



Traumatic Brain Injury



Independent Study Course Released: January 2004

Sponsored by
Department of Veterans Affairs
Employee Education System

This is a Veterans Health Administration System-Wide Training Program sponsored by the Veterans Affairs Employee Education System and the Office of Public Health and Environmental Hazards, Department of Veterans Affairs. It is produced by the Employee Education System.





DEPARTMENT OF VETERANS AFFAIRS
UNDER SECRETARY FOR HEALTH
WASHINGTON DC 20420

A MESSAGE FROM THE UNDER SECRETARY FOR HEALTH

I commend you for choosing to take advantage of this self-study module, “Traumatic Brain Injury.” This Veterans Health Initiative is being published at a time when our country is engaged in military conflict. Our injured active duty military are relying on the VA to provide them the best care possible and some will be counting on us for this care for the rest of their lives.

Warfare is changing and as weapons have become progressively advanced, injuries have become more complex. In VA, we are constantly preparing to meet these challenges by educating our clinicians. Traumatic brain injury (TBI) is a condition that requires specialized medical care and rehabilitation. Persons with a brain injury are often referred to as the “walking wounded.” Their greatest challenges are often invisible to those who come in contact with them.

VA and the Department of Defense recognized the need to provide specialty care for active duty military and veterans sustaining a brain injury. Through joint collaboration, the Defense and Veterans Brain Injury Program was created in 1992, with 3 military and 4 VA medical centers as the core treatment sites. Subsequently, VA established a TBI Network of Care with designated facilities that support the 4 VA TBI Centers.

The information in this module will assist you in your daily practice. By increasing awareness of the varied symptoms, treatment interventions, long-term care needs and resources; you will be better able to respond to this type of medical condition. Your commitment to continuing professional education ensures that veterans receive the highest quality care. I encourage all VA clinicians to take advantage of this opportunity to enhance their knowledge of caring for persons with traumatic brain injury.

A handwritten signature in blue ink that reads "Robert H. Roswell".

Robert H. Roswell, M.D.

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Independent Study Outline

This independent study presents an overview of Traumatic Brain Injury (TBI) issues that Primary Care practitioners may encounter when providing care to veterans and active duty military personnel.

Purpose

This independent study module is a part of the Veterans Health Initiative (VHI). This VHI is a comprehensive program of continuing education designed to improve recognition and treatment of health problems related to traumatic brain injury.

Background

After completing this independent study, participants will be able to:

Objectives

1. Identify the epidemiology and the nature of TBI;
2. Describe concussion management and treatment of mild TBI;
3. List the rehabilitation process for a Veteran with TBI;
4. Identify medical and physical problems of Veterans with TBI;
5. Identify cognitive problems for Veterans with TBI;
6. Recognize emotional and behavioral problems for Veterans with TBI;
7. Explain TBI in the Elderly and Aging with TBI;
8. List the issues and impact on the TBI survivor and their family;
9. Describe the issues related to driving for a TBI survivor; and
10. Describe the VA TBI System of Care

As a result of this program, clinicians will have a broader base of knowledge with which to provide effective care to patients with TBI and a better understanding of patients who experience this condition. Drug treatments and dosages provided in this study guide should be double-checked prior to prescribing therapy.

Outcome

This independent study is primarily designed for Department of Veterans Affairs clinicians and interested VA staff. Other health care providers, especially those working in veterans and military health care facilities in the U.S., also are encouraged to complete this study module.

Target Audience

This program is available in booklet form and on the Web at:
<http://www.vcampus.com/valo>

Format

Program Description

This Program Includes:

- Independent study written material
- Test for CME credits
- Program evaluation

This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of VA Employee Education System and Department of Veterans Affairs Office of Public Health and Environmental Hazards. The VA Employee Education System is accredited by the ACCME to provide continuing medical education for physicians.

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Independent Study Program Registration/Answer Sheet/Participation
Satisfaction Form

To receive credit for this course:

Program Implementation and VA Application Procedure

1. Read the independent study materials.
2. Complete the CME test questions. A passing score of 70% or higher on the CME test is required to receive credit. This test may be retaken.
3. Complete the program evaluation.
4. The estimated study time for this program is 4 hours.

If you are using the Independent Study Registration/Answer/Evaluation Form (two sided) at the back of the independent study booklet, please send the completed form within two weeks after reading the material to:

Employee Education Resource Center
Attn: SDU
Medical Forum, Suite 500
950 North 22nd Street
Birmingham, AL 35203-5300

NOTE: Scantron forms cannot be photocopied. For additional copies of this independent study, Scantron forms or other VHI independent study modules, please contact your facility education contact person.

If you have attained a passing score of 70% or higher, a certificate will be mailed to you approximately 6-8 weeks after your test has been graded. The test may be retaken.

The CME test and program evaluation can be completed at:
<http://www.vcampus.com/valo>.

After you take the test, you will receive immediate feedback as to pass or fail. You will be allowed to retake the test. Upon passing the test and completing the program evaluation, you will be able to immediately print your certificate according to instructions.

NOTE: If you experience difficulty reaching the course web site, please contact the VCAMPUS Help Desk via email at: valosupport@vcampus.com, or contact your local computer support staff or librarian for assistance.

NOTE: In order to complete the CME test and Evaluation, your computer must have Internet Explorer 4.0 or Netscape 4.0 or higher.

If you have questions or special needs concerning this independent study, please contact:

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This program will no longer be authorized for CME credit after December 2007.

For more information about the products and services provided by the Employee Education System, visit our Web site at: <http://vawww.ees.lrn.va.gov>.

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American Nurses Credentialing Education

VA Employee Education System designates this educational activity for 4.0 contact hours in continuing nursing education.

American Psychological Association

The VA Employee Education System is approved by the American Psychological Association to offer continuing professional education for psychologists. As an organization approved by the American Psychological Association, the VA Employee Education System is offering this activity for 4.0 hours of continuing professional education credit. The VA Employee Education System maintains responsibility for the program.

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The Employee Education System maintains responsibility for the program. A certificate of attendance will be awarded to participants and accreditation records will be on file at the Employee Education System. In order to receive a certificate from EES, you must read the material, complete and pass the CME test with a 70% or higher, and complete a program evaluation.

AMA and ANCC Continuing Education Credits

Report of Training

It is the program participant's responsibility to ensure that this training is documented in the appropriate location according to his/her locally prescribed process.

Disclosure Statement

The Employee Education System (EES) must insure balance, independence, objectivity, and scientific rigor to all EES sponsored educational activities. The intent of this disclosure is not to prevent faculty with a significant financial or other relationship from presenting materials, but rather to provide the participant with information on which they can make their own judgements. It remains for the participant to determine whether the faculty interests or relationships influence the materials presented with regard to exposition or conclusion. When an unapproved use of a FDA approved drug or medical device, or an investigational product not yet FDA approved for any purpose is mentioned, EES requires disclosure to the participants.

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Sources of Funding for Research/Grants - Defense and Veterans Brain Injury Center

Anti-seizure medications, anti-depressants, anxyolytics, and novel antipsychotics for treatment of post-traumatic agitation are discussed. This has not been approved by the FDA.

Sandra Lundgren, PhD, ABPP

The use of neuropsychological test materials is discussed. This is a commercial product or service.

David Jaffe, MS

Use of driving simulators

The potential of driving simulators to assess and improve driving performance after Traumatic Brain Injury is discussed. This has not been approved by the FDA.

Marina Waisman, MD

Sources of Funding for Research/Grants - Defense and Veterans Brain Injury Center

Anticonvulsants, anti-depressants, anxyolytics, and antipsychotics for treatment of post-traumatic agitation are discussed. This has not been approved by the FDA.

Disclosure Statement

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Stimulants after adult traumatic brain injury are discussed. This has not been approved by the FDA.

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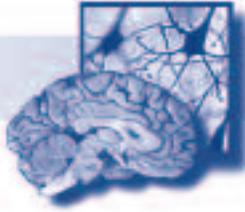
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Did not submit disclosures or FDA issues to EES

Americans with Disabilities Act Policy

The Employee Education System wishes to ensure no individual with a disability is excluded, denied services, segregated, or otherwise treated differently from other individuals participating in this independent study because of the absence of auxiliary aids and services. If you require any special arrangements to fully participate in this independent study, please contact Bob Smith, EdD, MCP, Program Manager, at 205-731-1812 extension 317, or e-mail bob.smith@va.gov.



Introduction

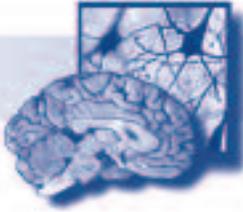
Traumatic brain injury (TBI), sometimes referred to, as the “silent epidemic,” is a condition that may result in lifetime physical, cognitive, emotional, and behavioral impairments. The Centers for Disease Control reports an estimated 5.3 million Americans live with disabilities associated with a TBI.

Providing specialized health care for active duty military and veterans sustaining a brain injury is a high priority of VA. In peacetime, over 7,000 Americans with a diagnosis of TBI are admitted to military and veterans hospitals each year. In times of combat TBI comprises at least 14-20% of surviving casualties, who subsequently consume a disproportionate share of available acute and long-term combat casualty care resources.

The TBI Network of care for active duty military and veteran beneficiaries is a unique partnership between the Department of Defense and the Department of Veterans Affairs with a common purpose — to develop an integrated disease management system and to ensure that military personnel and veterans with brain injury receive optimal care.

Most health care providers do not routinely see patients having a TBI. A major aim of this Veterans Health Initiative (VHI) is to provide a knowledge base of the epidemiology, medical treatment, and rehabilitative interventions associated with various types and severities of injury. Individuals having suffered a brain injury are often reluctant to reveal this when discussing their medical history. Increasing awareness of the varied symptoms that are a result of this condition will help providers to better care for these veterans.

This condition can have a profound impact on the brain injury survivor and his or her family. There are resources available within the VA, other federal, state and local government, and other organizations that represent persons with brain injury. This information has also been included in this VHI to assist with specific areas.



Testimonial - The Happiest of Happy Endings

David S. Knecht

This is an account of a man's recovery from traumatic brain injury.

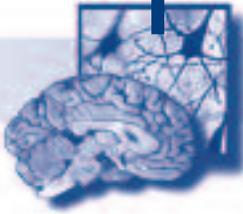
I was a soldier assigned to control and evaluate night live fire exercises for armored forces. One dark evening, a gunner aimed his weapon at my tracked troop carrier, thinking it was his assigned target. He squeezed off eight rounds that battered the armored flank of my command vehicle (the soldier earned an "A" for accuracy, but an "F" for target selection). I ducked for cover as shards of metal and glass lacerated my face. A broken projectile penetrated my skull. Three months and three brain surgeries later I was an inpatient in rehabilitation at a local VA hospital.

For the past eleven years, I have been a "brain injury survivor." Before my brain injury, I was a soldier and a family man. Of these, my role of "family man" supplied the motivation to recover from brain injury. I can never forget the crushing discouragement I felt as I struggled through six months of hospitalization. Much of what motivated me had been stripped away. Though I had been a soldier for a decade or so, I was hurt much too badly ever to return to soldiering. As gratifying as it had been to be the "daddy" (leader) of my home, I could not expect ever to be more than an invalid nursed by Mrs. Knecht and our children. Gloom gripped me as I sat in my wheelchair and wept. I felt doomed to be a "minus" to them, and never again a "plus."

My depression lasted only until I came to see the benefits of my traumatic injury. Were things really so disastrous? Sure, I was certain to be hemiplegic and half-blind. Any hope of completing a career in the Army was ruined. Even so, "no career" could really mean "new career"! What could be more rewarding than being a full-time dad and raising a family to maturity? Cue "theme from Rocky"! Now I had motivation for a comeback! After some months of rehab, I left behind a wheelchair and (later) a cane. Although I am still half-paralyzed and half-blind, I enjoy the privilege of being a full-time home-schooling dad. (I have even fathered four more children since that dark night years ago.) Although the brain injury is not what I would have chosen back then when it happened, I would choose my present life over that past one any day.

Does my story and situation have much in common with those of others? Everyone is unique, and I am certainly fortunate. Draw your own conclusions, but consider this: every man needs a motivation to recover from injury. My

motivation was my home responsibilities, and mine is now a happier home than it has ever been. “Disability” is outweighed by “ability” to devote myself to the things of life that matter most. This is the happiest of happy endings.



Epidemiology and the Nature of Traumatic Brain Injuries

Learning Objectives

1. State why traumatic brain injury is a significant health problem.
2. List three common causes of traumatic brain injury.
3. Describe three tools for classifying severity of brain injury.
4. Describe the main types of physiological changes in the brain resulting from trauma.
5. State common behavioral problems associated with damage to various areas of the brain.
6. Describe the course of cognitive recovery following traumatic brain injury.

Epidemiology of Brain Injury

Introduction

Traumatic brain injury is a leading cause of death and disability in the United States. One to one and a half million Americans incur a traumatic brain injury (TBI) each year (<http://www.cdc.gov/ncipc/factsheets/tbi.htm>), CDC Web site, March 2003; Sosin et al., 1996) and approximately 5.3 million individuals have enduring disabilities as a direct result of a TBI (CDC Web site, March 2003). Direct costs for hospital care, extended care, and other medical care and services were estimated at \$4.5 billion annually in 1985 (Max, MacKenzie, & Rice, 1991), with indirect costs pegged at \$37.8 billion. These figures do not include the physical, emotional, and social costs to the injured person and their family from TBI-related disability. For the year 2001, there were 1,361 veterans who received VA inpatient hospital care for TBI. These figures do not include veterans who may have received medical care for TBI but who did not require hospitalization.

TBI is also a significant health problem for the uniformed services. Ommaya, Ommaya, Dannenberg, and Salazar (1996) examined the 1992 discharge records for all military hospitals and private facilities that received CHAMPUS reimbursement. They found that for active duty military males aged 18-24, the hospitalization rate for TBI was 231 per 100,000. The rate was 150 per 100,000 for active duty military females of the same age range. Based on a military force of 1,597,000 men and 301,000 women in 1992 (Washington Headquarters Services, Directorate for Information Operations and Reports, 2000), approximately 4,141 military personnel would be expected to be hospitalized for TBI annually, and this number would likely increase during wartime. These figures also do not include individuals who received treatment for TBI but who did not require hospitalization.

Causes of TBI

The most common means of sustaining a TBI is through motor vehicle-related incidents. Data from the CDC indicate motor vehicle-related incidents account for 50% of all reported TBIs (see Figure 1). These include all incidents involving motor vehicles, bicycles, pedestrians, and recreational vehicles. Following motor vehicle-related incidents are falls (25%). Firearms account for 10% of all TBIs, with two-thirds of them associated with suicide attempts.

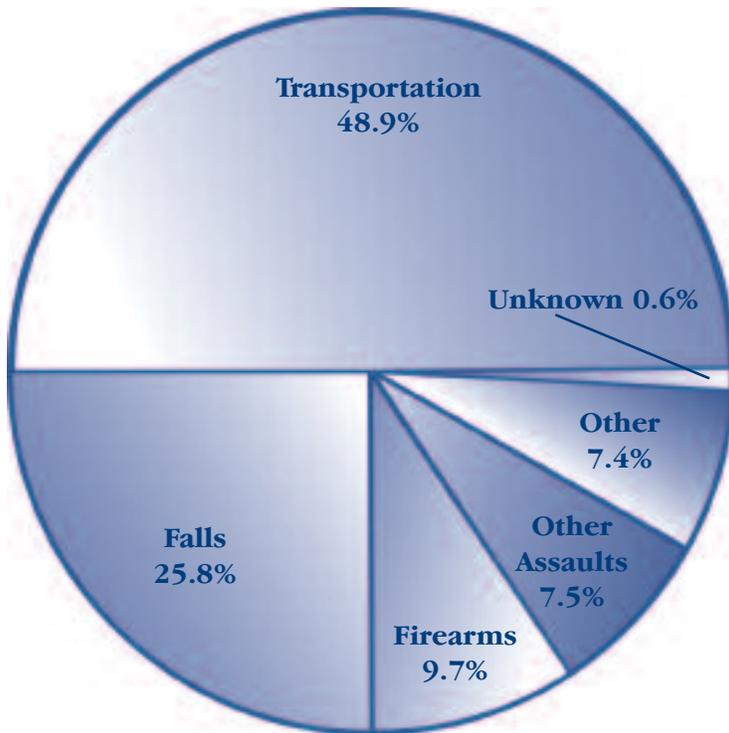


Figure 1

<http://www.cdc.gov/ncipc/images/figure8.gif>

Figure 1: Proportions of traumatic brain injury by cause - Arizona, Colorado, Minnesota, Missouri, New York, Oklahoma, and South Carolina, 1994

Who sustains a TBI?

Given the above causes, it should not be surprising that TBI is more common in certain groups of individuals than others. Males who sustain a TBI outnumber females by at least 2:1 (<http://www.cdc.gov/ncipc/factsheets/tbi.htm>), CDC Web site, March 2003). Individuals between the ages of 15 to 25 and those 75 and older are at highest risk for TBI of all age groups (CDC Web site, March 2003; Whyte & Rosenthal, 1988). As seen in Figure 2 (next page), motor vehicle-related incidents are the most common etiology in younger ages, while falls are the most common reason for TBI in older individuals.

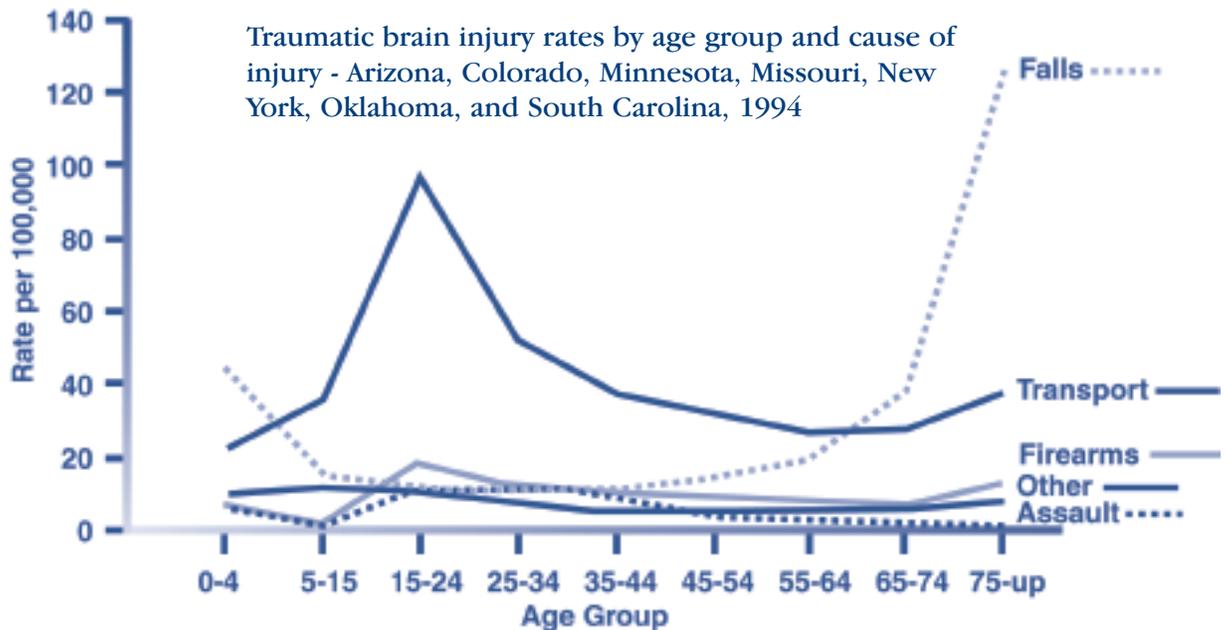


Figure 2

<http://www.cdc.gov/ncipc/images/figure10.gif>

Individuals who abuse substances represent a third group at increased risk for TBI. About 50% of TBI cases are believed to be alcohol related (Drubach et al., 1993; Kolakowsky-Hayner et al., 1999; Wong et al., 1993). Individuals in the age ranges of 26-35 and 56-65 are most likely to be intoxicated at the time of their injury.

Points to Remember

The above data indicate that Primary Care providers in the VA and DoD health care settings are very likely to encounter individuals who have sustained a TBI because:

- The majority of veterans and individuals on active military duty are male
- They fall within the age ranges of greatest risk
- The frequency of substance abuse among veterans and active duty military personnel is high (Bray & Marsden, 2000)
- The risk of falls is increasing with the aging of the veteran population

Severity of Brain Injury

Severity of TBI is an important determinant of outcome. The severity grades, i.e., mild, moderate, and severe, are defined by using one of three indexes: score on the Glasgow Coma Scale (GCS), length of loss of consciousness (LOC), and length of post-traumatic amnesia (PTA).

Glasgow Coma Scale (GCS)

The GCS is a 15-point scale based upon ratings of the patient's best eye opening, motor, and verbal responses (see **Table 1**).

While the GCS is recognized as a reliable measurement tool, it is influenced by factors unrelated to the injury itself such as intoxication and, particularly for mild TBI (MTBI), the length of time between the injury and measurement.

Glasgow Coma Scale	
Motor Responses:	Score
Obeys commands	6
Localizing responses to pain	5
Generalized withdrawal to pain	4
Flexor posturing to pain	3
Extensor posturing to pain	2
No motor response to pain	1
Verbal Responses:	
Oriented	5
Confused conversation	4
Inappropriate speech	3
Incomprehensible speech	2
No speech	1
Eye opening	
Spontaneous eye opening	4
Eye opening to speech	3
Eye opening to pain	2
No eye opening	1
TOTAL (Add above 3 scores)	Range = 3 - 15

Table 1

Loss of Consciousness (LOC)

LOC is the length of time the patient is non-responsive. Obviously, the longer the LOC, the more severe the TBI. Although this may appear to be an easy and objective measure, often patients are unaware of whether or not they have had a period of LOC (Levin, Mattis, Ruff et al., 1987). The injury may have been unwitnessed and the patient may have regained consciousness by the time they are evaluated.

Post-Traumatic Amnesia (PTA)

PTA is the time interval from when the person regains consciousness until he or she is able to form memories for ongoing events (Whyte & Rosenthal, 1988). During PTA, the individual is not fully oriented nor able to remember information after a period of distraction. Determination of the end of PTA in MTBI can be difficult and the clinician may have to rely on self or family report. Retrospectively, PTA can be assessed by asking the patient or family member about the first event which can be remembered following the injury, always distinguishing between what the patient actually remembers and what they have been told (Ruff & Barth, 1999). **Table 2** presents the severity grades and defining criteria of the three indexes.

Severity Grades of TBI		
Mild (Grade 1)	Moderate (Grade 2)	Severe (Grade 3 & 4)
Altered or LOC < 30 min with normal CT &/or MRI	LOC < 6 hours with abnormal CT &/or MRI	LOC > 6 hours with abnormal CT &/or MRI
GCS 13-15	GCS 9-12	GCS < 9
PTA < 24 hours	PTA < 7 days	PTA > 7 days

Table 2

Mild TBI

Approximately 80% of patients who sustain TBIs have had a mild TBI (Alexander, 1995). MTBI is believed to result when injury triggers the pathologic neurochemical cascade, but is insufficient to produce widespread neuronal dysfunction or the axonal disruption that characterizes more severe brain injuries. The majority of these patients make excellent neurobehavioral recovery, but many have persistent and disabling symptoms. The formal definition of MTBI as defined by the American Congress of Rehabilitation Medicine (Kay et al., 1993) is presented in **Table 3**.

Diagnostic Criteria for Mild Traumatic Brain Injury
<ol style="list-style-type: none"> 1. Traumatically induce physiologic disruption of brain function as indicated by at least one of the following: <ol style="list-style-type: none"> A. Any period of loss of consciousness B. Any loss of memory for events immediately before or after the accident C. Any alteration in mental state at the time of the accident D. Focal neurologic deficits that may or may not be transient 2. Severity of the injury does not exceed: <ol style="list-style-type: none"> A. Loss of consciousness of 30 min B. GCS score of 13-15 after 30 min C. Post-traumatic amnesia of 24 hr

Table 3

MTBI remains a somewhat controversial classification because of the typical negative findings on CT or MRI scans and the brief duration of altered consciousness. Issues of MTBI are discussed more fully in Section 2.

Points to Remember

- **Brain injuries can vary from mild to moderate to severe**
- **The majority of all brain injuries are mild**
- **The Glasgow Coma Scale (GCS), length of loss of consciousness (LOC), and length of post-traumatic amnesia (PTA) are common measures of severity of brain injury**
- **A number of non-trauma-related factors (e.g., intoxication) can complicate assessing severity**

Pathophysiology of Injury

Brain injuries also can be classified as focal, diffuse, or mixed depending on the mechanism of injury and the host response. The degree of diffuse injury generally determines the injury grade (mild, moderate, severe). Focal damage, such as contusion or hematoma, can be appreciated by standard neuroimaging studies such as CT or MRI. Widespread disruption of neuronal circuitry or *diffuse axonal injury (DAI)* is difficult to appreciate on standard neuroimaging. It is possible to have both types of injury from a single traumatic incident. Focal lesions are usually the result of direct impact of the brain against the cranium, most often from impact with the frontal and temporal bones or the occipital bone.

Primary Injuries

Diffuse Axonal Injury (DAI)

DAI results from rotational acceleration-deceleration forces. The larger focal lesions that are easily appreciated on neuroimaging are uncommon in MTBI. However, DAI has been observed in pathology studies following mild injuries (Blumbergs, Scott et al., 1994). While DAI can actually be observed in histology specimens, it is also sometimes inferred when neurological symptoms are noted in the absence of abnormal findings or in the presence of small white matter changes on standard neuroimaging (Warden et al., in press).

Historically, the disconnection of axons observed after TBI was thought to result from the direct tearing and disruption of axons at the time of injury, referred to as primary axotomy. Recent work, however, has shown that primary axotomy is rare, even in severe TBI (Maxwell, Povlishock et al., 1997). Rather, axonal disconnection seems to occur several hours after injury as a result of events in the axonal membrane and cytoskeleton (Povlishock, Becker et al., 1983). There may be delay or change in functional deficit over time as a result of this ongoing process. These

pathophysiological changes are observed diffusely throughout the subcortical white matter, corpus callosum, fornix, internal capsules, cerebellum, and brain stem (Adams, Doyle et al., 1989).

