial. The information taken from the physician during the discovery deposition is then “written in stone.” Thus, the physician cannot change conclusions or change testimony at a time thereafter. Once the physician has given a discovery deposition, the physician’s conclusions are locked in place and those same conclusions will be expected to be given at trial. There are exceptions. If new evidence is presented to the physician, an addendum report may be necessary to the original report, as the physician’s conclusions may change based upon the new data. Both the plaintiff and defense lawyers must be made aware of this change, and sometimes it is necessary for a physician to give a second discovery deposition should that occur. At other times, the plaintiff and defense lawyers may stipulate that the physician has changed conclusions and the issue will be addressed at trial.

In most instances, discovery depositions are recorded solely by transcript. However, there is a more recent move among some lawyers to videotape the discovery deposition of a physician. When this occurs, the physician should be extremely careful. During a discovery deposition, the rules of questioning of the physician are fairly loose. They are loose both for the lawyer taking the information and for the physician providing testimony. The strict rules of evidence relax during discovery, and they allow substantial latitude in how a question may be posed to the physician and also how the physician may answer the question. These rules of direct and cross-examination are far more stringent when a deposition is being taken for evidence. The purpose of the discovery videotape often is to catch the physician off guard during the discovery process, anger the physician, and attempt to cause the physician to be hostile or demonstrative to the lawyer taking the discovery deposition; then the tape is used at trial as a visual example to the jury to discredit the expert witness or show her as an advocate rather than an impartial expert. The physician testifying in the courtroom later may present an image of self-composure and compassion. Thereafter, during cross-examination, the lawyer who did not hire the physician may show the jury the discovery deposition tape or excerpts therefrom to demonstrate that the physician testifying in front of the jury is a very different person when outside the view of the jury. Another interesting technique often used by lawyers who videotape discovery depositions is to turn the discovery deposition into a deposition for evidence. Therefore, rather than ask the examining physician questions about the neuropsychiatric examination of the plaintiff, the lawyer may ask the questions in a manner designed to elicit evidence from the physician rather than opinions. For instance, during the videotape discovery deposition, the lawyer may read into the record excerpts from textbooks that provide opinions opposite that of the examining physician or may make statements to the physician in an effort to have the physician concede multiple points and elements outside the examination. These will then be shown to the jury at trial. Remember, when lawyers worry they are losing on the facts, they may attack the expert to avoid substantive or probative issues.

Lawyers who are very technically expert in brain injury cases usually do not engage in discovery deposition histrionics. These lawyers are quite efficient and realize they are not at a discovery deposition to change the expert physician’s opinion. Their discovery style is usually more direct and organized. They will ask the physician the elements of the examination, the diagnoses, and the conclusions. They generally will ask the physician to tell them “each and all conclusions” that will be given at trial. Often, they will end the deposition by asking the physician to make them aware of any further information that occurs after the date of the deposition that might change the physician’s opinions prior to trial.

## **DEPOSITION FOR EVIDENCE**

This deposition is usually taken by the lawyer or agency who hired the physician. It is usually taken for one of two purposes: (1) to preserve the evidence in case the physician should die, be injured, or otherwise be unavailable to testify at trial, or (2) to present testimony to the jury in lieu of live testimony. However, occasionally the deposition for evidence will be taken by a lawyer who

did not hire the physician. For instance, if the physician was hired by the defendant to examine a person who may have sustained a traumatic brain injury and the physician's conclusions do not assist the defense, the plaintiff lawyer may in turn take the deposition of the physician instead. This is perfectly acceptable within the law. While the physician should remember for whom he was employed, and he has no obligation to speak to the plaintiff lawyer prior to the deposition, he should provide a polite direct deposition and answer the plaintiff's lawyer's questions about his client. Obviously, the reverse could occur; that is, the physician examines a plaintiff and finds no evidence of brain injury. Then, the defense might call the physician to provide testimony regarding the examination. The same rules of civility apply.