Focal Lesions

Focal lesions occur primarily in moderate to severe TBI, but should always be a consideration in any head trauma. It should also be noted that these injuries may not always present acutely coinciding with the actual trauma, but may present days to months following the actual traumatic event. Focal lesions that may be seen in TBI include subdural hematoma (SDH), epidural hematoma (EDH), subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), and cortical contusion. Subdural hematomas result when small bridging veins between the skull and the dura are torn resulting in bleeding into the subdural space. This can occur following motor vehicle accidents, falls, and other types of trauma. These patients may not always be evaluated at the time of the injury or may not be evaluated for symptomatic complaints when they do receive early intervention. Elderly individuals are at particular risk for this type of injury following a fall, and the primary care physician may be the first to see and evaluate these patients for their symptoms. Epidural hematomas result from damage to dural veins and arteries and can result in rapid deterioration and death if not treated promptly. Subarachnoid hemorrhage results from damage to the microvessels in the subarachnoid space and are often associated with cerebral contusion. Intracerebral hemorrhage results from brain laceration and typically occurs in the frontal and temporal areas of the brain. Cortical contusions result from direct trauma to the brain parenchyma from impact with bony prominences of the skull. Typical areas of injury are the frontal, orbital frontal, anterior temporal and lateral temporal areas.

Secondary Injury

Following the initial injury to the brain, mechanisms of secondary injury, including cellular response and neurochemical and metabolic cascades, are set in motion. Contributing to secondary cellular injury are increased excitatory amino acids such as glutamate, neurotoxic free radicals and oxidants, lipases, arachadonic acid, and increased calcium (Graham, 1999). Hypoventilation or elevated intracranial pressure (ICP) can lead to secondary hypoxic brain injury. Secondary ischemic insults can occur in the form of focal cerebral infarcts or diffuse watershed brain injury from circulatory failure. **Table 4** (next page) presents frequent causes and functional correlates of various pathophysiologic changes.

Functional Correlates of Injury Pathophysiology		
Injury Type	Usual Cause	Deficits seen
Focal Cortical Contusion	Ground level fall (GLF) Assault Gunshot wound	Hemiparesis Aphasia Seizures Visuoperceptual
Diffuse Axonal Injury	Motor vehicle accident Non-ground level fall Geriatric GLF	Confused language Amnesia Apraxia Hypoarousal
Hypoxic/Ischemic	Anoxia Cardiac arrest Prolonged elevated ICP	Quadruparesis Spasticity Confusion Amnesia Hypoarousal

Table 4

Points to Remember

- Brain injuries can result in diffuse axonal injuries, focal lesions, or both depending on the mechanism of injury and the host response
- Diffuse axonal injury results from inertial (rotational acceleration-deceleration) forces and is thought to be a result of calcium influx and subsequent cytoskeletal damage
- Focal injuries are typically due to a direct blow to the head and include subdural hematoma, epidural hematoma, subarachnoid hemorrhage, intracerebral hemorrhage, and cortical contusion

Neuroanatomy and Common Deficits Associated with Site of Injury

Accurate prediction of deficits from TBI is difficult due to the high complexity of the brain, the variability of injury caused by any particular trauma, and the fact that injury sites are frequently not shown by standard neuroimaging. However, there are generally accepted functions associated with the major areas of the brain. Damage to these areas is often associated with specific impairments in day-to-day function.

Frontal Lobes

As mentioned previously, the frontal lobes are particularly susceptible to trauma. The anterior portion of the frontal lobes is considered essential to higher cognitive functioning, often called “executive” functions. These are integrative functions and include the ability to focus attention to appropriate stimuli, organize and plan, problem-solve, formulate good decisions, and exhibit appropriate judgment. They also play a role in behavioral control and emotion, and the ability to empathize with others. Damage to the anterior frontal lobes often results in difficulties with planning and carrying out activities that once were accomplished with little effort like grocery shopping, maintaining a job, completing household repairs, etc. There may also be more concrete thinking and decreased mental flexibility. Damage may also result in difficulties staying on task and completing activities. Persons with MTBI may have only vague complaints in these areas, while individuals with moderate to severe TBI may be more impaired. Behavioral change from injury to the frontal lobes may be quite paradoxical, showing either an increase in disinhibition and immaturity or a decrease in energy, motivation, and spontaneity. The posterior aspect of the frontal lobes contains the motor strip. Damage to these areas can result in reduced abilities to carry out motor tasks or actual hemiparesis or plegia often associated with spasticity. Damage anterior to the motor strip near the Sylvian fissure can produce an aphasia (Broca’s aphasia) characterized by nonfluent, agrammatic speech consisting of mostly nouns and verbs.

Temporal Lobes

The temporal lobes, also highly susceptible to injury, are integral to memory function and also play a role in modulating behavior. Damage to the temporal lobes, especially the hippocampal area, can result in difficulty with storing new memories while old memories are readily preserved. The right temporal lobe is associated with visual memory and the left with verbal memory. The left temporal lobe is also associated with language function and lesions may give rise to word-finding and naming difficulties (anomic aphasia). Behavioral problems include increased irritability and aggression.

Parietal Lobes

The parietal lobes are instrumental in processing sensory information. The right parietal lobe is associated with processing visual-spatial information and damage to this area can result in difficulty with getting lost in familiar and unfamiliar surroundings. The left parietal lobe is associated with language function and damage here may impair an individual’s ability to understand the spoken or written word. Again, deficits may vary from mild to severe depending on the severity of the injury.

Occipital Lobes

The occipital lobes are primarily responsible for the reception and processing of visual information. Damage to the occipital lobes results in various forms of visual deficits ranging from visual field cuts to the inability to recognize known objects to blindness.

Cerebellum

The cerebellum has a major role in coordinated movement. Damage to the cerebellum can make walking and the coordinated use of the upper extremities for day-to-day tasks difficult. Nystagmus and tremors can also be seen with cerebellar injury.

Diffuse Axonal Injury

Diffuse axonal injury, which is common in TBI, typically results in a generalized slowing of cognitive processes. Individuals with DAI often complain of a generalized slowness in thinking and difficulty getting things done. There is a reduction in the number of cognitive operations that the brain can engage in at any given time.

Points to Remember

- **There is no typical pattern of deficits associated with TBI because of the complexity and individual variation of the brain and variations in mechanisms of injury and the results of the trauma**
- **Specific impairments in function may be seen in behaviors that are strongly localized to or heavily dependent on particular areas of the brain**
- **Impairments in executive abilities and motor abilities are frequently seen with damage to the frontal lobes**
- **Consolidating new information into memory may be impaired with damage to the temporal lobes, especially damage to the hippocampal area**
- **Difficulties in language comprehension or comprehension of visual stimuli are seen with lesions to the parietal lobes**
- **Damage to the occipital lobes may produce deficits in visual abilities such as cuts in the visual field or cortical blindness**
- **Diffuse axonal injury is associated with slowed thinking and difficulty accomplishing tasks**

Course of Recovery

Recovery is a gradual process that occurs for at least 18-36 months as the brain recovers and regenerates. Recovery is most rapid over the first 6 months during which the majority (80-85%) of recovery occurs. Following diffuse axonal injury, damaged neurons may sprout ancillary axons and reestablish disrupted connections. Other axons that have not sustained total disruption are able to reverse the pathophysiological damage and return to function. There is evidence from animal studies that purposeful activity may stimulate axonal regeneration. Experimentally brain-injured rats placed in an enriched environment with maze-reward systems and other games showed improvement in cognitive function and, on dissection, strong growth of neuronal pathways. This was not observed, however, in rats placed in plain cages (Hamm et al., 1996; Passineau et al., 2001).

Symptoms of MTBI resolve by three months after injury in the majority of individuals (Rutherford et al., 1989; Levin, Mattis et al., 1987). However, for a minority of these individuals, symptoms persist for a more lengthy period of time and sometimes for life (Binder, 1986; Rutherford, 1989; Bohnen & Jolles, 1992). The prevalence of persistent symptoms varies from 7-8% (Binder, 1997) to 10-20% (Alexander, 1995) to 33% (Rimel et al., 1981). A commonly accepted estimate of prevalence is currently 20%. This group of patients will require ongoing medical care to manage these symptoms. There is also evidence that individuals who may appear to have recovered in normal day-to-day function may still reveal impairments during periods of psychological or physiological stress (Alexander, 1995).

The recovery course of cognitive abilities after moderate to severe TBI is highly individualized and depends on the initial distribution and degree of injury, as well as numerous cofactors. Damage to the highly complex brain cell bodies, axons and/or synapses can have far reaching implications for TBI survivors and their families. Poorer outcome has been associated with the following factors: age > 55, poorer premorbid function, lower socioeconomic status, increased length of coma, presence of brainstem abnormalities, lower motor score on the GCS, elevated ICP, prolonged PTA, presence of intracranial mass lesion, and low functional abilities on admission to rehabilitation (Sandel, Bell, & Michaud, 1998). The vast majority of survivors of moderate to severe TBI do not return to their full premorbid cognitive level and have troubling behavioral issues. **Figure 3** illustrates three hypothetical recovery paths of cognitive functioning.

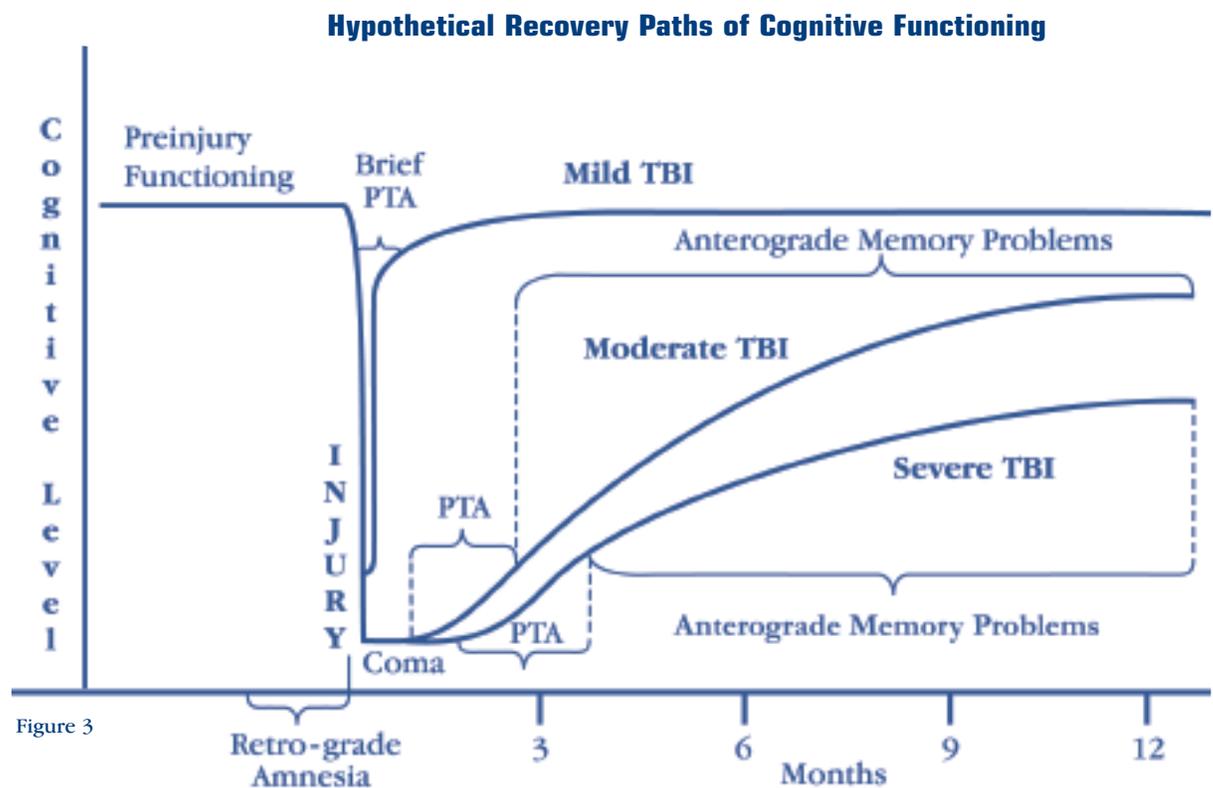


Figure 3

In the acute phase of recovery (initial several months) rehabilitation professionals frequently use the Rancho Los Amigos Level of Cognitive Function Scale (Hagen, Malkmus, Durham, & Bowman, 1979) to characterize the level of functioning and track the course of cognitive recovery over time. It is a descriptive instrument that characterizes the level of cognitive and behavioral deficits after moderate to severe TBI. Ratings range from Level I, indicative of no response (comatose), to Level VIII, indicative of a person whose behavior is purposeful and appropriate, although not necessarily functioning independently or at premorbid levels. **Table 5** presents the full range of Rancho levels and brief descriptions of the characteristics for each level.

Rancho Los Amigos Level of Cognitive Function Scale		
Rancho Los Amigos Level		Description
I	No Response	Unresponsive to sound, light, touch, or pain. The individual appears to be in a deep sleep.
II	Generalized Response	Individual reacts inconsistently in a nonspecific manner to stimulation. May be gross body movements, unintelligible vocalizations, etc. Earliest response is frequently to deep pain. Responses to stimuli often are delayed.
III	Localized Response	Reacts to specific stimuli (e.g., eye blink to strong light, turns toward sound, etc.). Responses are often inconsistent. May inconsistently follow simple, direct commands (e.g., close your eyes, squeeze my fingers, etc.).
IV	Confused - Agitated	Alert and active but has severely limited ability to process information. Disoriented to circumstances and responds primarily to internal stimuli. Behavior is not purposeful or is bizarre and the ability to focus and sustain attention is extremely limited. Does not differentiate among people or things. Verbalizations may not be coherent or may be patently bizarre. Short-term memory and recall are impaired and may confabulate.
V	Confused - Inappropriate	Alert and active and can respond consistently to simple commands. Disoriented to circumstances and requires redirection but is not responding primarily to internal stimuli. Short-term memory and recall are impaired and may confabulate. May be able to perform self-care activities with assistance and supervision.
VI	Confused – Appropriate	Alert and inconsistently oriented to time and place. Follows simple directions consistently and begins to show carry-over of new learning. Recognizes staff and has increased awareness of self, family, and others.

Table 5

Table 5 (cont.)

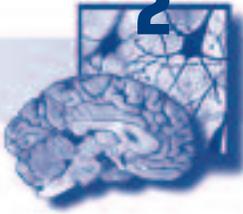
Rancho Los Amigos Level		Description
VII	Automatic – Appropriate	Alert and oriented to person, place, and time but shows a shallow awareness of medical condition. Performs self-care and daily routines with supervision but in a robot-like manner. Performance may deteriorate in unfamiliar circumstances. Shows carry-over of new learning but at a reduced rate. Judgment and problem-solving remain impaired.
VIII	Purposeful - Appropriate	Alert and oriented. Can recall and integrate past and current events. Shows carry-over of new learning and is independent, within physical limitations, at home and in the community. Cognitive abilities may still be lower than premorbid levels.

Adapted from Hagen et al., 1979.

Table 5

Points to Remember

- **Recovery is a gradual process that occurs for at least 18-36 months as the brain recovers and regenerates**
- **Recovery is most rapid and greatest over the first 6 months**
- **Approximately 20% of all individuals who sustain a MTBI will continue to experience prolonged symptoms and require ongoing medical care**
- **Recovery of cognitive abilities after moderate to severe TBI is highly individualized and depends on the initial distribution and degree of injury, as well as numerous other factors**
- **Individuals who have sustained moderate to severe TBI frequently never recover to pre-injury functional levels and may have behavioral difficulties**
- **The Rancho Los Amigos scale is frequently used to track cognitive recovery from moderate to severe TBI**



Concussion Management: Sequelae and Treatment of Mild TBI

Learning Objectives

1. List the levels of severity for concussions.
2. Describe the indications for an initial CT scan of the brain following mild TBI.
3. Describe the typical course of recovery from mild TBI.
4. List the common symptoms of post-concussion syndrome.
5. Describe factors complicating the assessment of post-concussion syndrome.
6. List potential interventions for treatment of post-concussion syndrome.

Introduction

This section will discuss the management of the individual with mild traumatic brain injury (MTBI) or concussion. Management will include both medical interventions in the emergency department or outpatient clinic, as well as educational interventions demonstrated to be effective in improving post-concussive symptoms. This section will be divided into the following sections:

- Acute Concussion Management
- Common Sequelae and Course
- Post-Acute Management of Mild Traumatic Brain Injury
- Factors complicating Assessment
- Appropriate referrals of patients with MTBI

Acute Concussion Management

A high degree of suspicion for MTBI is required in the individual who is being treated for other bodily injuries. With triage focused on urgency of care, health care providers at each level of care must question whether a concomitant brain injury may have occurred. Identification of an individual with MTBI allows for monitoring of return of full function and educating the individual on the natural course of MTBI recovery.

Obtaining a complete history of the injury is the first step in evaluating an individual who has sustained a recent MTBI. Necessary information includes:

Severity of Injury

Presence and length of disturbed consciousness, from a momentary alteration of consciousness only (i.e., having been “dinged” or having one’s “bell rung”) to the length of a period of clear unconsciousness; length of post-traumatic amnesia (PTA; period from injury until continuous memory or “new learning” resumes). A rating of 13-15 on the Glasgow Coma Scale (GCS; see Section 1) is also considered MTBI. The GCS is the most commonly used scale to quantify initial severity of brain injury and based upon the clinical evaluation (see below).

The American Academy of Neurology (AAN, 1997), has published guidelines grading the level of concussion into three categories:

Grade I:

1. Transient confusion
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities on examination resolve in less than 15 minutes

Grade II:

1. Transient confusion
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities on examination last more than 15 minutes

Grade III:

1. Any loss of consciousness, either brief (seconds) or prolonged (minutes)

These categories can be found at:

<http://www.aan.com/professionals/practice/pdfs/gl0013.pdf>

For awake but confused patients seen in the ER or Primary Care Clinic, an accurate determination of possible loss of consciousness (LOC) is enhanced by interview of any bystanders, as well as review of the ambulance report (if available). Evaluation of PTA requires review of available medical records along with the patient and observer interviews, especially in distinguishing a brief period of LOC from PTA. For example, an individual may report that he was unconscious until he arrived at the hospital, yet the ambulance report may say that he was walking around, but confused, at the scene of the accident. The individual may believe that he was unconscious at that time due to lack of recall of the initial events (i.e., in PTA). In cases of MTBI, PTA is often operationally defined as the length of time until continuous memory resumes, or when the individual scores a 75 or above on the Galveston Orientation and Amnesia Test (GOAT; Levin, O’Donnell et al., 1979). When the initial medical contact occurs after the resolution of PTA, the patient is often asked when s/he began to remember events

continuously after the injury. PTA technically extends from when consciousness was regained until continuous memory resumes, but without observer report, the distinction between brief LOC and PTA is difficult to confirm. The length of PTA may diminish over time with events immediately before and after the injury the least likely to resolve fully.

Mechanism and Context of Injury

It is important to determine and document whether someone had a blow to the head (increased likelihood of focal injury) or an acceleration-deceleration injury as in a motor vehicle accident (increased likelihood of diffuse injury). The rare, but potentially devastating, condition of an epidural hematoma may be suspected from a sharp blow to the side of the head, in a patient who is alert initially, but later begins to develop decreased level of consciousness.

Included in the context of the injury are time and circumstances of injury, and whether drugs or alcohol were involved. All these factors may assist in evaluating the severity of the injury, as well as in understanding the lifestyle of the injured person, which will be relevant to recommendations of how treatment may fit into the individual's routine. A tendency toward risk-taking behavior is important to note, as education about decreasing risks for a possible second injury is an important intervention.

Sports concussion guidelines provide recommendations regarding the timing of return to full activity (e.g., AAN, 1997; <http://www.aan.com/professionals/practice/pdfs/gl0013.pdf>). Such guidelines exist to decrease the potential for a second injury during the time period when the individual may be functioning suboptimally from a concussion and thus is more likely to be involved in a subsequent injury. For example, decreased reaction time has been documented to occur up to 4 days after injury in a group of individuals sustaining a Grade I or II concussion (no LOC, symptoms lasting up to 24 hours) (Warden et al., 2001), and up to 35 days following ER care for an uncomplicated MTBI (McAllister et al., 1999).

Assessment of Symptoms

Careful mental status and neurologic exams are indicated in all cases of LOC. Questions regarding recent memory (e.g., Orientation: Do you remember the injury? What was your last memory before the injury?) and the ability to form new memories (e.g., memory for 3 words after 1 minute delay) are sensitive to the effects of MTBI. The Sideline Assessment of Concussion (McCrea, Kelly et al., 1997; also <http://www.aan.com/professionals/practice/pdfs/gl0013.pdf>) is an example of a sports-based, focused mental status exam, which may be performed on site for individuals with MTBI.

Post-concussive symptoms should be recorded, either through a clinical history, or by the use of a standardized instrument such as the Post-concussion Symptom Checklist (Gouvier, Cubic et al., 1992) or the Kennedy-Johnson Post-concussion Scale (Cicerone & Kalmar, 1995). Headache is the most commonly reported symptom reported in MTBI, with

dizziness also frequently reported. Immediate post-concussive symptoms such as nausea, vomiting, and drowsiness are typically short-lived (Rutherford, 1989; Bohnen & Twijnstra, 1992). Other possible symptoms include decreased concentration, difficulty maintaining attention, fatigue, dizziness, irritability, depression, and social withdrawal.

In terms of acute management, the available evidence supports a treatment option (weakest level of evidence) that patients having a negative clinical examination and negative head CT who are greater than 6 hours post-injury may be discharged to home. Patients with responsible and informed caregivers at home may be discharged sooner (Jagoda et al., 2002).

Neuroimaging in MTBI

A head CT is the imaging study of choice in acute brain injury because unlike a skull x-ray, CT will demonstrate acute bleeding, either within the brain tissue, within the ventricles, or involving the dura, i.e., epidural or subdural hematoma. Because of availability, cost, and sensitivity to acute bleeding, CT offers advantages over the brain MRI scan. Cervical x-rays need be considered in all patients receiving trauma to the neck during the MTBI, and a cervical collar should be considered prior to the x-ray being read as normal.

The risk of intracranial pathology identified by CT varies depending on the initial or ER GCS score (13, 14, or 15). The incidence of intracranial lesions increases from 4% with a GCS of 15, 16% with GCS 14, and 28% with GCS 13, demonstrating that not all mild traumatic brain injuries are alike (Culotta, Sementilli et al., 1996).

A recent Evidence Based Guideline (Jagoda et al., 2002) group concluded that CT scanning was not indicated for patients who did not exhibit one or more of the following symptoms: headache, emesis, age > 60 years, drug or alcohol intoxication, short term memory problems, seizure, or visible trauma above the clavicle.

However, identifying intracranial pathology is important, even in non-surgical situations, as patients with a MTBI who have a cerebral contusion experience a 6-month outcome more similar to patients with moderate TBI (Williams, Levin, & Eisenberg, 1990). With this information, patients may be assisted in planning for a more gradual return to activity.

Only relatively large abnormalities may be detected by current clinical imaging techniques. However, the relative absence of structural abnormalities in MTBI does not indicate that cell loss and other damage have not occurred. Functional changes could result from a smaller degree of cell loss due to the widespread connectivity of neurons in the brain, especially shortly after injury before compensatory measures can take effect. Magnetic Resonance Imaging (MRI) has greater sensitivity to the smaller lesions seen in MTBI and may be indicated at follow-up in patients who are experiencing persistent symptoms in the absence of findings on CT scanning (Jenkins, Teasdale et al., 1986; Levin, Amparo et al., 1987;

Eisenberg and Levin, 1989). New and evolving MRI technologies such as Diffusion Tensor Imaging and Magnetization Transfer Imaging also tend to be more sensitive to structural changes following MTBI (Smith, Meaney et al., 1995; McGowan, Yang et al., 2000; Rugg-Gunn, Symms et al., 2001), but are currently being used more for research than clinical purposes.

Points to Remember

- **A high degree of suspicion for MTBI is required in the individual who is being treated for other bodily injuries.**
- **An epidural hematoma should be suspected following a sharp blow to the side of the head, in a patient who is alert initially, but later begins to develop decreased level of consciousness**
- **Although not always necessary, a head CT scan is preferable to an MRI scan following a mild TBI because of its sensitivity to acute bleeding**

Common Sequelae and Course of MTBI

Most individuals who suffer MTBI experience few post-injury problems and make a rapid recovery (i.e., return to normal functioning within hours or days of their injury) and have a complete recovery within three months (Binder, Rohling, & Larrabee, 1997). However, a significant minority continue to report distressing symptoms for months (Alves, Macciocchi, & Barth, 1993; Dikman, McLean, & Temkin, 1986; Powell, Collin, & Sutton, 1996); or years post-injury (Alexander, 1992; Deb, Lyons, & Koutzoukis, 1999; Hartlage, Durant-Wilson, & Patch, 2001). The size of the minority who become chronically symptomatic varies across studies from 7-8% (Binder, 1997), to 10-20% (Alexander, 1995) to approximately one third (Rimel et al., 1981; Vanderploeg et al., 2001).

Both the acute and chronic symptoms involve a constellation of physical, emotional, and cognitive complaints collectively known as post-concussion syndrome (PCS). The symptom cluster includes complaints of poor concentration, memory difficulty, intellectual impairment, irritability, fatigue, headache, depression, anxiety, dizziness, blurred vision, light sensitivity, and sound sensitivity (American Psychiatric Association, 1994; World Health Organization, 1978). This symptom complex typically occurs without demonstrable structural changes to the brain (Eisenberg & Levin, 1989) or neuropsychological dysfunction (Dikman et al., 1986; Levin, Mattis et al., 1987). However, the etiology of persistent PCS symptoms (greater than several months) remains controversial. Assuming medical complications such as subdural or epidural hematomas do not develop, the most commonly documented persistent PCS are noted in the **Table 1** (next page). Although many of these symptoms are common in normal individuals (e.g., difficulty concentrating, irritability, anxiety, memory complaints, headaches), the incidence of these symptoms increases following MTBI. Most individuals who suffer a MTBI will demonstrate only a few of these symptoms that typically resolve over time. Those patients

who do not make a full and rapid recovery typically report more post-concussion symptoms.

Frequency of PCS Symptoms following MTBI and in the General Population			
	MTBI PCS Symptom Frequency	Frequency in the General Population	PCS Increase after MHI
Symptom	Percent of Patients	Percent of People	Increase over base rate
Poor concentration	71%	14%	57%
Irritability	66%	16%	50%
Tired a lot more	64%	13%	51%
Depression	63%	20%	43%
Memory problems	59%	20%	39%
Headaches	59%	13%	46%
Anxiety	58%	24%	34%
Trouble thinking	57%	6%	51%
Dizziness	52%	7%	45%
Blurry or double vision	45%	8%	37%
Sensitivity to bright light	40%	14%	26%

Table 1

Table adapted from: Mittenberg, W., DiGiulio, D. V., Perrin, S., & Bass, A. E. (1992). Symptoms following mild head injury: Expectation as aetiology. *Journal of Neurology, Neurosurgery, and Psychiatry*, 55, 200-204.

Table 2 (next page) lists the formal diagnostic criteria for DSM-IV post-concussion syndrome (PCS).

It is useful to view the symptoms of MTBI as falling four periods across time, as the brain responds in the moments, weeks, and months following injury, and as adjustment and psychosocial factors come into play for those who experience ongoing difficulties.

Acute or Immediate Symptoms

The immediate symptoms following a MTBI are either any loss of consciousness of less than 30 minutes or any period of post-injury disorientation, confusion, or memory problems of less than 24 hours

(Definition of mild traumatic brain injury, (1993). Journal of Head Trauma Rehabilitation, 8, 86-87). Post-injury headaches are also a common symptom. If symptoms are more severe and ongoing, patients may be admitted overnight for observation, often because of concerns about intracranial bleeding, which are more likely in the elderly and those on anticoagulant medication.

DSM-IV Symptom Criteria for Determination of PCS	
1.	History of a head injury with concussion
2.	Following the head injury evidence of attention or memory difficulties on formal testing
3.	Following the head injury presence of three or more of the following symptoms: <ul style="list-style-type: none"> a. Becoming fatigued easily b. Disordered Sleep c. Headache d. Dizziness or vertigo e. Irritability or aggression with little provocation f. Anxiety, depression, or affective lability g. Changes in personality (e.g., social or sexual inappropriateness) h. Apathy or lack of spontaneity
4.	The PCS problems cause a significant disturbance of social or occupational functioning

Table 2

Figure 1 is a schematic representation of the stages of recovery from closed head.

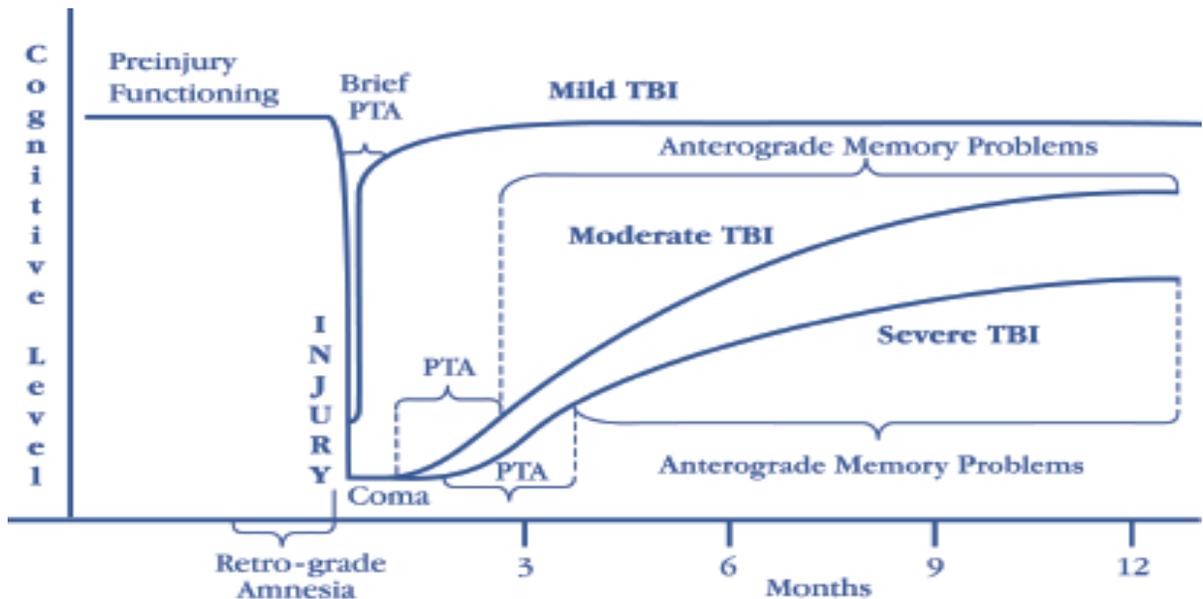


Figure 1

Figure 1 Legend

- Retrograde amnesia refers to loss of memory of events prior to the accident
- Loss of consciousness or coma are interchangeable terms and refer to a period when the patient is non-responsive.
- Post-traumatic amnesia (PTA) refers to the duration of time when the patient is unable to store continuous information (i.e., not fully oriented to time, place, or person with very poor memory for ongoing activities). PTA technically extends from when consciousness was regained until continuous memory resumes, but without observer report, the distinction between brief LOC and PTA is difficult to determine. Therefore, PTA is often operationally defined as the length of time until continuous memory or correct orientation resumes. A patient who has no recall for one hour immediately following the accident would be unable to recall whether or not he/she had an LOC immediately following the accident. This can lead to confusion in the determination of an LOC. One patient may actually deny an LOC simply because he/she has no memory for an extended period of time following the accident. More commonly, a patient may claim that he/she has a loss of consciousness for the entire time during which he/she had a post-traumatic amnesia. Therefore, in MTBI cases it is very difficult to separate PTA from LOC.
- Anterograde memory disturbance refers to a decline in memory function subsequent to the TBI. Objective evidence of residual memory problems several weeks after a mild TBI is very unusual (in fact, if they persist beyond 30 minutes the injury is no longer considered mild). Ongoing memory complaints reported by individuals who sustained a mild TBI usually cannot be verified on formal memory testing. However, a recent study by McAllister et al., (1999) found that despite similar memory performance between MTBI patients and controls, MTBI patient activated more cortical regions than controls on functional MRI suggesting that their brains were functioning less efficiently even 6 months post-injury. Persisting memory problems are more typical of moderate to severe TBI.

Initial Several Weeks

During the week or two following MTBI, the vast majority of patients recover fully without any noticeable symptoms. However, having some symptoms at this juncture is common, and is thought to be related to the neurochemical changes or neuronal degeneration that may occur following diffuse axonal injury. During this period, individuals often have initial cognitive symptoms of slowed information processing speed and difficulty with attention.

Other than initial and ongoing headaches, some patients don't develop many symptoms until days or even weeks after the accident, but they can develop

sooner. Eight out of 10 patients with a mild head injury show at least some symptoms outlined in **Table 1** (page 24) during the first several weeks or months after the accident. These symptoms are part of the normal recovery process and are not signs of permanent brain damage or medical complications. Again, the majority of patients with PCS recover completely in 3 to 6 months.

One to Six Months

People who have suffered a previous head injury are thought to be at greater risk for developing symptoms that last beyond two weeks. At this point, for the minority of patients who continue to exhibit symptoms, the diagnosis of MTBI changes to that of post-concussion syndrome (PCS). If the symptoms continue beyond six months post-injury, the diagnosis changes again to that of chronic or persistent post-concussion syndrome. Many individuals who fit this label continue to suffer symptoms for years. Interestingly, persistent PCS is more common after mild head injuries than after more severe injuries.

Chronic: Years Post-Injury

Only one study to date (Vanderploeg et al., 2001) has examined long-term morbidities in veterans with a MTBI who are not reporting to medical clinics. Such studies are necessary to draw conclusions regarding natural recovery and long-term outcome from MTBI. Four groups were examined, those who:

1. had not been injured in a motor vehicle accident nor had a head injury (n = 3214);
2. had a bodily injury in an accident but did not have a head injury (n = 539);
3. had sustained a head injury with no alteration of consciousness (n = 377); and
4. had a head injury with alteration of consciousness (n = 254).

Analyses controlled for demographic, co-morbid medical, and pre-existing mental health factors in examining outcomes. Over a median post-injury interval of eight years, a MTBI with loss of consciousness doubled the likelihood of a having a current post-concussion syndrome (prevalence of 30% with MTBI). Specific subjective post-concussion symptoms were categorized into neurological/medical, cognitive, and psychological symptoms (**Table 3**, next page).

Specific Subjective Post-Concussion Symptoms

<u>Neurological or Medical</u>	<u>Cognitive</u>	<u>Psychological</u>
Headaches Dizziness/Vertigo Tinnitus Blurred or double vision Light and/or noise sensitivity Nausea or Vomiting Fatigue Sleep Disturbance Physical Weakness	Memory Complaints Concentration Complaints	Irritability Increased Aggression Depression Anxiety

Table 3

A two- to three-fold increase in subjective PCS neurologic symptoms and memory complaints were associated with a MTBI with loss of consciousness. In contrast, PCS psychological symptoms and concentration complaints did not differ across the four groups once the effects of early life psychiatric problems and co-morbid medical conditions were controlled. Although this symptom pattern supports a neurologic etiology for these long-term adverse outcomes, no differences were found among groups on formal neuropsychological evaluation in any area. Thus, although subjective complaints are present, formal diagnostic procedures typically are negative.

In addition to the neurological PCS symptoms, a MTBI with loss of consciousness resulted in:

- A higher frequency of employment problems (Odds Ratio = 1.88)
- Low income (Odds Ratio = 1.91)
- Having an ongoing disability (Odds Ratio = 2.88)
- Being unmarried (Odds Ratio = 2.07)
- Having less than satisfactory social supports (Odds Ratio = 1.58)

Points to Remember

- **Most individuals with MTBI (approximately 70-80%) experience few post-injury problems and make a rapid recovery**
- **In a minority, post-concussion syndrome symptoms may not appear on initial examination, but turn up in one to three weeks following injury**
- **In MTBI, post-concussion syndrome typically occurs without demonstrable structural changes to the brain or measurable neuropsychological dysfunction**
- **There are significant long-term residual neurological symptoms in a small proportion of individuals who sustained a MTBI (about 10-30%), with consequent psychosocial work and relationship problems**

Post-Acute Management of MTBI**Overview**

Treatment for MTBI includes education, a period of rest and observation, and treatment of persistent or disabling symptoms (e.g., headache). A combination of “validating” the patient’s symptoms, i.e., that post-concussive symptoms derive from the physical injury, with expectancy of resolution of these symptoms over time, are mainstays to management of MTBI (Salazar & Warden, 1999).

The data from sports concussion studies demonstrating that impaired performance follows concussion and possibly increased risk for second injuries supports the indication for a period of rest (Macciocchi, Barth et al., 1996; Warden, Bleiberg et al., 2001). Additionally, when an individual experiences two MTBIs within a brief period of time, s/he is at risk for second impact syndrome, a rare but often deadly condition involving cerebral edema and bleeding. Determining the necessary period of rest is currently an area of active research.

As described above, in general, post-acute management of MTBI consists of closely evaluating individuals for signs of evolving injury or resolution of symptoms following the injury (e.g., headache, dizziness, vomiting). If further medical or surgical care is not indicated, individuals will be observed for a period of time and sent home, with educational materials, a period of rest and observation, and treatment of significant symptoms. Depending on the speed of recovery, recommendations regarding return to partial or full work are tailored to the individual.

Pharmacological Interventions for Post-concussion Symptoms

Pharmacologic and non-pharmacologic interventions are often used to treat specific symptoms following MTBI, especially when those symptoms, e.g., headache and sleep disorder, are disabling, or not improving. Post-traumatic headache may be treated with NSAIDs, Midrin, and tryptans. Individuals with headache and emotional dysregulation may benefit from valproate

acid. Selective Serotonin Reuptake Inhibitors (SSRIs; e.g., sertraline, paroxetine, etc.) may improve depression and irritability following brain injury (Fann, Uomoto, & Katon, 2001). A post-hoc analysis in a study of sertraline for depression after TBI revealed that sertraline decreased many post-concussion symptoms in the sample of depressed patients (Fann, Uomoto, & Katon, 2001). Although most studies target a specific symptom, Piracetam was effective for the spectrum of PCS in a small randomized controlled trial (Hakkarainen & Hakamies, 1978).

Non-Pharmacological Interventions

A crucial and often omitted component of MTBI management is the provision of education to individuals with TBI regarding fatigue, irritability, and mood lability that may occur during MTBI recovery. Many patients become distressed after a concussion because they don't realize that the symptoms that they are experiencing are related to the MTBI. Individuals may think they are "going crazy" due to mood lability, mental slowness, and memory lapses that they did not realize were part of the recovery process from their head injury.

A standardized post-concussion program was developed by Mittenberg (1996) based on the observation that PCS symptoms may be maintained at least in part by psychological mechanisms (Levin et al., 1987; Luis, Vanderploeg et al., 2003; Rutherford, 1989).

- Patients received a 10-page manual, *Recovering From Head Injury: A Guide for Patients* (Mittenberg, Zielinski, & Fichera, 1993). The manual is intended to support the reattribution of symptoms to selective attention, normal transient responses to stress, and anxiety arousing or depressive self-statements. An adaptation of this manual is available in **Appendix A** to this section.
- Met with a therapist who reviewed the nature and incidence of expected symptoms, and the cognitive-behavioral model of symptom maintenance and treatment (Mittenberg, DiGiulio, Perrin, & Bass, 1992). Base rate data for the symptoms was provided:
 - techniques for reducing symptoms, instructions for stopping bothersome thoughts, replacement of negatively biased thoughts, and the scheduling of reinforcing activities
 - instructions for gradual resumption of premorbid activities
 - interventions such as relaxation techniques, consistent diurnal routine, and moderation in exercise, also may be helpful.

Following several weeks of treatment, the treatment group experienced significantly lower symptom duration, significantly fewer symptoms and significantly lower average symptom severity levels at 6 months follow up.

Points to Remember

- **Pharmacologic interventions can be used to treat specific symptoms following MTBI such as headaches, depression, irritability, or emotional dysregulation**
- **Psychoeducational intervention, combined with support and cognitive-behavioral interventions can significantly reduce the extent of post-concussive symptoms**

Factors Complicating Assessment**Misdiagnosis or Missed Diagnosis**

In MTBI, evidence of minor cerebral injury is typically absent from the standard neurological examination, CT scans, and standard EEGs. **But . . . absence of evidence is not proof of absence.**

On the other hand, the presence of post-MTBI neurological symptoms also is not clear evidence of brain trauma. Post-MTBI neurologic symptoms can include headaches, vertigo, and dizziness, but patients with whiplash neck injuries or vestibular injuries can have similar symptoms, without any brain dysfunction. Although many of the neurological symptoms following TBI can be associated with structural brain injury, the association does not prove causation.

The same holds true for psychological symptoms such as anxiety, depression, lability, irritability, and aggression, which can be directly related to TBI. However, these are common complaints in healthy individuals, and increase under stressful situations. Also, other medical patients have an increase in anxiety or depression. Thus, whether the symptoms following a MTBI are caused by the injury, a consequence of adjustment to the accident, secondary to dealing with the medical-legal environment following many minor head, or related to a combination of these factors injuries remains unresolved.

In most first-time MTBIs, the immediate symptoms will often resolve within three months. However, a subset of individuals (15-30%) experience continued post-traumatic symptoms. When these are undiagnosed, misdiagnosed, or ignored, appropriate treatment, support, and education is not provided. Individuals with repeated MTBI also have an increased potential for persistent symptoms. Without appropriate interventions, these individuals are more likely to experience:

- Functional difficulties when trying to return to previous living patterns
- Depression and anxiety, which has an impact on the person's capacity to function
- A tendency to isolate and significantly limit themselves to the comfort of familiar surroundings and routines

- Activity avoidance
- Estrangement from his/her spouse, children, family, and friends — tempers flair, fears build, and the family structure may gradually deteriorate
- An increased frequency of anger at “the system.” This includes physicians and insurance personnel who are not knowledgeable and, therefore, are not providing education or guidance as to appropriate assessment and beneficial treatment
- Suicidal ideations and attempts
- Problems with the law
- A tendency for re-injury

Self-expectations and iatrogenic factors

Some brain injury researchers suggest that many of the symptoms following MTBI are the result of psychological mechanisms such as expectations following a MHI (Mittenberg, DiGiulio, Perrin, & Bass, 1992), poor coping styles (Bohnen et al., 1992; Marsh & Smith, 1995), or emotional reactions to an adverse event. For example, Mittenberg and colleagues (1992) found that implicit beliefs regarding a set of symptoms anticipated following a head injury account for attribution of the normal occurrence of these symptoms to the MTBI. In contrast, athletes’ implicit beliefs regarding symptoms differed such that they under-estimated the normal base rate occurrence of PCS symptoms and attributed any PCS symptoms they experienced following sports-related concussions to a normally occurring incidence.

This research suggests that the information provided by medical personnel to individuals who experienced a MTBI can either amplify and increase their symptomology and distress (**iatrogenic factors**), or can minimize and normalize their symptoms. Mittenberg and colleagues demonstrated minimization of symptoms through in a series of intervention studies. Individuals who sustained a MTBI who were provided with basic psychoeducational information about MTBI symptoms and their typical course of resolution, together with psychological support and stress management interventions, had a significant decrease in their symptoms and complaints compared to those without such interventions. At the same time, attribution and iatrogenic factors can’t be the entire story, because some individuals who receive early psychoeducational interventions continue to experience symptoms.

Psychological factors

Individuals with poor psychological coping or increased psychological distress have a higher rate of prolonged post-concussion symptoms compared to those with uncomplicated recoveries (Karzmark, Hall, & Englander, 1995). Other research suggests that persistent PCS reflects, in part, anxiety regarding the experience of an adverse event seen as life-threatening (i.e., post-traumatic stress response; DiGallo, Barton, & Parry-Jones, 1997; Bryant & Harvey, 1999).

Differential Diagnosis

In those individuals reporting long-term PCS symptoms following MTBI, their clinical presentation may be very similar to related disorders, including post-traumatic stress disorder, major depression, disability seeking behavior, or malingering. For instance, individuals may report sleep difficulties, memory problems, irritability, and anxiety that fit any of these diagnoses.

Mittenberg and Strauman (2000) suggest the following considerations in differential diagnosis:

- **Post-concussion Syndrome versus Post-Traumatic Stress Disorder.** PCS is not associated with persistent re-experiencing of the accident or numbing of general responsiveness, whereas PTSD is. In contrast, PTSD is not characterized by headaches, dizziness, generalized memory problems, or subjective intellectual impairment, while PCS is.
- **Post-concussion Syndrome versus Major Depression.** PCS is not associated with changes in appetite or weight, psychomotor agitation or retardation, suicidal ideation, or a history of depressive disorder.
- **Post-concussion Syndrome versus Disability Seeking or Overt Malingering.** While a malingerer may convincingly report many symptoms of PCS, level and pattern of performance on formal neuropsychological testing can be used to differentiate between the two presentations, at least in cases where the medical record clearly indicates that the head injury was mild. However, it is certainly possible that individuals with legitimate post-injury symptoms may also amplify or exaggerate their problems. These cases are the most difficult to tease apart contributing etiologies, although a careful history, collateral interviews, and formal neuropsychological assessment are often useful.

Points to Remember

- **Correct diagnosis of postconcussion syndrome is complicated by its overlapping symptom pattern with other conditions**
- **Referral to a neuropsychologist for careful evaluation may be helpful in difficult diagnostic cases**

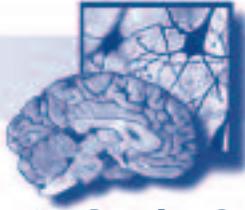
Appropriate referrals of patients with MTBI

Although a thorough physical examination and history are the initial elements of a post-concussion clinical workup, a variety of other tools are available to clarify examination findings. Individuals who continue to describe difficulties with concentration, attention, and memory may be referred for more comprehensive evaluation including cognitive assessment by neuropsychological testing. Magnetic Resonance Imaging (MRI) may be indicated at follow-up in patients who are experiencing persistent

symptoms in the absence of findings on CT scanning (Jenkins, Teasdale et al., 1986; Levin, Amparo et al., 1987; Eisenberg & Levin 1989). Clinically-directed appropriate referrals for additional assessment may include consults to the following disciplines:

Audiologist	Case Manager
Kinesiotherapist	Neurologist
Neuro-ophthalmologist	Neuropsychologist (psychologist)
Occupational therapist	Physiatrist
Physical therapist	Psychiatrist
Recreation Therapist	Social Worker (counselor)
Speech and Language Pathologist	Vocational Rehabilitation Counselor

A more detailed description about each of these disciplines is provided in Section 2 Appendix B.



Section 2 Appendix A

Recovering from Head Injury: A Guide for Patients

What Happens in a Head Injury?

A blow to the head can occur in a motor vehicle accident, a fall, when the skull is struck by a blunt or heavy object, or in other ways. In most cases, there are no lasting symptoms or ill effects from an injury to the head. This is because the brain is surrounded by shock absorbing liquid and covered by the skull. Often these are enough to protect the brain from any damage.

Sometimes the force of impact is more severe. This can cause the skull to break or fracture. When the skull fractures, this absorbs some of the force of the blow and protects the brain. This is the same way that a crash helmet works.

When the head is hit, the brain may be shaken around inside the skull. This can sometimes cause the brain to be bruised if it hits the inside of the skull hard enough. Like a black and blue mark on your arm or leg, this will recover with time. If there are many bruises on the brain, there will be some swelling that can take a while longer to return to normal.

The brain is made of many thousands of long, thin nerve fibers. Some of these nerves can snap or break if a blow to the head is severe enough. Although these nerves cannot be seen without a microscope, we know that they can recover because many patients recover completely in time.

Like any other part of the body, the brain has blood vessels in it. If a head injury is very serious, some of these blood vessels can tear and bleed. This happens soon after the injury. The bleeding often stops on its own and the blood vessels heal like any cut.

Bruises, swelling, snapped nerves and broken blood vessels are the causes of symptoms after a head injury. Your doctors have examined you for any signs of injury to the brain and prescribed treatment if you need it. Most people who suffer a head injury recover completely in time because the damage is minor and heals.

How Serious Was the Head Injury?

One way to tell if a head injury is serious is the amount of time the patient is unconscious afterwards. If you weren't knocked out at all or if you were unconscious for less than an hour, then the injury was most likely minor or mild. Although you may have some symptoms, there was probably little injury to the brain and complete recovery is expected. Most people who have a head injury fall into this category.

The longer you were unconscious, the longer recovery usually takes. If you were knocked out for more than an hour but less than a day, your injuries were most likely moderate. Return to normal will probably take a while.

Patients who are unconscious for more than a day, have suffered a severe injury. Although many patients make a good recovery even after a severe head trauma, symptoms can often last for some time. Treatment at a rehabilitation hospital is usually recommended and can help recovery.

How Long Will the Symptoms Last?

You have probably gotten a lot better over the last few days. The most rapid recovery occurs in the first 6 months after head injury, and most patients will be back to normal by 3 months.

If you still have some symptoms after 6 months, these will most likely disappear altogether or be greatly improved within a year after the injury. If you suffered a severe injury, recovery can take as much as two years. During the second year, improvements will be more gradual.

Not everyone recovers at the same rate. People who are under 40 recover faster and have fewer symptoms during the time they are recovering. If you are over 40, you won't get better as quickly and you may have more symptoms at first. Patients who are older or who have been hospitalized for head injuries before should expect full recovery to take 6 to 12 months even after a mild head injury.

Most doctors who treat head injuries agree that recovery is faster when the patient gets enough rest during the weeks after they leave the hospital. Work, exercise, social activities, and family responsibilities should be started gradually, not all at once.

What Symptoms Can I Expect?

The most common symptoms after a head injury are known as the post-concussion syndrome. Eight out of 10 patients with a mild head injury show signs of the syndrome during the first 3 months after the accident. These symptoms are part of the normal recovery process and are not signs of brain damage or medical complications. Like the itch of healing stitches, these symptoms are expected as you get better. They are not a cause for concern or worry.

Post-concussion syndrome is more common after mild head injury. The symptoms are less likely to trouble a patient with severe head injury. If you have these symptoms, this is a sign that your injuries were probably mild or minor. The majority of patients with post-concussion syndrome recover completely in 3 to 6 months. If you are older than 40, it may take a bit longer to return to normal.

Most patients don't develop the symptoms until days or even weeks after the accident, but the syndrome can begin sooner. Either way, the symptoms often disappear without any special treatment.

A list of the symptoms that you can expect is shown in **Table 1**, along with the percent of head injured patients who experience each symptom at some point in their recovery.

Symptoms of Post-concussion Syndrome	
Symptom	Percent of Patients
Poor concentration71%
Irritability66%
Tired a lot more64%
Depression63%
Memory problems59%
Headaches59%
Anxiety58%
Trouble thinking57%
Dizziness52%
Blurry or double vision45%
Sensitivity to bright light40%

Table 1

What Can I Do About the Symptoms?

Post-concussion syndrome is a normal part of recovery. Most patients will be back to normal by 3 months without any special treatment. The symptoms are not a sign of relapse or brain damage. The syndrome is expected even after minor head injury. Few patients will experience all of the symptoms, but even one or two of the symptoms can be unpleasant.

Some patients find that at first, post-concussion syndrome makes it hard to work, get along at home, or relax. The best way to deal with this is to resume activities and responsibilities gradually, a little at a time. The time

you spend at work, getting together socially, with your family, or exercising is determined by what you are comfortable with. You should pace yourself, and be sure to get all the rest you need. If your symptoms get worse, or if you notice new post-concussion symptoms, this is a sign that you are pushing yourself too hard.