In today's modern legal practice, most depositions for evidence are videotaped. Occasionally, depositions are recorded by transcript only. However, nothing is more boring to a jury than to have a third party sit in the witness box and read the transcript answers of a physician deposition. Therefore, most skilled lawyers avoid presenting this type of evidence at trial. The deposition also may be taken in a more technically advanced nature due to the mobility and location of persons within the U.S. For instance, on occasion, the physician may be deposed by computerized televideo. The physician may be asked to go to a video technical center, sit in front of a computerized television system, and be cross-examined by a lawyer in city A while providing direct testimony for a lawyer in city C with the expert witness sitting in city B. Courts and lawyers have found that for certain situations, great cost savings can be obtained with this new use of technology. In some instances, testimony may be given directly to courts wherein the judge receives the testimony by computerized video-conferencing with the expert physician over the Internet.

## COMMUNICATING THE MESSAGE

It has been repeatedly stressed, and is stressed again, that if the expert witness cannot skillfully communicate the message, the trier of fact will not receive it. Many physicians find in a medical-legal forum that their vast array of scientific knowledge and medical skills does not provide them the necessary artistry to communicate their findings. It cannot be overemphasized that plain talk regarding complex medical examinations and procedures is the communication style of choice when speaking to a trier of fact. On November 19, 1863, President Lincoln presented the Gettysburg Address. This is felt by many to be the most profound verbal monument ever erected to military achievement and human sacrifice. It is instructive to those functioning as expert witnesses to reread that short speech. While the language is elegant, it is also straightforward. A preponderance of nouns and verbs were used by Lincoln, rather than adjectives and adverbs. While the speech contained abstract principles such as liberty and equality, it focused on concrete manifestations of those principles to the benefit of the audience. The speech was given not as a lecture but as a kind of conversation. It is a dramatic representation of "plain talk."<sup>7</sup> A physician who expects to be an effective communicator in a courtroom must learn to speak simply and in a straightforward manner. The average juror in the U.S. is a high school graduate or, oftentimes, less educated, and the language style of Harvard, Stanford, or Johns Hopkins may not be well received. The expert should learn to tell a story to the jury when providing testimony. We speak to each other generally with stories, not cold, hard facts.

The expert should strive to engage the audience. It is not just what you say, but how you say it.<sup>8</sup> Communicating without enthusiasm, in a stiff manner with little animation, and in particular speaking in a monotone, is an exact recipe for dismissal of your message. An effective witness cannot engage the audience without making eye contact. The physician should speak to one person at a time on the jury. If being videotaped, it is particularly important to look directly into the camera lens repeatedly. If the physician has not been looking directly at the camera, then as jurors see the video monitor in court there will be almost no eye contact made between the jurors and the television screen. Can you imagine how you feel when you examine a patient who looks at the wall or the floor when answering your questions? You do not communicate with your patient and you have no

confidence in that person. The jury will feel the same about a physician who testifies with slouched posture, lounges within the chair, leans backward, looks at the floor or ceiling, and avoids making eye contact with the lawyer or the jury. In U.S. culture, we expect good and direct eye contact. All communication with humans begins with the eyes first. This is from the lowest level of mother–child interaction to the first contact with a prospective lover. Eye contact opens the channel of communication between people, and it establishes and builds rapport. It involves the audience in the presentation. Thus, regardless of whether you testify for the plaintiff or the defendant, your message will be lost if you do not engage the audience.

Lawyers are painfully aware that the attention span of the jury, or any audience for that matter, is reasonably short. Unfortunately, many trial lawyers forget this and drone on and on with arcane points that go beyond the understanding of the jury. The physician expert will do well to remember the advice of Charles Osgood.<sup>9</sup> His well-received text on public speaking points to the “12-minute secret.” The standard length of a Vaudeville act was 12 min. It was the belief of showmen that no act, other than the headliner, could sustain audience interest for longer than that. Many lawyers hold that 15 to 20 min is the most effective duration of presentation to the jury. Obviously, the physician expert is at the mercy of the lawyer who called him to trial. However, when presenting oral testimony, it is wise to remember that most jurors have grown up in the era of television. Thus, they are used to sound bites. Skilled news anchors present the news in short phrases and short sentences. Rarely do they present factual information with long paragraphs. Not only is this a well-recognized fact of television news reporting, but also it is a well-recognized fact of scientific evidence that attention and vigilance wane quickly.