Ignoring your symptoms and trying to “tough it out” often make the symptoms worse. Symptoms are your body’s way of giving you information. A broken bone or a torn muscle hurts so that you won’t use it and it has time to heal. Post-concussion syndrome is your brain’s way of telling you that you need to rest it. Most doctors who treat head injuries agree that recovery is faster when the patient gets enough rest and resumes responsibilities gradually.

Thinking and worrying about your symptoms can make them seem worse. This is partly because paying attention to a feeling seems to magnify or increase it. If you pay attention to your heartbeat or breathing for a minute or two, you will see that the sensations seem to become more noticeable. Concentrating on the symptoms of post-concussion syndrome will also make them more noticeable.

It is important to remember that the symptoms are a normal part of recovery and will go away on their own. Of course, we all have some of these symptoms once in a while anyway. After a head injury it can be easy to forget that we were sometimes irritable, tired, had headaches, couldn’t concentrate, or forgot things even before the accident. Try to deal with these things the same way you did before.

Some of the symptoms you notice may actually have nothing to do with your head injury. The symptoms of post-concussion syndrome are pretty much the same as the symptoms of ordinary day-to-day stress. A list of the symptoms is shown in **Table 2** (next page), along with the percent of people who experience each symptom even though they didn’t have a head injury.

The reason that the symptoms of post-concussion syndrome are so much like the normal signs of daily stress is that one main cause of these symptoms is exactly the same: everyday stress. Of course hitting your head also has a lot to do with it. But having a head injury adds more stress to your life, not just bumps and bruises to your head.

The accident itself, being in the hospital, and going back to work or school are all things that add stress to most patients’ lives. Bills can pile up, time is lost, and there may be injuries to other parts of your body. And just like a pulled muscle or a bruised leg, your brain takes some time to recover. You can have some trouble with work or school at first, and this is stressful also even though it is normal. Trying to do your regular work right after a head injury is something like trying to play baseball or swim with a pulled muscle. You can’t see it, it isn’t really serious, but it takes some time to get better.

Symptoms of Everyday Stress	
Symptom	Percent of Patients
Poor concentration14%
Irritability16%
Tired a lot more13%
Depression20%
Memory problems20%
Headaches13%
Anxiety24%
Trouble thinking6%
Dizziness7%
Blurry or double vision8%
Sensitivity to bright light14%

Table 2

Another main cause of stress after a head injury is worry about your symptoms. Scientific studies by neurosurgeons and neuropsychologists in New Zealand show that patients who get an information booklet like this one recover faster and feel better during recovery than patients who don't know what to expect. That's why we gave you this booklet! Doctors in the U.S. who treat head injuries agree that the single most important factor in recovery is that you know what to expect and what to do about the symptoms.

Of course, talking to a doctor about your symptoms is also important. Your doctor can prescribe medication that can help you if you need it, or you can talk to the person who gave you this booklet.

More about the Specific Symptoms

Poor Concentration

The main cause of poor concentration is tiredness. When it becomes difficult to concentrate on what you are doing, take a break and relax. Between 15 to 30 minutes should be enough. If you still continue to have problems, your workday, class schedule, or daily routine should be temporarily shortened. Trying to "stick to it" won't help, and usually makes things worse.

Reducing distractions can help. Turn down the radio or try to work where it's quiet. Don't try to do too many things at once. Writing while you talk on the phone or taking notes as you listen to someone talk are examples of doing two things at the same time. It may be difficult to concentrate on more than one thing at first. You will be able to concentrate better when you have had enough rest.

Irritability

One of the most frequent causes of irritability is fatigue. People lose their tempers more easily when they are tired or over worked. Adjust your schedule and get more rest if you notice yourself becoming irritable.

Everyone gets angry from time to time, often with good reason. Being irritable only becomes a problem when it interferes with your ability to get along with people from day to day. If you find yourself getting into arguments that cause trouble at home or at work, try to change the way you think about things. Thoughts often make us angrier than what actually happened. You can see this for yourself by imagining an irritating situation and why it would make you angry.

There is usually a reason why irritating things happen. When something makes you angry, ask yourself what caused it. Family, friends, or co-workers can do things that bother us at times. Try to think of why they did whatever it was that irritated you. What would they say the reason was? Thinking about what caused a problem is the first step to solving it.

Problems can usually be solved better if you stay calm and explain your point of view. The steps you need to take to solve a problem will be the same when you are calm as they would be if you were irritated. Try to remind yourself of this when you find yourself becoming irritable.

You can usually come up with several ways to solve a problem. Try to think of at least 5 different ways, and then decide on which is best. Just realizing that there are several things you can do to solve a problem will make it a lot less irritating.

Fatigue

It is normal to be more tired after a head injury. The only sensible treatment for being tired is rest. Avoid wearing yourself out. Gradually increase your activity level. Most patients have more energy in the morning than later in the day. You may benefit from scheduled test breaks or daytime naps. If your symptoms get worse, this means that you are pushing yourself too hard.

Depression

People become depressed when unpleasant things happen to them, and a head injury is unpleasant. We feel good when good things happen to us. An effective way to treat depression is to make sure that good things happen. One way to do this is to plan to do something enjoyable for yourself every day. Make your plan specific, and then be sure to stick to it. Decide on something you like and exactly when you're going to do it. That way you

can look forward to it. Anticipating and doing enjoyable things each day will improve your mood.

Thoughts can make us depressed. Thinking that things are bad or terrible will do it. Bad situations are often not as terrible as they may seem at first. Think back to an unpleasant moment in your own life and you will see that this is so.

Chances are that if you are depressed, you are telling yourself things that are depressing. Thinking that the situation is terrible, that there is no end to it in sight, that you aren't able to do anything about it, and that it is your fault are all depressing things to tell yourself. Thinking this way can become a habit if you do it enough.

Usually, when people tell themselves unpleasant things all the time it is out of habit, not because those things are really true. If you find yourself thinking depressing thoughts, stop. Simply stopping a depressing thought can make you feel better. See if what you are telling yourself is really true.

Memory Problems

Memory difficulties have several causes. The part of our brain that stores memories is called the temporal lobe. This is the part of the brain that is most likely to be bruised in a head injury. Some memory difficulties can be caused by the bruises, which is why you may not remember the accident very well. Like a black and blue mark on your arm or leg, these bruises will recover with time. Your memory will most likely improve as this happens.

If you can remember the accident, chances are that your brain was not bruised. Most of the memory problems patients notice after a head injury are not caused by bruising. They usually come from poor concentration and being tired.

For you to remember something, you have to pay attention to it first. If you don't concentrate long enough the information is never stored in your memory. Concentration problems are a normal part of recovering from a head injury and some memory trouble is a normal side effect of this.

You will probably be able to concentrate and remember better, when you get enough rest. Memory problems can be a sign that you are pushing yourself too hard. Writing things down or using a pocket tape recorder are other excellent ways of coping with temporary memory difficulties. They will help recovery and not slow it down.

Of course, nobody's memory is perfect anyway. After a head injury, it can be easy to forget that we sometimes had trouble remembering things even before the accident. Some of the symptoms you notice may actually have nothing to do with your head injury. A list of common memory "problems" is shown in **Table 3** (next page), along with the percent of people who experience each "symptom" even though they didn't have a head injury.

Things We Normally Forget	
Symptom	Percent of Patients
Forgets telephone numbers58%
Forgets people’s names48%
Forgets where car was parked32%
Loses car keys31%
Forgets groceries28%
Forgets why they entered a room27%
Forgets directions24%
Forgets appointment dates20%
Forgets store locations20%
Loses items around the house17%
Loses wallet or pocketbook17%
Forgets content of daily conversations17%

Table 3

Worrying about remembering things that you would normally forget can make your memory seem worse to you. If you can remember your memory problems, you probably don’t have much of a memory problem! People with serious memory difficulties are usually not upset by their symptoms. They don’t remember that they have any memory trouble.

If you are concerned about your memory, have it tested. Your doctor can send you for these tests if you need them, or you can also ask the person who gave you this booklet.

Headaches

Headaches are part of the normal recovery process, but that doesn’t make them any less bothersome. Headaches are another cause of irritability and concentration problems after a head injury. This guide cannot replace the medical advice that you should get if you are bothered by headaches. Headaches can have many causes, and your doctor will want to diagnose the problem and prescribe medication that can help if you need it.

One of the most common causes of headaches after a head injury is stress or tension. This is usually the cause when the headaches start for the first time several weeks after the injury. These headaches mean that you are trying to do too much. They will probably disappear if you take a break and relax. Your workday, class schedule, or daily routine should be temporarily shortened if you continue to have headaches.

Stress or worry cause tension headaches by increasing muscle tension in your neck or forehead. These muscles become tense and can stay tight without you realizing it, out of habit. They can become even tighter once a headache starts, because muscles automatically tense in reaction to pain. This muscle tension makes the headaches worse.

Anxiety

Worry about symptoms and problems at work are the main cause of anxiety for most patients. Anxiety should not be a problem for you if you understand that your symptoms are a normal part of recovery, get enough rest, and gradually increase your responsibilities at work.

If you are anxious, chances are that you are telling yourself things that are making you that way. Usually, when people worry all the time it is out of habit, not because the things that they are telling themselves are really true. The steps you need to take to solve a problem will be the same when you are calm as they would be if you were anxious. If you find yourself thinking anxious thoughts, stop. Simply stopping an anxious thought can make you feel better. See if what you are telling yourself is really true.

Trouble Thinking

This problem is usually a side effect of other symptoms. Concentration problems, being tired, headaches, and anxiety can all make it hard to think clearly. Like these other symptoms, trouble thinking is probably a sign that you are doing too much too soon.

Dizziness, Visual Difficulties, and Light Sensitivity

Dizziness and visual difficulties should be checked by your doctor. These symptoms usually go away by themselves in 3 to 6 months or less in most patients. If you find these symptoms troublesome, your doctor may want to prescribe medication for motion sickness or eyeglasses. Some motion sickness medications are very effective for dizziness, but can make you drowsy or reduce your attention span as side effects.

You may notice some increased sensitivity to bright light or loud noise, particularly if you have headaches. Some increased sensitivity is normal after a head injury. But scientific studies by neurosurgeons and neuropsychologists in New Zealand show that someone's actual sensitivity to light and noise has nothing to do with how much light and noise bother them. Paying attention to these symptoms makes them seem worse, because paying attention to a feeling seems to magnify or increase it. The less you think and worry about your symptoms, the faster they will usually go away.

Summary

The most common symptoms after a head injury are known as the post-concussion syndrome. These symptoms are part of the normal recovery process and are not signs of brain damage or medical complications. They are not a cause for concern or worry.

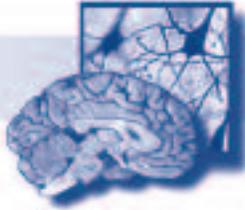
Post-concussion syndrome is more common after mild head injury. Few patients will experience all of the symptoms. The symptoms may not develop until days or even weeks after the accident. Most patients will be back to normal in 3 months without any special treatment.

Most doctors who treat head injuries agree that recovery is faster when the patient gets enough rest and resumes responsibilities gradually. If your symptoms get worse, or if you notice new post-concussion symptoms, this is probably a sign that you are under too much stress. Your workday, class schedule, or daily routine should be determined by your own comfort level.

About This Guide

This guide was created by The Defense and Veterans Brain Injury Center at Wilford Hall Medical Center, Lackland AFB, Texas. The information presented here is based on published scientific research and clinical studies.

Mittenberg, Zielinski, & Fichera, (1993). *Psychotherapy in Private Practice*, 12, 37-52.



Section 2 Appendix B

Clinically - Directed Referral Descriptions

Audiologist evaluates hearing deficits and defines the type of hearing loss. Hearing changes after MTBI may include tinnitus or loss of acuity especially in noisy environments. Hearing aides may or may not be prescribed, depending upon the nature and severity of the problem.

Case Manager provides effective coordination of patient care and appropriate utilization of resources. Whenever possible, one may consider bringing in a case manager who specializes in brain injury, to assist in coordinating medical care and keeping open lines of communication between the various specialists and the patient and family. The case manager is an advocate for, and an active participant in, the treatment process.

Kinesiotherapist can recommend a cardiovascular conditioning program that promotes wellness and reduces the risk of injury or further disability. Fitness can have a positive impact upon the person's mental and physical stamina, reduce pain, and elevate his/her feeling of well-being. The physical conditioning program should be initiated in the health care facility and gradually transferred to a community gym as the person becomes more independent.

Neurologist assesses and treats neurological sequelae, with emphasis on physical, such as movement disorders, seizures, and pain, as well as neurobehavioral sequelae, such as mood problems and memory complaints. The initial primary goal of the neurological evaluation is to rule out the presence of conditions requiring neurosurgical attention (hematomas, skull fractures, elevated intracranial pressure, etc.) and to consider differential diagnosis of MTBI and other neurogenic disorders with similar symptoms.

Neuro-ophthalmologist may address double vision, blurry vision, and/or other visual deficits following brain injury. Deficits in the visual system are often overlooked in the MTBI patient. A common visual deficit found after MTBI is convergence insufficiency, which is often described by the person as "blurry" vision. The neuro-ophthalmology evaluation should rule out potential eye damage involving the cornea, retina, vitreous fluids, as well as occipital lobe (visual cortex) and optic nerve functioning. Therapeutic intervention may involve prism glasses and/or eye exercises.

Neuropsychologist (psychologist) is the key player in diagnosing cognitive impairments and may also address emotional and behavioral

sequelae. The neuropsychological assessment evaluates the areas of intellectual functioning: attention and concentration; problem solving and judgment; memory and learning; flexibility of thought and speed of information processing. Evaluations of disorders in these areas are provided to help patients and families understand the nature and severity of deficits and to assist other team members when planning patient treatment programs. Treatment services provided by neuropsychologist (psychologist) are designed to help patients achieve maximum benefit from the rehabilitation program and to help them manage adjustment related problems. Counseling may be offered to patients and family members who wish to know more about brain injury and who may be having difficulty coping with family and/or work related stress.

Occupational therapist is the function expert who works with the brain-injured person to improve everyday function in daily routines. A thorough occupational therapy evaluation can provide a window into the ways that concussion impacts an individual's daily life. The occupational therapist will assess the patient's skills, which include visual, cognitive, and perceptual abilities to perform tasks in complex and multi-stimuli environments. Treatment goal is to enable patients to best manage their daily tasks, including self-care (feeding and dressing) and managing tasks in the community (shopping, driving, school and work activities). To be able to do these tasks, patients may need to use special techniques, modify their physical environment, or use equipment ranging from simple memory aids to more advanced technology such as computers and environmental controls.

Physiatrist is the rehabilitation specialist who treats physical, cognitive, and behavioral sequelae and provides leadership for the rehabilitation therapies. The physiatrist coordinates treatment to maximize the level of function and is responsible for medical evaluations and plans of care most suitable for the individual and his/her family. Physiatrists are actively involved with rehabilitation therapists in planning the patient's rehabilitation program, including team meetings and family conferences.

Physical therapist provides assessment and treatment for balance disorders, dizziness, functional mobility, physical problems, and pain. Physical therapists can evaluate and address peripheral nerve and musculoskeletal injuries related to brain trauma, along with balance issues that may be centrally caused. Treatment goals include improving mobility, increasing strength, decreasing joint stiffness, improving static and dynamic balance, decreasing vertigo and dizziness, and managing pain and discomfort. The physical therapist also evaluates a patient's needs for equipment, such as, canes or braces to improve safety and endurance of ambulation.

Psychiatrist can prescribe pharmacological and behavioral interventions for the treatment of psychiatric disturbances occurring as a result of brain injury. The range of psychiatric disturbances which may follow brain injury is extensive and embraces most of what can be found in psychiatric symptomatology. Premorbid personality, pre-existing psychiatric disturbance and genetic predisposition also play a part in psychiatric complications after

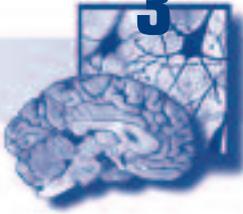
brain injury, but the contribution of the physical and neurochemical disruption to the brain should not be underestimated.

Recreation Therapist assists persons with brain injury to resume community life by helping them participate in play and leisure activities, which enrich life. Through leisure counseling, leisure education, leisure skills development, aquatic education, adaptive sports, resocialization programs and community readjustment outings, the person with brain injury learns how to participate in community life.

Social Worker (counselor) helps patients and their families respond to social, emotional, or financial problems resulting from physical disability or chronic illness. Treatment modalities include individual and group psychotherapy, crisis intervention, family counseling and family support groups. The social worker explores community resources and entitlement programs that are available to the patient and family.

Speech and Language Pathologist will evaluate and treat communication and cognitive deficits that may impact a person's everyday functioning. Speech pathologists assist patients who have speech, language, and cognitive problems to gain optimal communication skills. Communication problems may include difficulty understanding complex and abstract written or verbal information, difficulty finding words and expressing coherent ideas, and problems with the use of language in interpersonal relations. Cognitive problems, such as difficulty paying attention, learning and remembering information, organizing ideas, reasoning, and solving problems also interfere with communication skills. Academic skills may also be assessed to rule out potential deficits that may have an impact upon the person's return to community interaction, work, and school.

Vocational Rehabilitation Counselor may act as a treatment coordinator for patients who are having difficulty returning to work after a brain injury. This specialist may be asked to provide assistance in returning to work, collaborating with employer, patient, and co-workers to build a successful working relationship.



Rehabilitation of the Veteran with Traumatic Brain Injury

Learning Objectives

1. Describe standards of care for trauma care after TBI.
2. Identify references for consensus guidelines.
3. Describe the benefits of rehabilitation after TBI.
4. Identify what constitutes interdisciplinary “acute” rehabilitation.
5. Identify common medical complications seen in the acute recovery period after TBI.

Acute Care of the Individual with Moderate to Severe Brain Injury

Following the acute traumatic brain injury, veterans may need continued care in a variety of settings. If the injury is of sufficient severity, they will require acute trauma care followed by inpatient rehabilitation. Typical medical problems associated with the brain injury must be addressed during the inpatient period. Following a period of inpatient rehabilitation, they may need extended services to ensure maximum return to premorbid level of function. These services may be provided in the outpatient setting, or in other programs specifically designed for this purpose. Some individuals do not recover to the point of participation in rehabilitation programs and thus need long-term care services.

Trauma Care

All regions in the U.S. should have an organized neurotrauma care system for the initial treatment of severe TBI. Consensus guidelines for the acute management of traumatic brain injury have been developed by The Brain Trauma Foundation, The American Association of Neurological Surgeons, and The Joint Section on Neurotrauma, and Critical Care, and are available in *Journal of Neurotrauma*, 2000 Jun-Jul volume 17. These guidelines cover initial management, systems of care, resuscitation of oxygenation and blood pressure intracranial hypertension, cerebral perfusion pressure monitoring and control, hypotension, role of steroids, role of antiseizure prophylaxis, age, computerized tomography scan features, Glasgow coma scale, and nutrition. Aggressive surgical intervention has been linked to better outcomes whereas delayed surgical intervention has been linked to poorer outcomes.

Part 1

http://www.guideline.gov/summary/summary.aspx?doc_id=3121&nbr=2347&string=brain+AND+injury

Part II

http://www.guideline.gov/summary/summary.aspx?doc_id=3122&nbr=2348&string=brain+AND+injury

Acute Rehabilitation

Following acute emergency evaluation and medical stabilization, the individual with a moderate to severe traumatic brain injury usually requires a period of inpatient rehabilitation. These services are best provided in an established interdisciplinary brain injury program. The focus in the early rehabilitation phase is to restore the individual to maximal functional independence. To be eligible for admission to an inpatient brain injury program, the individual must be medically stable and able to participate in rehabilitation therapies. He/she usually requires frequent contact with a physician to monitor new or ongoing complications of the brain injury and the services of nurses specializing in rehabilitation care. There should be a reasonable expectation for functional improvement.

The benefits of rehabilitation after TBI have been well chronicled in the medical literature. Outcomes are better when structured rehabilitation interventions begin in the ICU. Early rehabilitation principles include:

1. controlled mobilization out of bed;
2. sensory regulation (avoid over- or under-stimulation);
3. avoidance of cognitively impairing medications;
4. regular pain assessment; and
5. early removal of catheter with implementation of timed voids.

When significant pain is recognized, acetaminophen is the drug of choice, utilizing scheduled rather than on-demand dosing.

The current standard of care dictates transfer to a formal interdisciplinary rehabilitation unit. Physiatrists, physicians specially trained in physical medicine and rehabilitation, are best equipped to direct interdisciplinary programs. Inpatient interdisciplinary programs generally provide 3 or more hours of formal therapy (physical, occupational, speech, recreational, neuropsychology) a day. Nurses and aides participate in the treatment process by reinforcing the skills being worked on in the therapies and encouraging patients to carryover and apply functional improvements during non-therapy hours. Those unable to actively engage in therapy for several hours daily, like minimally responsive or comatose patients, would not be appropriate to begin this level of rehabilitation.

Interdisciplinary refers to therapists and nurses, along with the patient, their family, and the physician communicating regularly and working toward common goals. Better outcomes have been shown in interdisciplinary

compared to multidisciplinary programs, so this an important distinction. The Commission on Accreditation of Rehabilitation Facilities (CARF) is the leading agency to certify the quality of interdisciplinary programs. CARF offers separate certification for brain injury rehabilitation, because brain injured patients have unique needs for sensory regulation, behavioral modification, cognitive retraining, and safety monitoring which are beyond the scope and training of most acute general rehabilitation programs. Better rehabilitation outcomes have been demonstrated in dedicated brain injury rehabilitation programs. There are currently four CARF accredited acute TBI programs in the VHA: Minneapolis, Palo Alto, Richmond, and Tampa.

Upon admission, the individual undergoes evaluation by the rehabilitation team and goals for expected short- and long-term progress are set. Therapies are established based on the current functional level and responsiveness of the individual. This section describes common rehabilitation interventions based on the individual's current estimated Rancho Los Amigos Level of functioning (Section 1, pp 16-17).

Rehabilitation at Different Stages of Recovery

1. **At Rancho Level I**, the patient is nonresponsive. Care is focused on preventing complications.
2. **At Rancho Level II**, the patient exhibits non-specific or generalized responses to environmental stimulation. The individual reacts inconsistently and non-purposefully. Responses may be physiological (increased blood pressure or heart rate), gross movement, or non-meaningful vocalization. Goals for this level include increasing the level of responsiveness, increasing initiation of responses, and increasing the localization or specificity of responses. The treatment process is one of controlled stimulation. Sensory stimuli utilizing all sensory modalities are presented in a systematic fashion and elicited responses are noted. When possible, meaningful stimuli such as familiar music, pictures, or voices are used. Stimulation is limited to short periods interspersed with scheduled periods of rest. Family members may often be involved. In addition to providing controlled stimulation, therapy at this stage focuses on prevention of complications and managing medical issues such as adequate nutrition, protecting the airway, etc. To prevent complications at this level, therapists are involved in splinting, positioning, serial casting, and range of motion. Excellent nursing care is essential for skin care, feeding, and bowel and bladder management.
3. Movement to **Rancho Level III** is marked by the emergence of localized responses. The individual will react specifically, but inconsistently, to stimuli that are presented. He/she may withdraw to pain, turn to sound, or track a moving object.

He/she may follow simple commands and pull at tubes. The goals at this level are to increase the level of responsiveness, increase the consistency of responses, and increase functional responses. Occupational therapy (OT) will begin to work on simple activities of daily living such as face washing using direct hands on contact to assist the individual in performing the motions involved. Physical therapy (PT) will work on activities related to mobility such as upright posture and truncal stability. Speech therapy (ST) will elicit responses to communication and swallowing. There is an attempt to establish voice and stimulate swallowing activities. Staff support and provide education to the family, which is critical during these early phases as well as throughout the rehabilitation process.

4. **Rancho Level IV** is marked by increasing alertness and agitation. The individual at this stage is alert and highly active. Behavior may be bizarre or aggressive and is usually non-purposeful. These individuals are unable to process information and form new memories. They tend to be confabulatory. They are often uncooperative with therapists. The goals for this level are to reduce agitation and increase consistency and functionality of responses. At this stage, the individual requires a controlled environment free of distracting stimuli and with continued scheduled rest periods. Verbalizations to the individual are kept simple and concrete and there should be external cues for orientation to person, place, and time. Therapists work on functional activities such as grooming and other activities of daily living (ADLs), mobility, and establishing a consistent yes/no response.
5. **At Rancho Level V**, the individual is less agitated but remains confused and highly distractible. Behavior is often inappropriate. Memory impairment is severe and there is no carry over from day-to-day. He/she is able to respond to simple and basic commands. Starting at Rancho Level V, restorative treatment of cognitive function begins. Specific treatment modules for attention (Attention Process Training; Sohlberg & Mateer, 1987), memory (Sohlberg & Mateer, 1989), and executive function (Sohlberg & Mateer, 2001), may be utilized at this level. Therapists continue to work on functional activities at this level as well. At Rancho Level V, OTs will assist with dressing and grooming at bedside.
6. **At Rancho Level VI**, the individual remains confused but behavior begins to be more appropriate. He/she begins to show goal directed behavior but continues to need structure and direction. He/she begins to show carry over and may recognize familiar staff. Ability to process information remains impaired. At this level, the goals of therapy are to decrease confusion, improve independence, improve cognition and information processing speed. At Rancho Level VI, the individual may be independent in many basic ADLs (bathing, grooming, dressing), but still require

cues or checklists to complete such tasks. Cognitive tasks are integrated into functional tasks during therapy sessions. PT at these levels will continue to work on mobility, utilizing special techniques to improve walking as needed, and for those individuals who have now become independent in ambulation, will work on endurance, pathfinding, and general exercise programs. ST will focus on dysarthria, aphasia, and swallowing deficits at this level. It remains important to maintain a routine with scheduled rest periods and external cues as needed. Therapies may be moved to more distracting environments to challenge emerging improvements in attention. Psychology at this stage is important to educate staff about the individual's cognitive impairments and suggest best strategies for approaching treatment. For example, some individuals may process verbally presented stimuli better than visual. Psychologists often must assist with behavioral issues beginning at these stages and must be available for counseling and support to families and the individual receiving treatment.

7. At **Rancho Level VII**, the individual becomes appropriate in an automatic fashion in highly structured surroundings such as the hospital unit. However, they still show impaired judgment and limited insight into their deficits. They are independent in basic ADLs, mobility, feeding, and communication.
8. **Rancho Level VIII** continues this improvement with the individual better able to function without supervision. They often continue to show cognitive deficits in information processing and abstract reasoning, and behavior may break down in emergency or unfamiliar situations. The goals for therapeutic intervention at Rancho Levels VII and VIII are to increase processing of complex information, increase awareness of deficits, and develop compensations for memory, communication, problem-solving deficits, and executive functions. Treatment at this level could be provided in the outpatient setting. Cognitive treatments continue to focus on higher levels of attention, and memory. Exercises in prospective memory — remembering things, which must be completed in the future — begin at this level (Sohlberg & Mateer, 2001). In addition, individuals who have deficits in social function, or pragmatics, may begin therapy to restore a more normal pattern of social interaction at this level. Also, strategies to improve problem-solving, planning, and other executive functioning will be initiated here. OT will begin work on instrumental activities of daily living (IADLs) including the management of money, balancing a checkbook, shopping and cooking, etc., and will provide an estimate for the individual's need for supervision. A driving evaluation prior to return to driving is advised at Rancho Level VIII. RT at this level will provide education for safe participation in leisure activities and provide references for community resources. They will provide continued exposure to

community activities increasing the individual's responsibility for planning and carrying out these activities. They will also explore alternatives to paid work for the individual who is seeking meaningful activity but is not yet ready to return to work.

At this point, the individual is transitioned into a community re-entry program to continue the rehabilitation process. They no longer need or benefit from the highly structured environment of the hospital, and medical problems have been addressed and stabilized. Individuals are independent in the use of medications and no longer need rehabilitation nursing care.

Individuals who do not reach a level at which they are able to function relatively autonomously are continued at the appropriate level of therapy and supervision while they continue to make improvement in their functional abilities. They can be transitioned to other treatment settings based on their need for medical care, nursing care, and supervision. Some individuals may require long-term care in a medical setting, not an acute rehabilitation facility. These individuals should have rehabilitation monitoring and evaluation available on a long-term basis to address problems and complications that may arise as a result of their brain injury. Others may require long-term supervision that their families are unable to provide, such as in a group home setting or supervised apartment. Identification of such facilities or programs can be facilitated through contact with specialized TBI case managers. Long-term monitoring and management by rehabilitation professionals is generally recognized as beneficial to individuals who have sustained a moderate to severe brain injury.

Points to Remember

- **Following acute emergency evaluation and medical stabilization, the individual with a moderate to severe traumatic brain injury usually requires a period of inpatient rehabilitation**
- **Brain injured patients have unique rehabilitation needs for sensory regulation, behavioral modification, cognitive retraining and safety monitoring that are beyond the scope and training of most acute general rehabilitation programs**
- **Rehabilitation needs and intervention of a TBI patient vary by their current Rancho Level**

Common Medical Complication during the Acute Rehabilitation Period

Seizures

(<http://www.emedicine.com/neuro/topic318.htm>)

All patients with TBI are at increased risk for new-onset seizures, termed post-traumatic seizures (PTS). The epileptic episodes may be due to direct damage of brain tissue which has resulted from shearing forces, infarction, or due to secondary irritation caused by hemorrhage. Seizures may also be triggered by secondary insults including metabolic disturbances and hypoxic episodes. Depending on the underlying cause, a seizure can originate from a focal or generalized area, as in cases of anoxia. It is important to be aware that seizures arising from the frontal lobe may resemble aggressive behavior – apparent purposeful thrashing and kicking, with possible verbal automatisms (Yablon & Dostrow, 2001). This must be recognized as potential seizure activity so that appropriate investigative and treatment measures can be taken.

Most studies have failed to demonstrate the benefit of anticonvulsant prophylaxis for late PTS. Therefore, after the first week following head injury, seizure prophylaxis is not recommended (Liebert, 2000; http://www.aan.com/professionals/practice/pdfs/ant_dru_pro.pdf). When late PTS presents, investigation for possible underlying triggers should ensue (metabolic, substance abuse, structural). If no correctable sources are uncovered, or if a second seizure occurs, treatment with anticonvulsant medication is warranted.

The incidence of late PTS (occurring beyond the first week post-injury) is approximately equivalent to the incidence of early PTS (within 7 days following TBI). New-onset of late PTS can occur at any time, but the likelihood diminishes over time. Most patients with late PTS (75-80%) experience onset within the first two years, 50-67% having their first seizure by the end of the first year (Yablon & Dostrow, 2001). Patients experiencing the onset of PTS should be referred to a neurologist for thorough evaluation and management.

Spasticity

(<http://www.emedicine.com/pmr/topic177.htm>)

TBI, like other upper motor neuron diseases, is associated with spasticity. Spasticity is a velocity-dependent increase in tone, resulting from the loss of inhibitory inputs from the CNS to the monosynaptic stretch reflex in the spinal cord (Watanabe & Sant, 2001). The degree of spasticity usually correlates with the degree of extremity motor weakness. Spasticity must be distinguished from other causes of resistance to passive movement including anxiety, joint pain, heterotopic ossification, and contractures.

Spasticity, by impeding normal physiologic range of motion, can both interfere with functional activities and lead to joint contractures. Contracture, fibrosis of tissues resulting in fixed resistance to passive stretch of muscles, presents a formidable obstacle to treatment once it develops. Spasticity can also lead

to pain syndromes and skin breakdown. On the other hand, spasticity can provide benefits, as when extensor tone in the leg aids standing activities.

Sudden worsening of spasticity warrants a search for potential triggers including infection, ulcers, and metabolic disturbance. Whenever spasticity develops, a stretching, positioning, and range-of-motion program should be instituted. A myriad of further treatment options are available including physical modalities, splinting, neurolytic, or botox injections, oral medications, and surgical procedures (<http://www.biausa.org/word.files.to.pdf/good.pdfs/roadToRehab7.pdf>). The decision to employ them is based on careful evaluation of the severity of the spasticity, the distribution of involved joints, the functional status, and risk-benefit analysis of each individual treatment option. Psychiatrists, occupational therapists, and physical therapists can provide important expertise in the treatment of spasticity and resulting complications.

Neuroendocrine Dysfunction

(<http://www.emedicine.com/pmr/topic109.htm>)

Dysfunction of the endocrine system can occur anywhere along the hypothalamic-pituitary-end-organ axis. For patients with TBI, the most likely source of dysfunction is in the central aspects of this axis. Skull fracture, hemorrhage, ischemia, and brain edema can all cause damage to the hypothalamus or pituitary gland. The most common resulting endocrine abnormalities are SIADH and diabetes insipidus (Watanabe & Sant, 2001). Because endocrine dysfunction is so common in TBI (affecting up to 20%), it is important to be able to determine the underlying cause of the various electrolyte abnormalities. This will allow for the use of appropriate methods for correction of imbalances in a given patient.

Panhypopituitarism

Significant trauma to the head can result in problems with the neuroendocrine axis. The pituitary gland is susceptible to injury based on its location in the sella tursica, a bony structure at the base of the skull. Symptoms are those of hypoadrenalcorticism, hypothyroidism, decreased growth hormone, and/or failure to lactate after parturition.

The syndrome of panhypopituitarism may manifest an insidious onset, weeks to months after the original closed head injury. The patient may become progressively lethargic or anorexic and may demonstrate hypothermia, bradycardia, or hypotension with hyponatremia. These symptoms result in a significant setback if they occur during the acute phase of rehabilitation of the patient who has sustained a closed head injury. Any unexplained onset of malaise and generally decreased vital signs with associated stagnation of rehabilitation progress in a patient following closed head injury should prompt the clinician to suspect the presence of panhypopituitarism or anterior hypopituitarism (AH).

The endocrine workup for panhypopituitarism includes serum hormonal assays (e.g., cortisol, testosterone, triiodothyronine (T₃), thyroxine (T₄), thyroid stimulating hormone [TSH]). Perform a complete blood count (CBC) and serum electrolytes as well.

Treatment involves multiple hormonal replacement therapy and monitoring of serum levels along with the clinical response of the patient. The patient usually responds with improved vital signs, improved constitutional symptoms, and increased endurance for participation and progress in the rehabilitation program. The hormonal replacement therapy usually is required long-term.

Gastrointestinal Complications

Individuals with TBI have an increased caloric requirement in the acute phase of recovery. This often presents an added challenge due to the fact that patients already have difficulty consuming normal caloric requirements, much less increasing their oral intake. Patients with TBI often have facial trauma or dysphagia, and demonstrate a low level of arousal, all of which present obstacles to oral consumption (Watanabe & Sant, 2001). These individuals also may experience a decrease in their appetite as well, resulting in little or no motivation to eat. Thus, feeding tubes are frequently required in the acute rehabilitation phase. Another complication in the GI tract is GI bleeding, with up to 69% of patients with TBI having gastritis or duodenitis following their injury (Watanabe & Sant, 2001). Due to their lower incidence of CNS side effects, proton pump inhibitors are preferred over H2 blockers for prophylaxis during acute hospitalization.

Hydrocephalus

(<http://www.emedicine.com/pmr/topic113.htm>)

Non-obstructive hydrocephalus is a frequent complication after severe TBI. The usual cause is disruption of the absorptive capability of the arachnoid villae. The classic clinical signs are similar to normal pressure hydrocephalus (imbalance, incontinence, and dementia), though a high index of suspicion is required of the treating physician because the onset in the severely injured is typically slow, with the earliest indication often a vague decline or plateau in functioning and/or subtle mental status changes. Head CT is the investigative study of choice, and when enlarged ventricles are seen, true hydrocephalus must be distinguished from hydrocephalus *ex-vacuo*. In hydrocephalus *ex-vacuo*, loss of parenchymal volume allows the ventricular system, which is under pressure, to expand giving the appearance of hydrocephalus on imaging studies (Bigler, 2001). If the CT findings are inconclusive, serial CT scans and sometimes CSF tapping tests may be needed.

Heterotopic Ossification

(<http://www.emedicine.com/pmr/topic112.htm>)

The formation of new bone in non-bony tissue can be seen in the acute recovery period following TBI occurring in 11-77% of cases (Watanabe & Sant, 2001). Risk factors include prolonged coma (>2 weeks), immobilization, skeletal trauma, and spasticity. The most commonly affected joints include the hip, knee, shoulder, and elbow. Complications include decreased range of motion, nerve and/or vascular compression, lymphedema, and ankylosis. The early signs of HO typically presents with

pain, erythema, warmth swelling and fever, and can resemble local trauma, fracture, cellulitis, or deep venous thrombosis (DVT). Therefore, it is important to keep HO in the differential when a patient with TBI presents in this manner. Heterotopic ossification typically begins within the first 2-3 weeks following injury, but onset can occur from 1 to 7 months post-TBI (Watanabe & Sant, 2001). Treatment remains controversial, with options including etidronate, non-steroidal anti-inflammatory drugs, range of motion, and surgery. Referral to a physiatrist for recommendations regarding treatment is recommended.

Urinary Incontinence

Urinary incontinence is common acutely after TBI, and in some may continue chronically. The usual etiology is loss of normal cerebral inhibition and control over bladder and sphincter activity, secondary to frontal or diffuse cognitive injury. Detrusor hyperreflexia and urinary retention can also occur, particularly early. In the absence of hyperreflexia, retention, or obstruction, the preferred treatment is bladder “training” with scheduled timed voiding attempts. New incontinence in the previously continent patient should elicit a search for inciting causes such as urinary tract infection or a new structural insult like hydrocephalus.

Deep venous thrombosis (DVT)/Pulmonary embolism (PE)

Virchow’s triad lists the three most significant risk factors for deep venous thrombosis – venous stasis, trauma, and a hypercoagulable state. Individuals with moderate and severe TBI typically meet 2 or 3 of these conditions, and are thus prone to DVT. Published incidence rates of DVT soon after severe TBI range from 20-54% (Watanabe & Sant, 2001). With increased risk of DVT also comes an increased risk of pulmonary embolism and death. While early recognition of DVT is important, most DVT’s are clinically silent. Effective prophylaxis geared toward the appropriate risk level is paramount. Consensus evidence-based guidelines for DVT risk stratification and prophylaxis during hospitalization are readily available (Chest Supplement 2000; http://www.chestjournal.org/cgi/content/full/119/1_suppl/1S).

Additional Information

The following Web site contains additional potentially useful information about post-TBI autonomic and endocrine complication, heterotopic ossification, hydrocephalus, and epidemiology and pathophysiology of TBI: http://www.emedicine.com/pmr/TRAUMATIC_BRAIN_INJURY.htm

Points to Remember

- **All patients with TBI are at increased risk for new-onset seizures**
- **Non-obstructive hydrocephalus is a frequent complication after severe TBI**

Other Resources for Rehabilitation Services

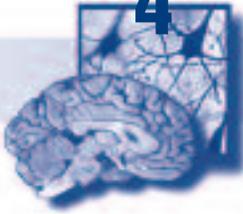
For patients who have progressed beyond, or are not appropriate for acute inpatient rehabilitation, a variety of other services can be accessed for specific impairments. These services include outpatient therapies, community programs, vocational rehabilitation, and services provided in long-term care facilities.

Often, impairments identified in the acute rehabilitation period require continued treatment after discharge. Also, patients with history of moderate to severe brain injury may present with new or worsened deficits in the post-acute period. Impairments such as dizziness, spasticity, balance disorders, falls, contractures, weakness, decreased coordination, problems with ADLs, etc., all can be addressed through outpatient therapies. Specific referral guidelines will be discussed later in sections devoted to management of chronic sequelae.

Occasionally, veterans with traumatic brain injury may require continued services in a community-based setting. Some examples of these programs include transitional living programs, supported employment, adult day programming, vocational rehabilitation, and specialized neurobehavioral programs.

Some patients may never reach the level of responsiveness necessary for participation in an acute rehabilitation program. For these individuals it is recommended that they receive long-term coma care in a long-term care setting. Evidence for coma stimulation programs which provide intensive levels of stimulation with the goal of increasing level of responsiveness and recovery is lacking in the medical literature. Care for persons in a state of low level of consciousness, often referred to as coma care, involves providing excellent medical care—providing adequate nutrition, pulmonary care, bowel and bladder management—and the prevention of complications such as contracture, skin breakdown, infection, etc.

The VA has a National Traumatic Brain Injury Coordinator, Gretchen C. Stephens, who is available to assist with identifying resources or appropriate placement for individuals with TBI. She can be contacted at (804) 675-5597 or e-mail at gretchen.stephens@med.va.gov.



Post-acute/Chronic Sequelae: Medical and Physical Problems

Learning Objectives

1. Describe a conceptual framework for intervening with the post-acute medical complaints of individuals with TBI.
2. Describe important principals for treating individuals with post-acute TBI problems.
3. Recognize the common medical problems in the post-acute phase of TBI.
4. List special considerations when making pharmaceutical interventions for TBI.

A Conceptual Framework

Given the heterogeneity of TBI, it is helpful to establish a framework for each patient who presents for medical evaluation. Answering the following four questions during the assessment process helps to prevent unrealistic expectations and target those individuals needing a multidisciplinary approach:

1. What was the medical severity of the initial injury?
(Discussed in Section 1)
2. What is the current neurobehavioral severity of the patient's "impairment"?

The assessment of the severity of the patient's current impairments should not be limited to a review of systems and reporting of symptoms. They should include assessment of any personality or mood changes, new onset interpersonal difficulties, and the impact of the injury on academic, vocational, and social aspects of current functioning.

3. What brings the patient to seek services right now?

The reasons the patient provides for seeking services can provide important information regarding the primary factors at play as well as the most appropriate treatments and/or referrals. For example, if the patient is five months post-injury and seeking services for the first time, it becomes critical to understand why the patient is presenting now versus two weeks ago, two months ago, or two days after the injury.

4. Is the patient's current neurobehavioral "impairment" driven more by person-centered factors (e.g., neurobehavioral symptoms, cognitive problems, premorbid vulnerability factors) or the patient's environmental demands (family/interpersonal, work, or school demands)?

The amount of prior treatment and services TBI patients have received may vary significantly. Understanding the patient's treatment history will provide invaluable information regarding potential first treatment steps. Persons who seek assessment and treatment might fall into one of at least five categories:

- **Never sought or received any treatment** – Persons who lack knowledge regarding the potential seriousness of TBI, who use denial as a coping mechanism, or who lack health insurance might be less likely to initially seek out services.
- **Hospitalized for TBI, never received inpatient rehabilitation services** – Many patients hospitalized with a diagnosis of mild or moderate brain injury are often deemed by acute care professionals as ready for discharge home without inpatient rehabilitation. Lack of service provision may be due to several factors. Many rehabilitation programs are tailored to meet the needs of persons with more severe TBI. Rehabilitation services for TBI are often unavailable in many rural areas. Lastly, managed care has contributed to reduced funding for rehabilitation services, especially for persons with less severe injuries.
- **Hospitalized for TBI, followed by inpatient rehabilitation services** – Patients with more severe injuries will likely have been medically hospitalized for acute medical care and then may have received a period of inpatient rehabilitation services (see section 3) for either a.) physical problems (Physical and Occupational therapy), b.) cognitive problems (Speech, Cognitive, or Occupational therapy) or c.) both (a comprehensive inpatient brain injury program). However, they may now be presenting months or years post-rehabilitation with newly emerging problems secondary to environmental changes (e.g., lost job, new vocational responsibilities, loss of social support).
- **Hidden or "Occult" TBI** – Patients who are acutely hospitalized for other medical problems (e.g., spinal cord injury, orthopedic injury) often have their neurobehavioral problems neglected or viewed as secondary in comparison to other medical issues. Many of these persons have long-term difficulties that are rarely addressed by outpatient medical and rehabilitation systems (Bohnen, Jolles, & Twijnstra, 1992; Englander, Hall, Simpson, & Chaffin, 1992).
- **One or more unsuccessful treatments** – Many patients with mild TBI become emotionally distressed because their symptoms fail to resolve. For some patients, treating professionals may have misattributed the emotional reaction as the primary cause of

disability and might not have fully assessed the patient’s medical condition. Conversely, for other patients, the emotional reaction may have been untreated but plays a significant role in mild TBI symptom chronicity.

Points to Remember

- Try to determine whether the patients complaints are driven more by the medical and neurological impairments or by the demands of the patient’s current environment
- Carefully review prior treatment attempts and their success before initiating new interventions

Medical Assessment

The post-acute medical evaluation of TBI patients should include a thorough history and physical examination. Pertinent elements are listed in **Table 1**. Historical information should include accident circumstances to help gauge severity of injury, including presence of acute alteration in consciousness, presence, and duration of coma and amnesia, blood alcohol level, and initial GCS. All pertinent medical records should be sought out and reviewed, with attention to brain imaging tests. Also important is an understanding of previous treatment interventions and their effectiveness, an understanding of the patient’s beliefs about brain injury and long-term prognosis, whether or not the patient has been provided with education about TBI and the typical course, and whether or not the patient has pending litigation related to TBI. Physical examination should focus on the neuromusculoskeletal systems, including a careful mental status examination and cognitive assessment.

Comprehensive Assessment of Acquired Brain Injury	
History	Physical Examination
Accident related facts	Neurological
Initial neurologic presentation	Cranial nerves 1-12
Pre-Injury information	Deep tendon and pathological reflexes
Past medical and surgical history	Sensory exam
Substance abuse	Cerebellar exam
Developmental history	Motor exam
Educational history	Mental status exam
Military and legal record	Behavioral assessment
Vocational history	Emotional/Psychological status
Psychosocial history	Musculoskeletal
Life stressors	Head
Family history	Face and temporomandibular joints
Post-injury treatment interventions	Extremities
Current functional status	Axial structures (neck, back, pelvis)

Table 1

The goal of the history and physical is to define current impairments and functional limitations and to identify all disability related factors. When multiple symptoms, neurobehavioral impairments, and/or disability related factors are identified, a referral to a **physiatrist** with brain injury expertise is desirable. Also, a comprehensive **neuropsychological assessment** allows more complete evaluation of cognitive, emotional, and behavioral functioning. Neuropsychologists are uniquely qualified to collect data and sort through the complexity of interactions between physiologic, psychogenic, premorbid vulnerability, and environmental factors that lead to the development of chronic TBI symptomatology. Head MRI should be undertaken in those presenting with ongoing cognitive problems or post-concussive symptomatology if prior brain imaging studies were not completed. Imaging is also recommended in those with new focal neurologic findings or whose condition has deteriorated. The need for further diagnostic imaging tests in those who received peri-injury brain imaging is rare.

Points to Remember

- **When multiple chronic symptoms are present, a referral to a physiatrist with brain injury expertise is desirable**
- **A comprehensive neuropsychological assessment may help identify complex interactions among cognitive, emotional, and behavioral sequelae to better focus treatment interventions**

Guiding Principals for Treating TBI Patients

Adopt a Multidisciplinary, Comprehensive Mindset for Treatment Provision

Patients who have sustained a TBI often feel misunderstood by family, friends, and colleagues. While patients' are keenly aware of post-injury changes, laypersons may insist that they "look and sound normal." Conversely, treating professionals sometimes focus exclusively on patients' symptoms and complaints during the course of assessment and therapy, ignoring emotional adjustment issues. Thus, many people with brain injury experience significant frustration and failure.

A "dose effect" understanding of head trauma suggests that higher levels of long-term impairment are associated with greater initial severity of trauma (Levin, Benton, & Grossman, 1982). However, research has often found that patients with mild TBI often report problems at equivalent or even greater rates than patients with more severe TBI (Larrabee, 1997, 1999). As a general rule of thumb, regardless of the severity of the initial injury those patients with multiple sustained and more severe symptoms are likely to require a more intensive multidisciplinary treatment approach.

TBI rehabilitation professionals have emphasized the need to take an interdisciplinary, holistic approach to treating patients who have sustained mild and more severe TBI (Prigatano, 1989; Ben-Yishay & Prigatano, 1990; Prigatano, 1999). This holistic mind frame emphasizes not only the treatment of specific physical and cognitive symptoms, but attending to the patients' individual needs which might include providing information, helping to motivate, instilling hope, and taking environmental factors into account. However, in the post-acute outpatient general medical setting, a multidisciplinary approach is more realistic, with the overall management of the patient's care directed by the patient's primary care physician.

Developing a successful TBI treatment framework is not limited to just attending to the patients' cognitive and physical symptoms. Successful treatment often will include additional interventions including educating, advocating, counseling, and support. Providing caring attention, helping the patient to feel understood, providing accurate information on symptoms and outcomes, and instilling hope contribute to symptom improvement and return to pre-injury functioning.

General Rules of Thumb for Prescribing Treatments

At the risk of oversimplifying the diagnosis and treatment of TBI sequelae, it is often helpful to have a general starting point from which to individualize approaches and treatment plans. A set of general rules for conceptualizing patients' difficulties can provide an intervention strategy. The following four rules of thumb provide a general compass for directing treatment of TBI sequelae.

1. The relationship between initial injury severity and current symptom presentation should guide the general approach to prescribing interventions.
 - More severe injury with ongoing severe, diffuse symptoms require Multidisciplinary care
 - More severe injury with few ongoing or focal symptoms require Symptom-specific interventions and monitor progress
 - Less severe injury with more ongoing severe, diffuse symptoms require Psychological coping interventions
 - Less severe injury with few ongoing or focal symptoms require Education and Monitoring
2. Don't prescribe what already hasn't worked.
3. Don't take on the "savior" role. If several other health care providers haven't "fixed" the symptoms from a relatively mild TBI, then you won't either.
4. If the patient has not responded to initial interventions, a comprehensive multidisciplinary approach to treatment coordinated by an experienced TBI-case manager or psychiatrist is highly recommended (http://vaww.va.gov/health/rehab/TBI_Case_Managers.htm).

Point to Remember

- **Adopting a multidisciplinary, comprehensive clinical mindset is important in dealing with the individual with chronic TBI medical problems**

Addressing Specific Medical Symptomatology

Common Medical Problems post-TBI

TBI typically involves a complex interplay of brain injury, extracranial tissue injury of the head and neck, and secondary or reactive symptoms. The resulting symptomatology can have somatic, cognitive, and affective/psychological/behavioral components. Common post-TBI medical and physical problems are discussed below, while the common cognitive and affective/psychological problems are discussed in Sections 5 and 6, respectively.

There are a number of common somatic medical problems that occur following TBI. Treatment for these conditions varies minimally regardless of severity or acuteness of the original brain injury. The following common acute medical complications that usually present during the initial hospitalization or acute rehabilitation phase were discussed earlier in section 3:

- Seizures (pg 55)
- Spasticity (pg 55)
- Neuroendocrine Dysfunction (pg 56)
- Panhypopituitarism (pg 56)
- Gastrointestinal Complications (pg 57)
- Hydrocephalus (pg 57)
- Heterotopic Ossification (pg 57)
- Urinary Incontinence (pg 58)
- DVT/PE (pg 58)

This section will discuss the following symptoms or problems, which are more likely to remain ongoing medical concerns during the post-acute or chronic phase of recovery from TBI:

- Post-Traumatic Headaches (PTHA)
- Pain
- Dizziness
- Fatigue
- Sleep disturbances
- Spasticity, Hydrocephalus, and Seizures
- Visual Impairments
- Vestibular Impairment

Post-Traumatic Headaches (PTHA)

(<http://www.biausa.org/word.files.to.pdf/good.pdfs/roadToRehab2.pdf>)

Treatment should be based on the category of HA as determined during the assessment and should target not only the physical pain but also the person's reaction to pain in daily life. Specific treatment options include short-term use of medication, conventional physical therapy, biofeedback, and psychotherapy for the development of coping techniques. Counseling sessions should include gradual exposure to the cause of the anxiety, cognitive reinterpretation, and systematic desensitization. "Habit reversal" involves detection, interruption, and reversal of maladaptive habits. These include jaw clenching/tension, head posture, and negative cognition or

thinking. Awareness training and deep breathing exercises are also beneficial. **Tension headache, including the cervicogenic variety**, are the most common form of post-traumatic headaches. Directed treatments include physical therapy, relaxation techniques, medications, and myofascial trigger point injections. Choice of analgesic medications is based on the principle of avoiding side effects that might precipitate or exacerbate cognitive or behavioral symptoms. Thus, the analgesic of choice is acetaminophen, followed by non-steroidal anti-inflammatory drugs, which occasionally cause neuropsychiatric effects. The third line of analgesic options are muscle relaxants, tramadol, midrin and/or light narcotics (darvon, codeine). In chronic daily tension type headaches antidepressants, including tricyclics and the selective serotonin reuptake inhibitors, are often efficacious, particularly when irritability and/or depression coexist. The treatment of **migraine type headaches involves** prevention and abortive strategies. Preventative medications include propranolol, amitriptyline, verapamil, and gabapentin. Abortive agents include selective serotonin agonists (e.g., sumatriptan), ergotamines, and the combination products of butalbital/aspirin/caffeine and isometheptene/dichloralphenazone/acetaminophen. For post-traumatic **TMJ Syndrome**, dental management is recommended with possible treatment by a physical therapist. **Neuralgic headache** types including occipital neuralgia may respond to nerve injections or neuropathic pain medications (anticonvulsants, especially gabapentin or topiramate, and tricyclic antidepressants). Patients who remain unresponsive to treatment may benefit from a comprehensive chronic pain treatment program (e.g., <http://www.vachronicpain.org>).