The physician expert providing neuropsychiatric testimony should be aware that nonverbal communication is often as important or more important than the content of the message. In order to develop effective nonverbal communication or “body language,” self-awareness is the starting point. The Johari window emphasizes that several aspects of the self are not separate and distinct pieces. In order to be an effective communicator, it is necessary to understand the four selves:<sup>10</sup> (1) the open self, information about yourself that you and others know; (2) the blind self, information about yourself that you do not know but others do; (3) the hidden self, information about yourself that you know but others do not; and (4) the unknown self, information about yourself that neither you nor others know. As you increase self-awareness, you will develop those skills of communication that enhance effective body language. This will reduce apprehensiveness, as some research suggests that people respond more negatively to those they perceive as apprehensive than to those they perceive as more confident and less fearful.<sup>11</sup> Apprehension is best managed by acquiring communication skills and experience, focusing on one’s success, and reducing unpredictability by studying the material one wishes to testify about until it is known effectively. Furthermore, apprehension and improved body language communication can be enhanced by testifying in an unhurried manner. Pacing is important. Articulate each word in a sentence. Do not use gestures that are not natural to you. Gestures should be enhancing and “direct the traffic” of ideas and images conveyed to the trier of fact.

Further important principles of body language in effective communication include sitting to one’s full height. Keep the shoulders straight and lean forward slightly to enhance communication between oneself and the jury. Maintain eye contact with the lawyer and then back to the jury. Pay careful attention to prosody (see [Chapters 4 and 6](#)). The affective components of language are important and prevent the perception of dullness. Testifying about brain injury is an interesting and engaging act. However, some physicians have a remarkable talent for allowing exciting concepts to be treated as deadly dull clunkers. Describe dull facts as narratives. Use stories and make analogies that the jury can relate to their personal lives and the experiences of themselves with their children or parents. For instance, when describing the language dysfunction of a brain-injured person who displays severe circumstantiality and poor language monitoring, the physician can state that the plaintiff’s language during mental status examination was like that of one’s grandmother who cannot get to the point and causes the person to want to jump in and say, “Granny, tell the

story.” The humanizing of complex neuropsychiatric issues is a skill that expert witnesses should strive to learn. When testifying to a jury, the physician should never appear arrogant or haughty. In fact, it is important to remember that when asked by a lawyer to testify at court, the physician is a medical doctor first, specialist second, and forensic expert last.<sup>12</sup> The jury will be most interested in one’s basic skills as a physician and only secondarily interested in one’s skills as an expert. The projection of physician compassion and humanness will go a long way to remove any faults made apparent on cross-examination. None of us can testify without at least a few warts being recognized. Project the image of medical competence always. Dress should be appropriate to the occasion: for men, a conservative business suit and tie; for ladies, an appropriate business suit or dress. Excessive jewelry, gold chains on men, and casual appearance suggest that if the physician is this laid back in court, examination and conclusions may be sketchy and laid back as well. Moreover, histrionic dress or dress that is too casual suggests that the physician does not respect the importance and decorum of the court. This is another form of negative nonverbal language that even if done with no ill intent may be misperceived by the jury and the judge.

## CROSS-EXAMINATION

Anyone who graduated from medical school should be able to provide a reasonably decent direct examination for a lawyer. This is because the lawyer who hired the physician will throw “softballs” to the physician and allow her to express her opinions in an easy, nonchallenging format. It is cross-examination that tests the mettle of the physician and demonstrates best weaknesses in her communication skills, projection of empathy, and credibility. The object of cross-examination is to test the truth of statements of a witness made on direct examination and to sift, modify, or explain what has been said. Furthermore, its effect is to weaken or disprove the case of the lawyer’s adversary. A clear-cut, forceful answer given by a witness on cross-examination is more deadly in its effect on a jury than the same answer given on direct examination.<sup>13</sup> In fact, lawyers are instructed to focus their fire on the main adverse witness. If you are the witness upon whom the proof of brain injury damages hinges, you may rest assured that many arrows will come your way during cross-examination. You may be suave and skilled at the country club, but if your facts fail you during cross-examination, you may go the way of suave psychiatrist Carl Binger, M.D., who was brought in as an expert witness to destroy Whitaker Chambers as a “psychopathic liar” in the Alger Hiss trial. Chambers was the principal witness against Hiss. The defense lawyer planned to use Dr. Binger to discredit Chambers’ damning testimony against Hiss, his client. When Thomas F. Murphy, the prosecutor, rose to his full height of 6 ft, 7 in. to cross-examine Dr. Binger, he turned the case around and Hiss was convicted. Murphy spent more than 100 h in preparation for the cross-examination of Dr. Binger. He did not attack Binger as a person but demonstrated that Binger’s conclusions about Chambers were based upon erroneous facts that led to an inadequate and erroneous conclusion. Binger was accepted as an expert and then hoisted on the petard of his own poor factual database.<sup>14</sup>