Pain (Acute & Chronic)

(<http://www.biausa.org/word.files.to.pdf/good.pdfs/roadToRehab1.pdf>)

Both acute and chronic pain can develop following traumatic brain injury. Headache is the most common pain complaint in this patient population but other pain sources are also frequent. Given the traumatic nature of the brain injury, it is likely that other areas of the body are also injured. Fractures, nerve injuries, and internal organ injury can also occur and may not be diagnosed in the acute care setting. Treatment depends on the etiology of the pain and should focus on interventions that are least likely to cause cognitive side effects and abuse. Once a thorough work-up is completed and pharmacologic management is indicated, drug choice should include consideration of concomitant symptoms. For instance, if a patient has neuropathic pain and mood instability then carbamazepine or gabapentin could be considered as a means to treat both problems. Narcotics should only be used for the treatment of acute pain and used for the shortest duration possible. Analgesic balms, acetaminophen, and NSAIDs should be considered as a first line treatment choice for most types of pain. Consideration for therapy or referral to a specialized pain program should be entertained (e.g., <http://www.vachronicpain.org>).

Dizziness and balance problems

Dizziness and balance problems, while common after mild TBI, most frequently subside spontaneously. Persisting dizziness and vertigo requires careful evaluation to find the underlying cause. This could include hypertension/hypotension, medication effects, alcohol/drug use, visual dysfunction, or other medical conditions. The most common cause of dizziness following mild TBI is related to post-trauma vestibular system dysfunction, also known as **benign positional vertigo**. Treatment is very sophisticated and is discussed under separate cover (Furman & Cass, 1999). **Cervical vertigo** symptoms often respond to multiple therapeutic interventions provided by a physical therapist.

Fatigue

Fatigue can have several contributing factors, so a wide range of possible causes should be explored including frustration, depression, vocational, family, and social demands. For the fatigued patient who tries to maintain a pre-injury lifestyle that included managing a staff of 40, taking three night courses, and having a wife and four small children, a reduction in environmental demands would make a practical first step intervention. **Chronic fatigue** with related decreased attention, concentration, and memory problems, may respond to moderate use of caffeine or short-term trials of methylphenidate (Ritalin) or amantadine (Symmetrel). Medications should only be used after non-pharmacologic options have been exhausted.

Sleep disturbances

Sleep difficulties are common sequelae of mild TBI. With poor sleep, the patient often develops progressive problems with daytime fatigue that contribute to increased irritability and reduced cognitive performance. Treatment should begin with removal when feasible of all medications with stimulant properties, both prescription and non-prescription (including caffeine and alcohol). Once other confounding factors like sleep apnea, nocturnal seizures, and pain are ruled out, behavioral strategies should be implemented (avoiding daytime naps, avoiding caffeine after the morning, avoiding late night snacks, avoiding use of bed for activities other than sleep). Sleep inducing medications can be judiciously added with the understanding that their effects can carry over into the daytime contributing further to memory impairment, increased irritability, and depression. Some individuals can also react with paradoxical worsening of insomnia. Low doses of Trazodone, 50-150 mg can be highly effective in addressing post-traumatic insomnia, particularly when there is an anxiety component. Low doses of amitriptyline, 10-50 mg at bedtime, also may be an effective option, particularly when headache or negative affect coexist. Anticholinergic side effects can be troublesome with amitriptyline, in which case nortriptyline offers an alternative.

Spasticity, Hydrocephalus, and Seizures

The post-acute TBI patient, especially in cases of moderate-severe injuries, may present post-acutely with signs or symptoms related to spasticity (pg 55), seizure (pg 55), or hydrocephalus (pg 57). For further discussion of these conditions, refer back to the pages next to each condition.

Visual Impairments

Visual impairments frequently go undetected following brain injury, in part because of the patient's unawareness of visual changes or inability to communicate their altered experience (Gianutsos, Ramsey, & Perlin, 1987). The major visual impairments associated with TBI includes disorders of accommodation, oculomotor control, and binocularity.

- **Accommodative Dysfunction:** Visual accommodation is the ability to focus on an object at various distances (Suchoff et al., 2001). Accommodative dysfunction is one of the most common visual impairments following brain injury (Gianutsos, Ramsey, & Perlin, 1987), occurring in nearly 66% of patients. Individuals lose the ability to focus automatically, and thus must consciously attend to clearing blurred images. It may interfere with ADLs, reading, computer use, driving, and return to work (Scheiman, 2002). There are two basic approaches to treating accommodation problems: prism lenses and vision therapy (Suchoff et al., 2001). Prisms can be prescribed by a neuro-optometrist. Vision therapy is typically conducted by occupational therapists.
- **Oculomotor Dysfunction:** Specific oculomotor skills include fixation, saccades, and pursuits. Post-injury deficits may include problems with scanning, tracking, saccadic dysfunction, and nystagmus (Scheiman, 2002). Nearly 40% to 50% of individuals with brain injuries have some form of oculomotor dysfunction (Gianutsos, Ramsey, & Perlin, 1987; Suchoff et al., 1999). Patients may report dizziness, nausea, losing their place while reading, or eyestrain. Again, treatment includes use of prisms and/or vision therapy (Scheiman, 2002).
- **Binocular Dysfunction:** Binocular vision is dependent on the ability to align the two eyes so that they focus on the same point in space. Suchoff et al., (1999) found that more than half of patients with brain injuries had binocular disorders of which the overwhelming majority were exo-deviations. Symptoms include double vision, eyestrain, and headaches. Persons with diplopia often recover spontaneously within the first six months. If not, a patching regimen, Fresnel (stick-on prisms), vision therapy, corrective surgery, or any combination of these may be necessary (Falk & Aksionoff, 1992).

Vestibular Impairment

Vestibular injuries manifest as complex complaints including dizziness, vertigo, balance problems, disorientation, or visual disturbances. Fitzgerald

(1996) reported that between 15 to 78% of all individuals with mild brain injury have dizziness as a clinical symptom, and vertigo is clinically seen in as many as 75% of mild brain injuries and almost all of the patients with moderate brain injuries (Gizzi, 1995). However, that does not mean these symptoms necessarily indicate underlying vestibular impairment. A formal vestibular function evaluation may be indicated for patients reporting sustained symptoms, and might consist of caloric and rotary tests, electronystagmography (ENG), and post-urography of dynamic balance. Vestibular, visual, and proprioceptive systems are challenged in an effort to isolate the malfunctioning sensory modality. Audiometry and a thorough neuro-ontologic evaluation may also be indicated. Benign paroxysmal positioning vertigo (BPPV) is a condition commonly identified following TBI (Furman & Cass, 1999). Those with a bilateral complete loss of function may benefit from an exercise program that focuses upon compensatory strategies for postural control (Cohen, 1994; Gizzi, 1995; Herdman, 1990; Shumway-Cook, 1992). Emphasis upon the remaining visual and proprioceptive systems and balance control may optimize therapeutic outcome. Those with unilateral lesions often respond to a program of habituation exercises, patient education, and postural/balance retraining (Godbout, 1997).

Special Considerations when Making Pharmaceutical Interventions for TBI

It is critical for the treating physician to identify potential drug interaction effects with TBI symptomatology. In particular, medications for headaches, musculoskeletal pain, or depression/anxiety must be carefully reviewed to avoid sedation, which can have an impact upon a person's attention, cognition, and motor performance. The rule universally applied to the elderly of "start low and go slow" applies to the TBI population as well. In addition polypharmacy should be minimized.

Precautionary Steps

(<http://www.biausa.org/word.files.to.pdf/good.pdfs/roadToRehab6.pdf>)

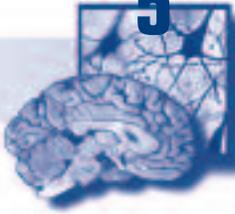
- **Provide appropriate therapeutic drug trials and allow adequate time for one drug to clear out of the person's system before changing to another medication**
- **Allow adequate time for any drug trials**
- **Rule out other causes of symptoms/behaviors; know the person's history**
- **Know all the medications the person takes and their possible interactions**
- **Evaluate the person's potential for addictions**
- **Check for non-compliance in taking medications that could be either intentional or due to poor memory**
- **Inform all those who are treating the person of his/her current medications and any medication changes**

Role of the Primary Care Physician in the Management of Medical Symptomatology

In the post-acute or chronic phase, the primary care physician is the individual most likely to manage the ongoing care of the survivors of moderate to severe TBI. A careful medical history and examination will help identify any residual problems, and the likelihood that they require additional intervention. The primary care physician may well want to consult with other specialists, or enlist the aid of a TBI case manager if available (http://vaww.va.gov/health/rehab/TBI_Case_Managers.htm). Clinically directed appropriate referrals for additional assessment may include consults to the following disciplines:

Audiologist	Physiatrist
Case Manager	Physical therapist
Kinesiotherapist	Psychiatrist
Neurologist	Recreation Therapist
Neuro-ophthalmologist	Social Worker (counselor)
Neuropsychologist (psychologist)	Speech and Language Pathologist
Occupational therapist	Vocational Rehabilitation Counselor

See page 45-47 for a description of the care provided by each discipline.



Post-acute/Chronic Sequelae: Cognitive Problems

Learning Objectives

1. List the common chronic cognitive problems affecting individuals with moderate to severe TBI.
2. Describe pharmacological agents that have shown some promise in improving cognitive functioning in some individuals with TBI.
3. Describe the indications for cognitive therapy in the chronic phase of recovery from TBI.

Common Chronic Cognitive Problems Affecting Individuals with Moderate to Severe Brain Injuries

While TBI is characterized by substantial heterogeneity in its pathophysiology, long-term cognitive deficits from moderate to severe TBI can be viewed as generally resulting from frontal-temporal damage superimposed on more diffuse pathology. Orbitofrontal and anterior temporal regions are particularly susceptible to damage. In keeping with these characteristics, the following symptoms have been seen as the most prominent cognitive sequelae following moderate to severe TBI:

- Attention and concentration problems
- New learning and memory deficits
- Executive control dysfunction

A variety of other problems also may be present at various stages of recovery depending on injury differences. The predominant symptoms, their assessment, and recovery course will be summarized next.

Attention problems are common following moderate to severe brain injury, particularly among those, which occurred under conditions of rapid deceleration. Several subdivisions of attention have been considered (Sohlberg & Mateer, 1987, 2001):

- Focused attention: the basic ability to respond to stimuli.
- Sustained attention: vigilance or the ability to maintain attention over time during a continuous activity. Working memory is a similar concept and refers to the ability to actively hold and manipulate information.
- Selective attention: reflects the ability to maintain an attentional focus in the face of environmental distractions.

- Alternating attention: the capacity for mental flexibility, appropriately shifting one's attention between two or more ongoing activities.
- Divided attention: the ability to respond to two tasks simultaneously.

Attention problems after TBI are seen particularly on novel and speeded tasks, and in part are due to slowed information processing speed associated with diffuse axonal injury. However, the dorsolateral aspects of the frontal lobes are also responsible for directing and controlling attentional resources.

New learning and memory problems are among the most commonly reported deficits after brain injury and typically are associated with medial inferior temporal lobe damage. Such deficits are a major reason for failure to return to work or school, or for difficulty performing complex activities of daily living. Memory span (e.g., short-term memory as reflected in digit span forwards) and remote memory for events that happened long before the injury usually are relatively unaffected. Psychomotor skill learning also may be preserved. Difficulty learning new information, retaining it, and subsequently retrieving it are the most common memory impairments. Following TBI, alcoholics, particularly those intoxicated at the time of injury, are likely to have greater memory impairments than persons with no alcoholic history.

Executive control dysfunction often is associated with frontal damage and is common after brain injury. Examples of executive control functions include:

- Abstraction
- Reasoning, problem solving, and conceptualizing
- Planning and organization
- Foresight (anticipation)
- Flexibility/adaptability
- Initiative and drive
- Response inhibition
- Task persistence
- Generative thinking

Patients with executive control dysfunction typically show symptoms including loss of spontaneity, increased impulsivity, difficulty moving flexibly from task-to-task, or perseverating in a non-productive manner. When injuries are predominantly frontal, the patient may perform well on familiar, highly structured tasks but is likely to have difficulty functioning independently. Diminished awareness of deficits also can occur with frontal injury. Without such awareness, patients are not motivated for retraining and do not monitor their performance properly. Deficits associated with frontal lobe injury often are the most handicapping as they interfere with the ability to use otherwise intact skills adaptively.

Other prominent cognitive sequelae of moderate to severe TBI can include impairment in orientation, mental and behavioral slowing, communication problems, visual-perceptual changes, and psychomotor problems. Although aphasia is relatively rare following traumatic brain

injury, a tendency for breakdown in linguistic competence has been associated with more severe damage. Word finding, misnaming, and poor conversational fluency are common problems. Communication-relevant skills, including pragmatics, also often are impaired. Communication pragmatics include the social and non-verbal aspects of communication such as social appropriateness, voice volume, rate, and tone, gestures and body posture, and interpersonal style.

Deficits in general intelligence are not characteristic after TBI. Lower scores on intellectual tests have been related to severity, but recovery is seen over time. For example, long after the acute stages have passed, TBI adults achieved score patterns on the Wechsler Adult Intelligence Scales that tended to approximate the population average. Abstract reasoning skills are more affected than knowledge of more over-learned information.

Points to Remember

- **The three most common areas of cognitive difficulty in chronic moderate to severe TBI are: a) Attention and concentration problems, b) New learning and memory difficulties, and c) Executive control dysfunction**
- **Executive control dysfunction often is associated with frontal damage, and includes impairment in higher-order reasoning, problem-solving, and cognitive flexibility**

Assessment of Cognitive Problems Affecting Rehabilitation and Community Re-entry

In the acute and early post-acute stages, patients often are not capable of meaningful cooperation or valid responding on psychometric cognitive tests. The following assessment instruments typically are used to track improvement in responsiveness and orientation in the early stages after moderate to severe brain injury:

- Rancho Los Amigos (RLA) Scale: Levels of Cognitive Function
- Galveston Orientation and Amnesia Test

In the late post-acute or chronic recovery period (3 or more months post-injury), a referral to a neuropsychologist may be useful in helping determine the nature and extent of relatively permanent cognitive problems and the effect these are likely to have on daily functioning. **Neuropsychological evaluations** generally take 2-5 hours to complete, and include measures of general intelligence, attention and concentration, learning and memory, language, visuospatial abilities, and executive functions. Often an evaluation of psychological and personality functioning is conducted as well to help assess issues of depression, anxiety, and adjustment to the brain injury. If a neuropsychologist is not available within one's facility, **speech pathologists** often can complete similar cognitive evaluations of attention, memory, language, and thinking abilities.

Recovery from Cognitive Problems

In the weeks to months after return to consciousness many aspects of cognition improve, some quite rapidly. In fact, in the first few months rapid change is considered the rule. The most typical sequence of reorientation is for person, place, and time in that order. Early after the injury, the period of retrograde amnesia (loss of memories immediately prior to the accident) may range from hours to months. Retrograde amnesia for early life events typically is considered to reflect psychological rather than neurological factors. As the individual recovers, the length of retrograde amnesia typically shrinks to a few hours or a couple of days prior to the accident.

Recovery in performance on intellectual tests is seen over time. Activities that have a large attentional component tend to improve quickly and reach a plateau within the first year after injury. Activities, such as new learning and memory tend to improve over time, but often do not reach normal levels. Many patients with **moderate injuries** can and, for the most part do, function independently. Many return to work or resume their usual responsibilities. Yet, they tend to differ from neurologically intact persons and from what they were prior to injury, in that most continue to experience cognitive and emotional/personality (see section 6) problems associated with frontal and/or temporal damage. Frontal lobe problems, in particular, tend to show up in subtle ways. Patients display diminished initiative and spontaneity and no longer plan for non-routine activities.

Few patients with **severe injuries** return to work or even to fully independent living. Particularly common, persistent long-term cognitive problems include cognitive and motor slowness, poor memory, and executive control dysfunction. Fluctuations in performance are common in patients with impaired executive functions, which is considered to represent these patients' lack of internal stability and self-regulation. Quality of life as reflected in patient and family satisfaction and distress also tends to be increasingly compromised with increased severity of injury. Severely head injured patients have been found to have a four to five time greater likelihood than that of the general population of developing dementia.

After the first year post-injury, improvement in cognitive performance becomes increasingly gradual and more reflective of compensatory strategies than recovery of function. Common cognitive problems noted after moderate to severe TBI persist with no consistent, significant change after a two-year period. Such difficulties contribute more than physical changes to eventual level of disability.

Points to Remember

- **Cognitive functioning improves, but usually reaches a plateau within the first year after injury, at which time some ongoing problems typically remain in the individual with a moderate to severe TBI**
- **Many patients with moderate injuries can and, for the most part do, function independently**
- **Few patients with severe injuries return to work, or even to fully independent living**

Medication Management of Cognitive Deficits

To date, there is no proven pharmacologic intervention to treat and potentially improve post-TBI cognitive deficits. Different mechanisms causing brain injury (diffuse axonal injury, focal or multifocal frontotemporal lobe damage, or anoxic injuries) can cause varied neurochemical imbalances. These imbalances occur in various neurotransmitter systems. Cholinergic, noradrenergic, dopaminergic, serotonergic, and gabaminergic neurotransmitter pathways ascend and descend between the frontal cortex and subcortical structures, including the brainstem. In individuals with TBI a number of medications have been studied which adjust neurotransmitter imbalances. Many agents are still being studied in the animal model, although some have been tried in case studies or even groups of patients with various medical diagnoses. This section will review the literature in the following categories:

1. neurostimulants,
2. antidepressants;
3. dopaminergic agents;
4. acetylcholinesterase inhibitors; and
5. other agents.

Neurostimulants

(Methylphenidate (Ritalin), dextroamphetamine, pemoline)

Methylphenidate (MPH), now used primarily in attention-deficit/hyperactivity disorder (ADHD), is a central nervous system stimulant, which binds to dopamine transporters in the pre-synaptic cell membrane. It blocks the reuptake of dopamine, causing increased extracellular dopamine levels. It also has effects on norepinephrine reuptake, and binds to serotonin transporters.

One of the initial reports of the use of methylphenidate in brain injury was a case report of a severely brain injured individual who had a marked cognitive improvement with methylphenidate (Weinberg, Auerbach, & Moore, 1987). The patient was then placed on lecithin, which did not have any effect on the patient's status, and subsequently physostigmine. The patient did have improvement with physostigmine, which was lost after the medication was discontinued.

Since then, there have been mixed reports addressing the effects of methylphenidate on memory and attention after TBI. Two review articles have been published (Challman & Lipsky, 2000; Kraus, 1995), reviewing 11 studies. These studies were primarily uncontrolled, but the authors concluded that neurostimulants have some utility for certain types of mood, behavior, and cognitive symptoms following brain injury. Promising studies include one subacute administration study of MPH in 23 moderate brain injured patients (Plenger et al., 1996). Disability Rating Scale, tests of attention, memory, and vigilance were performed at 30 and 90 days after discontinuation of the MPH or placebo. MPH affected the rate of recovery (with improvement at 30 days) but not the ultimate level of recovery (at 90 days). Kaelin et al., (1996) studied the effects of MPH on attention and functional outcome in acutely injured TBI patients and found improvement in digit span, mental control, and symbol search when on MPH as compared to the scores pre-MPH administration. Benefits were sustained 1 week after discontinuation of the medication. In a double-blind, placebo-controlled TBI study, MPH improved mental processing, but had no effect on orienting to distractions or sustaining attention (Whyte et al., 1997). Finally, in an animal model, rats have shown improvement in water maze performance after TBI after treatment with MPH for 18 days (Kline, Yan, Bao, Marion, & Dixon, 2000).

Antidepressants

(Tricyclic antidepressants (TCA's))

The tricyclic antidepressants, which seem to have the best potential for the brain injury population, are those with a stimulant effects, due to their adrenergic profile such as protriptyline, nortriptyline, and desipramine. Wroblewski et al., (1993) reported that protriptyline was effective in a series of 8 case reports as an activating/stimulant medication in patients with head injury. More recently, at least in part due to their more limited side effects, attention has been focused on the use of SSRI's in head injury deficits.

SSRI's

(Sertraline (Zoloft) and Fluoxetine (Prozac))

Fann et al., (2001) reported improvements with sertraline in recent verbal memory, recent visual memory, and general cognitive efficiency in patients with depression after mild TBI. However, Meythaler et al., (2001) failed to show any significant improvement in arousal and alertness in 11 individuals with severe TBI.

Dopaminergic Agents

(Amantadine (Symmetrel), Seligiline/Deprenyl (Eldepryl), Bromocriptine (Parlodel), Pergolide (Permax), L-Dopa/Carbidopa (Sinemet), Ropinirole, and Pramipexole)

Amantadine (Symmetrel) is a dopamine agonist that has suspected pre-synaptic and post-synaptic actions. Nickels and colleagues (1994) retrospectively reviewed 12 TBI patients treated with amantadine; of these, 10 exhibited cognitive and/or physical improvement while on amantadine. In contrast, in a double-blinded, placebo controlled study of amantadine in

acute TBI patients there were no significant improvements on cognitive measures (Schneider et al., 1999).

Selegiline or L-Deprenyl (Eldepryl) is a selective MAO-B inhibitor that has antioxidant and cellular protective mechanisms as well. It is commonly used to treat the symptoms of Parkinson's Disease. In a rat TBI model, selegiline resulted in improved cognitive performance, but not motor improvement (Zhu et al., 2000).

Bromocriptine (Parlodel) is a directly acting dopamine agonist, which has been demonstrated to improve performance on tasks of executive function and dual-task performance in a double-blind, placebo-controlled study with 24 TBI subjects (McDowell et al., 1998). Similarly, it has been shown to improve working memory (water maze performance) in rats with TBI (Kline et al., 2002).

The effects of L-Dopa/carbidopa have been studied in 12 patients with diffuse brain damage (Lal et al., 1988). All subjects had reached their plateau in their rehabilitation programs for at least 2 weeks before the medication was started, but after treatment showed some cognitive and behavioral improvement of varying degrees.

Cholinesterase Inhibitors

(Physostigmine, Donepezil (Aricept), Rivastigmine (Exelon), and Galanthamine (Reminyl))

Although a number of case studies have reported potential benefits of Donepezil (Aricept) following TBI (Taverni et al., 1998; Masanic et al., 2001; Bourgeois et al., 2002), no controlled studies exist to date.

Other Pharmacological Agents

There are a number of other agents, which are actively being studied for potential recovery post-brain injury; many are still in the stages of being studied in the animal model. The nootropics (nifiracetam, piracetam, pramirecetam) potentially increase the glucose and oxygen consumption in the ischemic nervous tissue and increases blood flow through cerebral terminal vessels. McLean (1991) reported that pramirecetam improved memory in young males with head injury or anoxic injury.

In conclusion, there are a number of pharmacologic agents that show potential to improve cognitive sequelae post-brain injury. However, the number of clinical trials for these agents in head injury has been limited to date. With the newer, more specific neuronal agents now available, there is potential for targeted use of such agents in both acute and possibly subacute stages of brain injury.

Point to Remember

- **To date, there is no proven pharmacologic intervention to treat and potentially improve post-TBI cognitive deficits**

Rehabilitation/Cognitive Therapy/Environmental Management of Cognitive Deficits

Most individuals with moderate to severe brain injury will have completed an inpatient rehabilitation program, during which they will have received cognitive therapies either from speech therapists or occupational therapists. These therapies help individuals learn and practice various compensation strategies to deal with residual cognitive problems. However, if a patient has not received inpatient rehabilitation, they may not have been provided the education and compensatory training necessary to help them achieve their maximal level of cognitive and functional independence. In addition, if their environmental or social situation has changed (moved to a new residence, divorced, death of a caregiver), individuals may well benefit from a trial of additional cognitive therapy. In addition, there are patient-centered educational materials, which may be useful (e.g., <http://www.biausa.org/word.files.to.pdf/good.pdfs/roadToRehab3.pdf>).

Impaired attention/concentration

In the post-acute or more chronic phase of recovery, ongoing problems with attention and concentration are best addressed through education and the development of compensatory strategies. Patients often report problems in high demand situations where they must process several different ongoing aspects of the environment (answering the telephone and taking a message, while working on paperwork). They can benefit from education that their ongoing problems in attention and concentration may well result in difficulty following tasks through to completion and frequent errors, especially in a stressful work environment. Useful management tips include:

- pace yourself
- perhaps work more slowly
- frequently check work for errors
- work on one task at a time
- clear away materials from one task before beginning work on another
- take regular breaks to refocus attention
- work in a quiet environment with minimal noise and few interruptions
- when reading, allow time to re-read or re-study sections
- if problems persist, outpatient cognitive rehabilitation may be beneficial

Learning and memory difficulties

The impact of learning and memory difficulties on daily functioning, school, and work performance can also be reduced through the use of compensatory strategies. Examples of memory strategies that are often useful include:

- using a daily organizer/calendar to record appointments, daily tasks, important telephone numbers, etc.
- allowing extra time to learn material
- breaking material to be learned into smaller portions

- repeatedly rehearsing material to be learned
- designating specific places for frequently misplaced items (e.g., keys, purse/wallet, etc.)
- using a pill box to organize medication schedules or a timer to signal medication times
- making “to do” lists and posting them in a designated place where they can be easily seen
- making checklists of tasks to be accomplished everyday and marking them off as they are completed
- making a checklist of procedures used on the job
- writing down important information (e.g., phone messages)
- taking careful notes and/or making outlines when reading
- if visual memory is substantially better than verbal memory, using visual imagery and symbols may aid in learning new material
- tape recording class lectures and replaying them slowly to allow for effective note taking at a later time

If a patient has not had a course of cognitive rehabilitation during the more acute phase of treatment, for persisting and more severe learning and memory problems, outpatient cognitive rehabilitation should be considered. Cognitive retraining has been shown to be beneficial, particularly in higher-level patients. Depression and anxiety can also impact learning and memory performance. Clarifying the precipitating causes of impairments can help determine the most appropriate intervention — referral to speech pathology for cognitive rehabilitation or referral to psychology or psychiatry for treatment of depression, anxiety, or adjustment issues.

Executive control dysfunction

Many of the same tips and compensatory strategies that assist with attention and memory problems are also useful in managing executive difficulties. These include using strategies and extra time to manage complex tasks.

- working on one task at a time
- working more slowly and carefully
- checking work for errors
- breaking tasks down into small steps, and then working through each small step to complete the larger task
- making checklists of tasks to be accomplished everyday and marking them off as they are completed
- using a pocket organizer of some kind to keep track of what needs to be done
- making “to do” lists and post them in a designated place where they can be easily seen

Communication problems

For patients with slowed communication and/or word-finding difficulties, patients should be encouraged to speak slowly and allow time to formulate their thoughts before speaking. The pressure to speak in a social situation can be reduced by allowing more time for listening to others’ conversations and developing active listening skills.

When referring patients for **cognitive rehabilitation**, the following considerations should be taken:

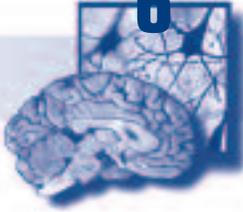
- Many third party payors will not reimburse for cognitive rehabilitation; referrals may need to be made for speech therapy services in which cognitive therapies are embedded
- Cognitive remediation should be embedded into tasks that are directly relevant for the particular patient
- Case management (http://www.va.gov/health/rehab/TBI_Case_Managers.htm; (Section 10 Case Management)) is absolutely essential to be able to tease out what is overkill, and determine what treatments are efficacious
- Given that most patients' ultimate goal is to return to work or return to effective performance, outpatient rehabilitation programs with a vocational track might be most beneficial
- Lastly, if a patient with a TBI expects to be able to return to pre-injury functioning levels at the end of cognitive treatment, then the entire endeavor of cognitive remediation may be set up for failure

Points to Remember

- **Most individuals with moderate to severe brain injury will have completed an inpatient rehabilitation program, during which they will have received cognitive therapy**
- **In the chronic phase of moderate to severe TBI, a new trial of compensatory cognitive training may be helpful if the environmental or social situation has changed resulting in a decrement in functional independence**
- **In the post-acute or more chronic phase of recovery, ongoing cognitive problems are best addressed through education and the development of compensatory strategies**

Role of the Primary Care Physician in the Management of Cognitive Deficits

In the post-acute or chronic phase, the primary care physician is most likely to coordinate the ongoing care of the individual with a TBI. Common symptom complaints are likely to be memory problems, behavioral changes including irritability and perhaps increased aggression, and difficulty modulating affective responsiveness. Initial consults to neuropsychology for evaluation of the cognitive complaints if they have not previously been evaluated and to psychiatry for medication management are appropriate. Other referrals may be indicated as discussed above, depending on prior medical workups and previous rehabilitation interventions. Providing patients and families with education material (e.g., Guidelines on TBI <http://www.guideline.gov/search/searchresults.aspx?Type=3&txtSearch=brain+injury&num=20>) and supportive listening are also likely to be useful interventions.



Post-acute/Chronic Sequelae: Emotional and Behavioral Problems

Learning Objectives

1. Recognize common neuropsychiatric sequelae of TBI.
2. Describe the interplay of cognitive and emotional problems following TBI.
3. Distinguish between similarly presenting neurological versus psychological symptoms and problems.
4. Describe the assessment data needed to prescribe efficacious treatments for TBI.
5. Describe symptoms and problems that may require referral to mental health professionals.
6. Describe basic pharmacological treatment for these emotional and behavioral sequelae.

Introduction

Emotional difficulties and behavioral problems are common following brain injury (Morton & Wehman, 1995; Sohlberg & Mateer, 2001). Furthermore, premorbid psychiatric problems, such as impulse control difficulties, substance abuse, and family problems increase the risk for head injury (Bennett & Raymond, 1997; Hanks, Temkin, Machamer, & Dikmen, 1999; Kersel, Marsh, Havill, & Sleight, 2001; McCauley, Boake, Levin, Contant, & Song, 2001; Sarapata, Herrmann, Johnson, & Aycock, 1998), and are unlikely to improve following head injury. Therefore, following a brain injury one has to deal with both pre-injury emotional/behavioral problems, typically compounded by the emergence of new post-injury behavioral problems.

Damage to the frontal lobes are common in motor vehicle accidents and can cause specific behavioral problems including difficulty tolerating frustration or higher levels of stimulation. This in turn can lead to agitation, cursing, aggression, and potentially destructive behavior. In addition, behavior problems can be the result of an impaired ability to process information or understand situations accurately. Finally, post-TBI behavior problems also can occur because individuals become fatigued much more easily, increasing irritability and poor frustration tolerance. Emotional sequelae following a brain injury include increased anger, lowered frustration tolerance, increased anxiety, depression, and low self-esteem.

Behavioral and emotional problems may be long-lasting and take a tremendous toll on family members, caregivers, and friends. In addition,

they tend to lead to social problems including over-dependency; loud, tangential, or incessant talking; immature behavior; inappropriate use of humor; obsessive eating; inappropriate sexual behavior; poorly controlled spending; self-centeredness; and general difficulty appropriately reciprocating in social interactions. Divorce is very common and frequently accompanied by a loss of social group membership for the injured person, further resulting in long-term obstacles for a successful recovery.

For persons with mild brain injuries (also referred to as post-concussive disorder), many emotional problems may go unreported, either altogether or not until the symptoms have produced a significant negative impact on the person's life. Sleep disturbance following concussions is particularly unappreciated as an event following mild injuries (Kelly, 2002). These sleep problems can worsen other problems that frequently occur in mild head injuries such as inattention, poor concentration, depression, relationship difficulties, and decreased problem solving abilities. People with mild injuries frequently have been found to change or lose their jobs within six months of the injury (Gasquoine, 1997) and particularly are at risk for not understanding why they now are having problems in their life (Prigatano & Schacter, 1991).

Denial of problems by injured persons and/or their families often lead to difficulties with getting services and maintaining the injured person's involvement or willingness to participate in the rehabilitation process. Often, problems are discovered beyond the time when health care providers can effectively prevent major social losses of family and friends, thus losing the "window of opportunity" for remediation of some problems.

When mildly injured persons are identified in a timely fashion, their rehabilitation often will eventually approximate their premorbid status. Indeed, simply being identified has the effect of anxiety relief that comes from being able to attribute one's problems to the appropriate source (i.e., the brain injury). Treatment for anxiety and depression are frequently needed in addition to the rehabilitation process that addresses their deficits.

Resources (e.g., emotional, physical, financial, social, etc.) are much more likely to be exhausted in the recovery of someone with a more severe injury. The family burden can be too much for many families, resulting in family disintegration and the loss of this important resource that plays a major role in obtaining a successful outcome for the injured person.

The information contained in this section is intended to provide health care providers with sufficient information to understand when a referral might be useful to psychology or psychiatry.

Points to Remember

- **Damage to the frontal lobes are common in motor vehicle accidents and can cause specific behavioral problems or changes in personality**
- **Behavioral and emotional problems may be long-lasting and take a tremendous toll on the family system**

Interplay of Cognitive and Emotional Problems

Cognitive deficits (e.g., mental slowness, memory problems, inattention, poor concentration, impaired problem-solving skills, etc.) clearly impact emotional reactions. Impaired self-awareness (i.e., difficulty seeing one's strengths and deficits) is common in moderate to severe injuries and typically results in the person's having unrealistic goals and expectations for him/herself. Difficulty arises when the injured person cannot see how socially inappropriate their goals or behaviors are. The continued lack of successful social interactions, in part due to the interplay of cognitive deficits with emotions, results in ongoing issues with anxiety, depression, guilt, shame, anger, fear, decreased self-esteem, and an erosion of self-confidence.

Sometimes, emotional symptoms are the direct result of neurological damage rather than psychological reactions secondary to having a brain injury, despite similar clinical appearance. Judd (1986) has illustrated this point well in the list of psychogenic vs. neurologic etiologies listed below in **Table 1**.

Psychogenic/Psychiatric Symptoms	Neurogenic Symptoms
Denial	Anosognosia (lack of awareness of impairment)
Anger and irritability	Frustration, catastrophic reaction, reduced information
Depression	Lack of initiative, impaired emotional expressiveness, lowered crying threshold, fatigue
Rigid compulsive/hypervigilant	Distractibility, inability to deal with more than one task at a time, dependence on external controls
Emotional lability	Lability of emotional expressiveness (not the underlying feeling state)
Social withdrawal	Lack of initiative
Sense of futurelessness	Impaired planning
Thought disorder	Aphasia, anomia, or confusion
Personality or conduct disorder	Impulsivity, social disinhibition

Table 1

Points to Remember

- **Impaired self-awareness is common in moderate to severe injuries and often results in the person's having unrealistic goals and expectations**
- **Similar appearing emotional and behavioral problems can be the result of either neurological damage or psychological reactions secondary to having a brain injury**

Predictors of Outcome/Adjustment to Disability

Predicting eventual outcomes and overall adjustment to living with a chronic disability involves a complex interplay of multiple factors. These factors include premorbid status, severity of brain injury, the ability to return to work or school, and the amount of available resources from which to call upon (including financial, emotional stability, level of family support, stability of social relationships, specific deficits, etc.).

Assessment

Obtaining collateral information from family or caregiver is essential due to the frequent lack of self-awareness, denial of impairments, or language/communication disorders common following TBI.

Sample structured interview questions to get at these issues include:

- How has your injury changed your life?
- Have you experienced changes in your mood such as sadness, depression, or anxiety?
- During the times you have felt sad, do you ever feel like harming yourself or anyone else? (If yes to suicidal question, assess current feeling and thoughts regarding active suicidality. Do you have a plan; do you feel like killing yourself right now?)
- Have you experienced changes in frustration level or anger control?
- Have you experienced increased difficulties in interpersonal relationships with your spouse or friends?
- What resources (e.g., family, friends, doctors) have been the most helpful for you?

Consider a referral to psychology if a more detailed diagnostic interview, psychological testing, and/or psychotherapy appear to be warranted. Consider a referral to psychiatry for a diagnostic interview, medication management of emotional or behavioral difficulties, and/or psychotherapy. If it is uncertain whether the emotional/behavioral problems are neurologic versus psychogenic in origin, then an additional referral to a neuropsychologist or neurologist may be indicated. The more information provided to consultants about the problems for which one is seeking analysis and recommendations, the more likely the referral would provide useful information and a targeted answer that will assist future decision-making.

Point to Remember

- **Obtaining collateral information from family is essential due to the frequent lack of self-awareness or denial of impairments common following TBI**

Therapies to Address Emotional and Behavioral Issues

The main goal of this section is to increase understanding of the common neurobehavioral disorders affecting TBI patients and the current recommended treatment strategies. General principles apply to the management of all psychiatric sequelae and these include:

- Interdisciplinary approach
- Psychotherapy/behavioral management
- Pharmacotherapy
- Caregiver education/support

Common non-pharmacological behavioral interventions

Cognitive-behavioral interventions

Cognitive-Behavior therapy includes teaching self-monitoring, self-instruction, and relaxation techniques. Patients are often encouraged to track emotions and use reminders such as “keep cool” or “chill out” before reacting to a frustrating situation. Evaluating the situation before responding is also recommended. This self-evaluation can be assisted by teaching a series of questions to ask oneself before acting:

1. What is the problem?
2. What would I typically do?
3. What would happen if . . . ?
4. What should I do?
5. What will result from a different course of action?

Behavior or cognitive-behavior therapy may be particularly useful for specific behavior problems such as:

- Irritability and low frustration tolerance
- Anger management
- Adjustment difficulties
- Depression
- Anxiety
- Inappropriate or disinhibited behaviors
- Social skills deficits (including socially inappropriate behaviors such as loud talking, “overly friendly” touching, invasion of personal space of others, reading social/emotional cues in conversations, conflict resolution skills, etc.)

Supportive Psychotherapy

Supportive therapy helps address issues of poor self-esteem, confusion, loss of relationships, unrealistic goal setting, and grief responses to impairments from the brain injury (including physical, emotional, social, personal, and family).

Group Therapy

Group therapy can be effective in addressing:

- Social/emotional interactions
- Support and education regarding brain injury related problems

Family or Marital Therapy

Family or marital therapy would help families deal with the common post-injury issues such as change in family roles, burden of care of the individual, relationship conflicts such as hypersexuality of injured member, loss of family independence, expenditure of family resources for the injured person, etc.

Spiritual guidance

Is based upon their specific belief systems to provide hope, comfort, support, etc.

Education

Providing education regarding brain injury, what to expect, and how to handle difficult circumstances is a potentially powerful intervention. For the achievement-oriented patient, providing education that impairments are related to the injury, and that pushing oneself harder may not result in success. Pushing oneself too hard can result in stress and increased fatigue, which can further intensify impairments. Patients can be encouraged to allow time for recovery and to break goals into smaller, more manageable steps. Specific information about available resources is also helpful:

- Contact information for state and national brain injury organizations
- Information about local self-help and support groups

To secure these types of services for brain injured clients referrals can be made to psychology, psychiatry, or various mental health service organizations.

Points to Remember

- **There are multiple psychological interventions that can be helpful in dealing with the emotional and behavioral problems following TBI**
- **Information and education to patients and families are potentially powerful interventions**

Psychopharmacological Treatment - Post-Acute Symptom Management

Despite the lack of randomized controlled trials to guide pharmacological treatment of the psychiatric sequelae of TBI, the use of pharmacologic agents has become standard practice in the rehabilitation community. The psychiatric disturbances associated with TBI do not fit easily into DSM-IV diagnostic categories. Therefore, medication management is typically symptom-based, not diagnosis-based (Silver, Yudofsky, & Hales, 1994).

The pharmacologic approach is based on the severity and acuity of the predominant symptoms while taking into account other factors. These include other medical and/or psychiatric conditions, the hypothesis regarding the pathophysiology of the symptoms, and medication side effect profiles. The rule universally applied to the elderly of “start low and go slow” applies to the TBI population as well. In addition, polypharmacy should be minimized. **Table 2** (next page) adapted from Arciniegas, Topkoff, & Silver, (2000) provides useful information about medications potentially useful in treating various symptom profiles.

Medication	Depression	Affective lability Or Irritability	Mania	Psychosis	Agitation or Aggression	Anxiety	Apathy	Cognition	Risk of Adverse Event
Nortriptyline	++	+	-					-	+
Desipramine	++	+	-					-	+
Amitriptyline	+		-		+++			---	+++
Protriptyline	+	+	-				++	-	+
Fluoxetine	+++	+++	-		++				++
Sertraline	+++	+++	-		++				+
Paroxetine	+++	+++	-		++				+
Lithium			+		++			-	+++
Carbamazepine			++		+++			--	++
Valproate		++	+++		+++				+
Benzodiazepines						+		---	+++
Bupropion	+	++			+	+			+
Typical antipsychotics				++	+			--	+++
Atypical antipsychotics				+++	+			-	+
Methylphenidate	++	++			++		++	++	
Dextroamphetamine	++						++	++	
Amantadine	+	++			++		+	+	
Bromocriptine			-	-			++	+	+
L-dopa/carbidopa			-	-			+	+	+
Beta blockers	--				+++		-	-	-
Donepezil								++	+

Only medications for which there are reports in the literature are listed here. Additional adverse events include increased likelihood of seizures (antidepressants), neurotoxicity (lithium), impaired cognition (mood stabilizers and benzodiazepines), and psychosis (bromocriptine and L-dopa/carbidopa).

Table 2

+ Indicates potential benefit; - indicates potential adverse effects.

Acute Agitation/Aggression

This problem is seen most commonly during the early phase of recovery from moderate to severe brain injury during Rancho Level IV and can severely affect the patient's ability to participate in therapy. Post-traumatic agitation is a diagnosis of exclusion. This means that provoking or aggravating medical (infection, pain, drug withdrawal), neurological (seizure, hydrocephalus, etc.), or pharmacological factors should be investigated and treated.

Medications that should be used with caution in TBI patients include benzodiazepines, antispasticity drugs, typical antipsychotics, narcotics, H2 blockers, certain anticonvulsants, anti-hypertensives, clonidine, and steroids as they can all contribute to agitation or restlessness. Nevertheless, benzodiazepines and antipsychotic medications may be useful for the rapid resolution of acute agitation. However, they should be used for the shortest period of time at the lowest possible dose. Benzodiazepines are known to impair memory/attention, prolong post-traumatic amnesia, and can result in a paradoxical reaction while typical antipsychotics (haldol, thorazine) can lower the seizure threshold, delay motor recovery, and impair cognitive recovery. Valproate, carbamazepine, and beta-blockers are the medications of choice for acute agitation, followed by amantadine and the SSRIs.

Chronic Agitation/Aggression

Treatment of chronic agitation includes using atypical antipsychotics, anticonvulsants, serotonergic/noradrenergic antidepressants, buspirone, and beta-blockers (Yudofsky & Hales, 2002).

Atypical antipsychotics (i.e., risperidone and quetiapine) are used for agitation as they have a much lower incidence of extrapyramidal side effects and tardive dyskinesia as compared to typical antipsychotics and are the drugs of choice for treatment of agitation that presents with psychotic symptoms. Side effects include weight gain, hyperglycemia, hypotension, and QTc prolongation.

Commonly used anticonvulsants for the treatment of agitation include carbamazepine, (Tegretol) and Valproate sodium (Depakote).

Antidepressants can also be used for the treatment of chronic agitation. Trazodone can help normalize sleep-wake cycles and has side effects including sedation, orthostatic hypotension, and priapism (incidence < 1%). SSRIs and SNRIs are helpful in treating agitation associated with affective lability and sexually inappropriate behavior. These medications are well tolerated but have delayed benefits. Tricyclic antidepressants (i.e., Elavil & imipramine) are generally avoided due to anti-cholinergic properties and lowering of the seizure threshold. Beta-blockers are useful in the treatment of agitation but use is limited by cardiac side effects. Less lipophilic beta-blockers (nadolol) are preferable due to fewer CNS side effects (Brooke, Patterson, Questad, Cardenas, & Farrel-Roberts, 1992). Buspar, a novel anxiolytic, is non-addicting and does not affect cognitive function. Psycho-stimulant medications (i.e., Methylphenidate) have also been used for the

treatment of agitation as they can improve level of arousal and cognitive function.

To date there is no FDA approved drug for agitation or aggression. Most of current treatment practice has been modified from psychiatric research with non-TBI patients.

Mood Disorders/Apathy

Depression occurs in 25-50% of TBI patients and risk factors include prior psychiatric history, prolonged PCS, left hemisphere damage, and psychosocial factors (loss of social support, work, etc.; Jorge, Robinson, Arndt, Starkstein, Forrester, & Geisler, 1993). Studies report an increased risk of suicide following TBI (Yudofsky & Hales, 2002). Treatment for depression following TBI includes supportive psychotherapy and pharmacologic treatment. Drug choice is guided by side effect profiles but dictated by clinical presentation and history. SSRIs and SNRIs are most frequently used. Mania and emotional lability are treated similarly. Mania occurs in 4-10% of TBI patients and risk factors include right hemisphere damage and family history of mood disorders. The treatment of choice for mania in the TBI population are the anticonvulsants.

Apathy presents as a lack of initiation, motivation, and pleasure. Depressed mood, feelings of hopelessness or worthlessness, identifiable stressor, and changes in sleep are generally not present (Rao & Lyketsos, 2000). Treatment of choice is use of dopaminergic agents such as amantadine and neurostimulants.

Anxiety Disorders

TBI often impairs the ability to understand or adapt to external and internal stimuli. Approximately 29% of TBI patient have measurable levels of anxiety (Yudofsky & Hales, 2002). This occurs most commonly with lesions involving the right orbital-frontal region. Treatment should include a review of all factors that might possibly play a role in provoking or maintaining symptomatology such as work, family dynamics, and environment. Medications that can be effective include SSRIs, buspar, SNRIs, and propranolol.

Disinhibited Behavioral Control Disorders

Personality changes frequently occur following TBI and empirically we see this as one of the most difficult adjustment issues. These are frequently a consequence of frontal and temporal lobe damage and treatment depends on the subtype of the personality change. DSM-IV classifies the following personality change subtypes: labile, disinhibited, aggressive, apathetic, paranoid, combined, and unspecified. The disinhibited personality subtype may respond to anticonvulsant or SSRI medications, whereas the apathetic type may respond to psychostimulant medication.

Psychotic Disorders

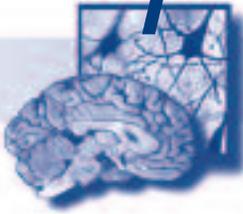
The incidence of post-traumatic psychosis ranges for 0.7 to 20% (Ahmed & Fujii, 1998). Risk factors include left hemisphere injury, specifically left temporal lobe damage, and can occur early (during PTA) or after a long latency. Interestingly, there is a higher incidence of head trauma in schizophrenic patients. Treatment includes the use of atypical antipsychotics or anticonvulsants.

Points to Remember

- **To effectively treat neuropsychiatric disorders following TBI, one must be sure to rule out confounding medical and neurological factors as well as alcohol or drug induced symptoms**
- **Polypharmacy should be minimized. An attempt to minimize unnecessary medications should occur prior to initiating new medications to treat symptoms. Familiarity with side effects is crucial, and helps guide selection of medication**
- **Family/Caregiver education and support are key for the effective treatment and management of neuropsychiatric disturbances following TBI**

Role of the Primary Care Physician

In the post-acute phase, TBI patients and their families are most likely to present to their primary care physician with various complaint. The family may well report problems with irritability, anger control, and disinhibition, while the patient is more likely to report general life dissatisfaction, poor mood, or low self-esteem. Once the history of TBI has been clarified and the onset of the reported symptoms dated to the TBI, the primary care physician may well consider referrals to psychiatry, psychology, or neuropsychology for further evaluations and treatment intervention. However, the primary care physician is likely to remain the primary provider and is likely to follow the patient on their medication regimen once an effective regimen is determined with psychiatry.



TBI in the Elderly and aging with TBI

Learning Objectives

1. Describe the unique aspects of traumatic brain injury (TBI) epidemiology in the elderly.
2. Describe strategies to prevent falls (and TBI) in the elderly.
3. Explain the influence of age on outcome after TBI.
4. Identify indications for head CT after TBI in the elderly.
5. Describe the relationship between physiological changes in the elderly and adverse cognitive medication side effects.
6. Describe the link between TBI and Alzheimer's Disease (AD).

Epidemiology of TBI in the Elderly

Rates of ambulatory care visits for TBI to physician offices, outpatient departments, and emergency departments are higher in the elderly, especially in those greater than 75 years old (Englander & Cifu, 1999). Given current demographic and longevity trends in the United States, the number of older adults with TBI should increase over the next several decades. Individuals between the ages of 15 to 25 and those 75 and older are at highest risk for TBI of all age groups. Whereas motor vehicle collision is the leading cause of TBI in adults, falls are the leading cause of TBI in the elderly (Kraus & Nourjah, 1988; Thurman et al., 1999). Being struck as a pedestrian is also a more common etiology in the elderly compared with other adults. Men account for a higher percentage of TBI in the young elderly group. Whereas in those, over 80 years old, greater female longevity leads to a female preponderance for a TBI. (Englander & Cifu, 1999).

Prevention of TBI in the Elderly

Despite the fact that falls is the most important cause of TBI among the elderly, relatively little research has been done on fall prevention. Approximately 30% of persons over 65 fall each year and may sustain injuries requiring hospitalization (Englander & Cifu, 1999). The cause is usually multifactorial, with weakness, poor balance and medication side effects frequently contributing. A recent controlled trial of a multiple risk factor intervention strategy showed a reduction of falls and head trauma in subjects compared with controls using gait training by physical therapist, assistive devices, and balance and resistive exercises (Tinetti et al., 1994).

Another randomized study of elderly persons living in the community who presented to the emergency department after a fall found that a program of patient evaluation, referrals, and home visit by occupational therapists (n = 183) resulted in significant improvements in outcome at 12 month follow-up compared to usual treatment (n = 213). The risk of falling was significantly reduced; odds of hospital admission lowered; and the Barthel score declined less (Close et al., 1999).

Prognosis

Age is an important factor in functional and cognitive outcomes after TBI. In short, the probability of poor outcome increases with advanced age (Braakman et al., 1980; Jennet et al., 1979; Stablein et al., 1980; Chestnut et al., 2000). Possible explanations include reduced reserves with which to tolerate brain injury or a more fragile physiologic status in the elderly resulting in a more destructive injury (Rothweiler et al., 1998). Despite slower recovery rates and longer lengths of stay, the majority of elderly TBI survivors undergoing rehabilitation achieve functional improvement and community discharges (Cifu et al., 1996).

When assessing the impact of TBI on the older adult, one must take into account significant accumulated chronic comorbidities (e.g., arthritis, cardiopulmonary, diabetes, atherosclerosis, renal failure, cancer). In addition, one needs to consider chronic premorbid conditions that could reduce cerebral functional reserve at baseline (cerebrovascular disease, chronic alcoholism, advanced liver or renal disease, dementia) and potentially impede or prolong the recovery process. Another impediment to rehabilitation is higher levels of psychosocial limitation seen with advanced age (Rothweiler et al., 1998).