Frankly, a physician expert who states that he does not feel at least somewhat nervous during cross-examination is disingenuous. It is during cross-examination that one’s communication skills and comportment come to the front. Never be arrogant with the cross-examining lawyer. While one should not be deferential, it is extremely important to be polite, respond with, “Yes, sir” or “No, ma’am,” and refuse to be baited into anger. The greatest ally a cross-examining lawyer has in the courtroom is to anger the expert witness in front of the jury. By anger, the witness may demonstrate to the jury advocacy or the lack of composure that jurors expect in their own physician when faced with stressful circumstances. By treating the cross-examining lawyer with the utmost respect, even if he or she rants and raves at the physician witness, the jury will grant you respect. If the jury dislikes the lawyer’s attack of you, they will dislike the lawyer’s client.

It is extremely important during cross-examination to maintain credibility in the face of a vigorous verbal attack. For instance, if the physician is asked if he has ever made a mistake, the

appropriate response is “yes,” assuming that one has made mistakes. In most instances, the jury would not believe otherwise. Moreover, if it becomes necessary to testify that the person who was examined demonstrated symptom magnification or even malingering, the physician must have unequivocal scientific facts and medical evidence to support the conclusion. It can be assumed that if it is necessary for a physician to state that an alleged traumatically brain-injured person is malingering the examination, vigorous cross-examination will ensue in an effort to discredit the physician or to show the physician to be an advocate for the person or agency who hired the physician to perform the examination. The most effective physician expert witnesses are those who present themselves with humility, composure, scientific skill, and expertise, and are at all times polite and respectful of the cross-examining lawyer, even if he or she makes *ad hominem* attacks upon the physician.

## USE OF EXHIBITS IN THE COURTROOM

The use of exhibits by the expert witness is controlled by the attorney who hired the witness. However, during a video deposition, or in a courtroom, little is more effective than visual exhibits. Their use cuts both ways. They can be useful to demonstrate lack of brain injury or extremely useful and effective in demonstrating actual brain injury. Depending on the lawyer’s needs, the exhibits may be rendered by a medical illustrationist, produced graphically by computer, or displayed as an MRI or CT image using a standard shadowbox or PowerPoint™ exhibit.

If visual aids are used, they should be clear, consistent, and dynamic. The visuals should match the evidence given by testimony. Since visual aids in the courtroom are evidence, they must be presented to the other side in a litigation prior to their introduction into the courtroom. This is an issue for and between lawyers rather than for physicians. However, as lawyers must present their witness list to the opposing party in a trial, they also must list evidence and visual aids that they will use at trial.

Visual aids in the courtroom provide substantial enhancement of one’s testimony. They provide an outline for the jury so they know where the expert has been and where the expert is going with the testimony. They provide an unwritten outline to assist with the presentation of testimony. They support the oral testimony and will increase what the jury remembers as the jury has then received both an auditory and a visual stimulus. If necessary, brain images may be supplemented with numbers and graphs, such as alteration in WAIS-III IQ scores or the graphical display of abnormal findings on MMPI-2.<sup>15</sup>

In terms of enhancing the message to the jury, exhibits provide another opportunity for increasing communication by the physician expert witness. They enable the expert to teach directly to the jury. With the judge’s permission, the expert can leave the witness box, place the exhibit in front of the jury, and provide a more intimate teaching format to the jury. This enables even greater levels of eye-to-eye contact with the jury, and the movement afforded the expert witness enhances bodily communication with the oral message. These factors, if one plans to use them in the courtroom, should be discussed beforehand with the lawyer who retained the physician expert.

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