Assessment of TBI in the Elderly

Severe trauma in individuals over 70 is 6 times more likely to cause intracerebral lesions than severe chest, abdominal, or pelvic injury. Older individuals are more likely to have “complicated” mild TBI, a term used for patients meeting clinical criteria for mild TBI and who also have abnormal findings on head CT. Thus, when an individual with mild TBI presents to the clinic or emergency department (ED), age should be considered in the decision whether to obtain a head CT. Based on retrospective review of 1448 patients seen in ED with mild TBI, Borczuk (1995) recommends CT scan for patients after TBI when age is greater than 60 years, or if any of the following is present: focal neurologic deficit, evidence of basilar skull fracture, or cranial soft tissue injury.

For the elderly TBI survivor with significant functional limitations, a full medical rehabilitation evaluation by a physiatrist is strongly recommended when available. Lower physiologic reserves make the recovery process more challenging in the elderly. An estimation of physiological reserve allows

more specific structuring of the rehabilitation program, and is made possible by delineating preinjury activity level and pertinent acute and chronic medical issues. Impairments (e.g., hemiparesis, dysphagia, incontinence, and cognitive deficits) and functional deficits (mobility status, activities of daily living) should likewise be fully delineated. The evaluation should also seek to identify any of the potential medical complications after TBI, which will be discussed further below.

Management: Behavior and Cognition

Due to the physiological and pathological changes that accompany aging, older adults experience more frequent and more severe cognitive impairment when compared to younger adults with similar severity of TBI (Rosenthal, Kreutzer, Griffith, & Pentland, 1999). This is primarily due to lower cognitive reserve capacity and a higher incidence of premorbid dementia. Significant premorbid memory deficits are present in nearly 10% of community-dwelling older adults (Beard et al., 1995), and greater than 20% of those over 85 (Skoog et al., 1993).

Older individuals with TBI also have slower rates of drug metabolism and excretion, which creates a greater propensity for cognitive side effects from medications including agitation, somnolence, and increased confusion. To complicate matters, most elderly are chronically on regular prescription medications, with more than 90% of the entire population over age 65 taking at least one prescription medication daily, and most taking two or more (Chutka et al., 1995). After TBI, thorough review of both prescription and non-prescription medications is imperative, and all non-essential medications should be stopped or tapered. General principles for TBI pharmacology should be applied. For example, acetaminophen is the preferred medication for pain syndromes; proton-pump inhibitors are favored over H2 blockers for peptic ulcer disease or reflux; beta-blockers that cross the blood-brain barrier should be avoided; benzodiazepines and metoclopramide should especially be avoided; and low-dose trazodone is the preferred medication for insomnia.

Although major depression is less common, depressive symptoms are more common in elderly TBI patients compared to younger adults (Rosenthal et al., 1999). When pharmacological treatment of depression is indicated, SSRIs are the recommended first line in elderly patients with TBI. In elderly patients with mild TBI, the development of a significant post-traumatic dementia with transient psychotic symptoms at night can be seen in association with increased nighttime agitation and disorientation (Goldberg, 2001). This can be viewed as stemming from a disorganized circadian sleep-wake cycle and an exaggeration of the effects of aging on circadian physiology. The use of low doses of risperidone, olanzapine, or quetiapine at bedtime can be helpful in reducing these disturbing symptoms and re-establishing a normal circadian rhythm.

Chronic Complications

Common medical complications after TBI are discussed in sections 3 and 4. Compared to younger individuals with TBI, the elderly have a higher risk for DVT and a higher risk for urinary retention and incontinence. Those who remain sedentary will continue to be at risk for complications of immobility such as decubitus ulcers, DVT, pneumonia, and deconditioning weakness. As previously mentioned, polypharmacy should be avoided, and non-essential medications should be eliminated.

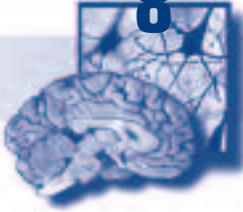
Motor and balance functions tend to recover more slowly in the older adult with TBI because of premorbid limitations in cognition, sensation, strength and balance, along with decreased tolerance for intensive therapy sessions, and increased levels of joint and musculoskeletal pain. Special mobility considerations in the older adult with TBI include alterations in vision, decreased peripheral sensation, imbalance, decreased strength, and limited physical endurance. Many elders have a difficult time comprehending and adjusting to these limitations and therefore pose a significant risk in safety, judgment, and fall propensity. Comprehensive caregiver education and training are paramount. A home evaluation by the rehabilitation team can optimize household level mobility.

TBI/Alzheimer's Disease (AD) Link

Evidence is accumulating that links TBI to the onset and progression of AD (Graham, 1999). Beta-amyloid peptides, which are associated with both AD and Parkinson's Disease, and increased amyloid precursor protein immunoreactivity, which represents plaque-like formation, are demonstrated in post-mortem evaluations after severe TBI and may be an early maker for axonal injury. Among individuals diagnosed with AD, a prior history of TBI is associated with a significantly earlier onset of the dementia. Furthermore, the presence of Apolipoprotein E gene with E 4 allele is associated both with onset of AD and poorer outcome in TBI. It seems that ApoE is critically important to the process of protecting against oxidative insults to the brain. At least in the case of ApoE genotype, the clinical effects of head injury are significantly influenced by the genotype of the affected individual, and there is a close neuropathologic link between the reaction to brain trauma and the process involved in the development of AD (Graham, 1999).

Points to Remember

- Falls are the leading cause of TBI in the elderly
- Gait training, the use of assistive devices, and balance and resistive exercises can decrease the rate of falls and therefore TBIs in the elderly
- For the same type of injury, the elderly are more likely to have intracranial findings (e.g., bleeds) than younger individuals
- In the elderly, a relatively mild TBI can disrupt the normal circadian sleep-wake cycle and result in a reversible post-traumatic dementia accompanied by transient psychotic symptoms at night
- There is an increased risk for Alzheimer's disease in those with a history of prior TBI



Living with TBI: Impact on the Survivor and their Family

Learning Objectives

1. List common questions posed by the family member of someone with a TBI.
2. Describe potential responses to common questions posed by family members.
3. Describe why TBI family members often present to health care professionals in an angry manner.

Introduction

One of the major difficulties confronting the health care professionals assessing and treating TBI patients is that symptoms of TBI are not pathognomonic, and are often confused with psychiatric symptoms. Treating symptoms of TBI individually, without considering the etiology of the complaint, can have several negative effects:

- The patient may be placed on inappropriate medications that do not treat the symptomatology
- The patient can be inappropriately labeled with a psychiatric diagnosis
- Most importantly, the physician will not serve as the patient's and family's primary source of understanding about the nature and course of the cognitive and emotional changes that have occurred

The purpose of this section is to present the explanation and treatment suggestions that the primary care physicians can give to TBI patients and their families for the most common complaints regarding changes in behavior, function, and personality.

Family members often complain of vague "personality" changes in the patient. When questioned specifically, they mention fatigue, anger, emotional outbursts, social insensitivity, lack of organization, memory problems, loss of motivation, and changes in family roles.

Although these have been mentioned in previous sections, they will be addressed here in order to provide the health care professional with educational information to provide to family members as well as the patient. The goals in giving this information are:

- To reduce the family stress level
- To give the family a framework for tolerating TBI symptomatology
- To start the process of treatment of the patient's symptoms by using the family to provide daily structure

The organizing principle is that most of the perceived personality changes are secondary to reduced cognitions from the TBI.

Eight Frequently Asked Questions by Family Members, and How to Answer Them

1. Why is my family member with TBI so tired all the time?

Family members, particularly spouses, often state that the individual with a TBI has no energy, naps at all times of the day, and wakes up tired and unrefreshed. They sleep late or have a chaotic day/night schedule that does not match the rest of the family, nor the normal work-a-day world.

The explanation helpful for family members is two-fold.

Explanation 1. The natural day/night cycle, regulated by the chronobiological clock in the suprachiasmatic nucleus, is permanently disturbed in many TBI patients. Stage three sleep, during which body repairs occur and that is highly correlated with “feeling refreshed”, may be permanently reduced. Patients often wake up many times in the night, rather than going through the normal six 90-minute sleep cycles. This sleep disruption is often mistaken for a vegetative sign of depression.

Additionally, fatigue exacerbates all the symptoms of head injury. When fatigued, patients will be more memory impaired, obtunded, confused, clumsy, and irritable.

Management Strategy Disturbed Sleep/Wake Cycle. In addition to medication, the treatment for this sleep cycle disruption is to artificially impose a rigid bedtime and rise time on the patient. Choose a total sleep time, which is 1 to 1.5 hours longer than the patient's premorbid length of sleep at night. Patients must go to bed at a set bedtime, whether or not they fall asleep. They need to rise at a set rise time and not nap during the next day, even if very tired. It takes about 3 weeks to establish a new artificial day/night cycle. It must be applied 7 days/week. After the three weeks, patients can be allowed afternoon naps, but the schedule must be maintained. If the artificial pattern is broken, it always takes about three weeks to re-establish again.

This is the first intervention that physicians can make as both the family and the patient will come back with statements that the patient has more energy available during the day. It's an easy, highly reinforcing intervention.

Explanation 2. Many of the cognitive functions, which are automatic and reflexive for people without brain injury, require 2-3 times the mental effort to accomplish for people with TBI. This is due to the fact that people with TBI often have to think about, and do with conscious effort, what the rest of the world does automatically, without thinking.

All thinking requires some expenditure of mental energy. Cognitions such as paying attention, switching attentional focus to a new person, keeping up with the topic of conversation, organizing an answer to a question, making a decision, trying to decide what to do next, organizing your day's activities in the morning – all cost mental energy.

If each of us wakes up with 100 units of mental energy to spend each day, patients with TBI spend more of their energy budget on simply paying attention to casual conversation, activities of daily living, and making choices, than does the rest of the world. They “use up” their energy budget by mid afternoon, and have to wait for a good night's sleep to recharge the next day and start over with 100 more mental energy units. This is why they are tired “without having done anything exhausting”. Making choices, having to think spontaneously or flexibly or conditionally (“if A happens, I will do...”, but “if B happens, I will do...”) — all of these are high-energy expenditures.

Treatment recommendations:

- Make important decisions when the patient has the greatest amount of mental energy, usually in the morning.
- Make as many activities as possible into a routine to minimize choice. This saves mental energy.
- Do not fill up the days with scheduled activities, do one important thing/day.
- The use of an organizer, either paper or electronic is essential. Premorbid “blithe spirits” who premorbidly made decisions based on how they felt at the moment, have the greatest difficulty adjusting to life by familiar routine. Patients often need help in how to use these. Often speech and language pathologists can help in the organizer training.

2. Why is my family member angry all the time?

Explanation. Cognitive deficits — slowed rate of information processing, reduced span of attention, loss of the ability to multitask (“Now I'm a one-trick pony.”), memory problems for new information, visuospatial difficulty in perceiving the environment — all serve to make the world seem a more difficult place for the patient to comprehend. The anger expressed by patients is often a symptom of stimulus overload.

“Catastrophic reactions” are emotional responses of neurologically impaired patients when the environment is too complex for them cognitively. There are four variants:

- Laughing hysterically when overwhelmed, the patient appears “silly” to family.
- Bolting, running away when overwhelmed, “I’m out of here!”
- Crying when cognitively overwhelmed. This is often mistaken for depression. The condition does not respond to antidepressants. The treatment is to simplify the situation, the patient immediately stops crying.
- Anger outbursts when overwhelmed. This is the most maladaptive of responses, as people go away, the situation simplifies, then the patient calms down. The method the patient learns is maladaptive as it breaks psychosocial bonds.

Patients can show all four, but usually evidence one consistent response to being overwhelmed, anger and tears are the most common responses. Patients are not aware of the connection between the environment being too complex and their anger or tears. If asked why they are angry, they cannot explain, and simply get angrier.

Treatment recommendations:

- First, the family can point out the irritability, frustration, or anger when it occurs, and suggest to the patient that too much is coming at them too fast.
- Family members can be taught to speak with pauses (Speak as if you threw a handful of commas into your speech.) When you pause in parts of the sentence, the patient can “catch up” in information processing.
- The patient can be asked to talk to people one-on-one rather than in groups (two or more people places a strain on attentional mechanisms).
- Patients can ask for verbal slowing, repetition of information, or writing down steps.
- Patients can ask if they can audiotape important information to review later. All physician sessions should be recorded by patients so they can retain the information for a later review.

3. Why is my family member more emotional?

Explanation. Emotions come from the limbic system in the core of the brain, but whether or not they are behaviorally expressed is controlled by inhibitory neurons in the frontal cortex. These frontal inhibitory neurons are at high risk in TBI. Patients are having the same emotions they experienced pre-morbidly, but their control of emotional expression is lessened. Families experience this as “a personality change”.

Treatment recommendations:

- Explain this phenomenon to patients and their families. A poker analogy may be useful to explain the difference between emotions and emotional control. “If I were playing poker with you right now, and I had a royal flush, I would be experiencing great joy. But I wouldn’t want to show it to you, or I couldn’t sucker you into betting. My emotion is the feeling inside, the joy, but my expression is bland — that’s what’s meant by a poker face, it’s my ability to control what emotions I let out.”
- Intensity of emotion is often expressed as tears — either happiness or sadness, or the feeling of being emotionally connected. This is often misinterpreted as depression, and families try to avoid topics, which brings on tears. A better way to handle patient tears is to:
 - Ask permission to bring up topics which induce tears “to desensitize” patients.
 - During the tears, have the patient breathe deeply to dissipate the energy — they often hold their breath in an attempt to inhibit the tears.

4. Why does my family member seem so insensitive and hurtful since the injury?

Patients with TBI appear to be insensitive to partners, children, and friends due to 4 possible cognitive deficits. The first three deficits are related to frontal lobe injury, the fourth to right parietal lobe damage.

Explanation 1: Difficulty with Inhibitory Control. The primary source of social difficulty is a moving of the line, which distinguishes private thoughts (those which should remain unspoken) from thoughts, which are expressed as public statements. Patients describe this as “things just spill out of my mouth, my stop sign has been moved.” “Gumballing” is a better term — thoughts occur, they drop like a gumball onto the patient’s tongue and just roll right out with no mental processing at all. Families need to know that they are hearing the patient’s thoughts, that the barrier between private and public thoughts has been damaged.

Treatment recommendation. After the physician’s explanation, family members need to tell the patient “That’s an inside thought, not an outside one.” This allows feedback without criticism or humiliation (e.g., “that’s inappropriate”).

Explanation 2: Difficulty with Multitasking. A second source of social difficulty is the inability to do two things at once, the loss of the ability to multitask, the reduction of thinking to one channel at a time. In everyday life, most of us are doing at least two things simultaneously when we function socially. We respond, and, at the same time, we monitor ourselves, we watch ourselves responding in

order to self-correct any errors or problems. Some patients with TBI can only do one thing at a time. This means they can either respond, or they can self-monitor, but they can't do both. Adding additional people in the social situation makes this worse. Channel capacity cannot be modified.

Explanation 3: Brain-damage Related Egocentricity. The third source of social difficulty is the inability of the patient with TBI to step outside his or her own view and see someone else's point of view. Spouses in particular find this frustrating.

Treatment recommendation. Patients can be taught a basic form of negotiation with their partners. Asking the spouse to a.) State their point simply, b.) Have the patient state the spouse's point, c.) Have the patient state their own point, and d.) Have the patient state a position that includes half their own and half their spouses position.

Explanation 4: Difficulty Reading Facial Expressions. The fourth source of social difficulty is difficulty reading facial expressions. All human adults are able to distinguish the following emotions on other people's faces, regardless of culture: happy, sad, fearful, anger, surprise, disgust, and contempt. Of the emotions listed, most are discernible by a single part of the face — except for anger. Anger requires accurately reading three parts of the face, with two possible variations in each part. This makes anger a harder task visuospatially. Patients with visuospatial difficulty due to parietal lobe injury may interpret anger as surprise or confusion, and miss significant facial feedback from their spouse.

Treatment recommendation. Having spouses verbalize their feeling (“that makes me angry”) often helps.

5. My family member used to be a real go-getter, the source of energy and ideas. Why is he/she so passive now, just sitting around unless I tell him/her what to do?

Explanation. We exhibit two types of behavior: spontaneously emitted behavior or behavior elicited in response to a certain stimulus. For patients with TBI, spontaneously emitted behavior can be reduced. This problem with behavioral completion fall into two categories:

1. Difficulty with inertia, with starting up a behavior. Often this is the result of both frontal dysfunction and low mental energy. In addition, when presented with too many choices, individuals with TBI have difficulty deciding what to do and may make no choice.
2. Interruption of an ongoing behavior before completion. This is a loss of internal focus, resulting in tangentiality in speech, or unfinished tasks at home. An activity is normally defined by a beginning, middle, and an end of response. Patients with TBI often drop off the end of the response and start another action. This results in tasks that are incomplete, or just sitting in distracted confusion.

Treatment recommendation. Initiation difficulties can be partially remediated with a daily routine; temporal conditioning replaces spontaneous choice and reduces the dilemma of decision-making. If the problem is too much initiation (i.e., impulsivity), slowing them down by requiring mental counting before acting is recommended. If they are easily distractible, this gives family members an opportunity to distract them from an unwise course of action. Verbal distractibility (tangentiality) is best treated by asking the patient to announce when he/she is changing topic. This may appear conversationally awkward, but will focus the survivor's attention on the problem.

6. What can I do, I feel as if I no longer have a spouse, but another child in the family?

Explanation. The partner of the patient with TBI often feels as if their role in the marriage and family has changed. This is a real perception; their roles and responsibilities have changed, they have grown enormously. It is best to give spouses several premises about brain injury, so they can perform their increased role without anger.

Treatment recommendation.

- Think of the patient's brain as Swiss cheese. There is plenty of good cheese — intact circuits that were unaffected by the injury. But now there are a few "holes" where cells were injured or destroyed. Whatever these injured circuits did before, will now be a problem for the patient. It is important that spouses loan the patient **only those abilities that the patient has holes in — only those areas where the patient needs help.** It is important that patients with TBI be permitted to practice behaviors that are deficits, which they are poor at — so they can improve with practice.

This accomplishes three things:

1. Family members (and patients) stop thinking globally, that all abilities are lost.
 2. Family members will stop doing everything for the patient in the name of "efficiency" and can tolerate slow, inaccurate, messy performance, if it results in "brain rewiring."
 3. Patients need to be given family tasks, which are within the "good cheese" portion of their abilities — practical tasks that are their contribution. Again, structure to the day and reducing choices allow maximum function.
- The following is an example of saving energy with routine that can reduce a spouse's sense of caretaking load with a neurologically injured partner who has slowed speed of dressing or getting started in the morning. The patient is to have clothes for the next day arranged in complete outfits, including underwear, socks, shoes, etc. The pile of clothes can be arranged

in the order of dressing the body. Routines save time and help the patient feel more independent.

- Return to compensated work is often impossible for patients with TBI. Cognitive functions necessary for most jobs are speed and memory — these effect production rate and independence of action, necessary from an employer’s point of view. The two areas most affected in TBI are slowed information processing and memory difficulties. Often an individual with TBI is on social security disability, medical pension or some compensation for injury. This can be viewed as “income” brought into the family — the individual may not be able to work at a salaried job, but they provide means for families to economically survive.
- TBI can be thought of as “premature aging” of the brain. What were the couples’ intentions as they aged, and grew towards retirement? Unfortunately, many Americans have a poor model of aging — physical decline, leading to social irrelevance, followed by death. Other cultures recognize that there is a developmental task for each age. A potential developmental task for those who are older is to become a sage or a mentor, rather than compete in the work-a-day-world. The patient with TBI now has unique experiences — he/she knows what it is like to deal with adversity, to have a chronic condition, to fight their way through each day spending more energy than most to function. They understand handicap and disability. It is strongly recommended that husband and wife attend a TBI survivor/caretaker group as a couple. These groups often break into two meetings, TBI survivors, and spouses/family members. Knowing that others have coped and having a place where both get their needs met is crucial.
- Caretaking spouses need to have some respite/alone time. This is not meant to be time to do errands, but to replenish their energy, doing something fun and joyful. Spouses often feel guilty about taking time off. Remind them that the same logic of putting on the adult oxygen mask first before putting on a child’s in an airplane emergency holds true. If the spouse breaks down, both the patient and the spouse will falter.
- Caretaking spouses often feel unappreciated for their extraordinary efforts. Writing a couple of phrases of appreciation “You are such a wonderful partner, I’m so lucky to have you”. Or “Have I told you I love you?” in the patient’s memory book or electronic organizer every few days, (which the patient reviews after each meal) can make a spouse feel that their efforts are noticed.

7. There seem to be so many problems, new ones keep cropping up. Will our lives ever get back to normal?

Explanation. In TBI, all impairments are present at the moment of damage — at the initial impact. All difficulties may not show up at

once, but they are all present at the moment of injury. It is only an illusion that new problems are emerging over time. The expression of some impairment is initially masked by larger functional problems, but the masked impairments have been present all along. For example, if the TBI survivor cannot scan visually in an effective way, if their gaze is random rather than orderly, he/she cannot read. Training to scan from left to right, top to bottom, can give survivors back their ability to see the printed words in sentences in an orderly sequential manner. Now that the survivor can see the words, it may be discovered that his/her reading comprehension is only 60%; that is, they don't comprehend 40% of what they are reading. To the family, this appears to be a discovery of another problem, a new one, but the reading comprehension difficulty was present all along. It could only be exposed if the patient can see words on an orderly manner.

Treatment recommendation. It is helpful to explain the emergence of masked cognitive problems (or finer and finer difficulties) as peeling an onion. Fixing problems at one level allows the survivor to emit more complex behavior, which permits subtle impairments, which have been present the whole time, to emerge.

8. How long is the psychological recovery going to take?

Explanation. The standard answer to “How long does it take for the brain to heal?” is usually 18-24 months (the period of spontaneous healing, based on age and metabolism, with younger patients requiring 18 months, older ones requiring 24 months). The answer to the question, “How long before he/she is as good as they're going to get?” is harder to answer. There is evidence that language and intellectual recovery are still occurring at 5 years post-injury, albeit at a slowed rate. It is best to think of the TBI survivor's psychological/cognitive problems in the following manner:

- **Some problems are primary:** They are due to alterations in the nervous system as a result of physical damage. Examples are memory problems, spatial difficulties, emotional lability, and attentional difficulty. These primary lesion effects are best handled by environmental modification, psychoeducation, (explanations) and cognitive retraining.
- **Some problems are secondary:** They are emotional reactions due to changes in functional ability, role, and status. These emotional reactions to existential changes are best dealt with by short-term psychotherapy.
- **Some problems are tertiary:** That is, they are long-standing in nature, either developmental and/or characterological. They are determined either by the personality defensive structure of the individual, or, their developmental stage in life. Here's an example of the effect of developmental stage on the TBI survivor's concerns and willingness to work on issues. Two

identical cases of TBI, both with left upper extremity hemiplegia, a tendency to drool (secondary to reduced swallowing and pooling of saliva), and moderate memory problems. One is 18, the other 43 years of age. The 18 year old will be most concerned about his left arm hemiplegia and drooling, and will consider the memory difficulty as the least important to him. This is because his developmental task at this stage in life is to find a partner. Being considered “dating material” will be his primary concern, drooling and physical impairment will be highest on his list of what needs remediation, memory less so. For the 43 year old who is concerned with earning a living for himself and his family and working up the ladder of success (his developmental task), the drooling and hemiplegia are of concern, but not nearly as much as the memory problem, which interferes with his ability to be economically successful. Paying attention to the age of your patient and the developmental life task they are most concerned with will tell you what your patient will be most concerned about.

Treatment of characterological tertiary problems is long-term psychotherapy; treatment of developmental stage problems is short-term psychotherapy.

Points to Remember

- **Listening to family members and providing them with education about TBI is a powerful intervention supporting the family system**
- **Helping family members understand the cause for various problems and ways of handling them gives them a sense of control over a very difficult situation**

Other Important Family Issues Facing the Health Care Provider

1. Human Sexuality: Ask patients about changes in their sex life after TBI.

Changes in sexual functioning often occur in a couple after one has had TBI. Physicians should ask both the husband and wife (separately) about their sexual functioning. Here is a suggested way to bring the issue up. First tell the patient that you are going to ask a personal question which he/she may choose not to answer.

“I always ask whether patients are having any sexual problems. Your sexual health is one important part of your life. Sometimes an illness, accident, or a medication can affect sexuality too. How has your sexual relationship been going lately?”

Other problem areas and potential solutions:

- For patients who are now disinterested in sexual activity — a laboratory test for gonadal hormones, particularly testosterone, may be in order.

- Difficulties with achieving or maintaining an erection may be resolved with medication (Viagra) or mechanical devices (Erec-Aid, a plexiglas tube into which the penis is placed, which creates a vacuum, the penis is engorged and a penile ring at the base maintains an erection for up to 30 minutes). Referral to a urologist or human sexual dysfunction clinic (within the VA, the andrology clinic) can result in more treatment suggestions.
- Reduced vaginal lubrication — replacement with gels.
- Altered sensation after neurological injury — numbness, hypersensitivity, tingling, painful. A good neurological examination can alert the partner to these changes, especially areas to avoid. Other recommendations, approach from the “good side” of sensory intactness, emphasize intact senses, more talking, more touching, use of perfume (although patients with frontal injury are frequently anosmic).
- Awareness of position of affected body parts, for example, a numb arm. Suggested treatment is a change in intercourse position with the “good” side up.
- If spasticity or hypertonicity is a problem, a warm bath before intercourse can relax the tone.
- Fatigue effects sexual functioning. Naps before sex or switching sex to the morning when the patient is more awake, can reduce brain injury effects.
- Other neurological symptoms of altered communication (incorporate more touching, physically guiding the hand of the person affected), difficulty reading facial expression (use more verbal descriptions of how you feel), keep dialogue simple, use a familiar routine.
- If bowel or bladder incontinence is a problem, reduce the chance of accidents during sex by recommending:
 - Routine bowel program — use of suppository at specific, convenient time of day.
 - Routine bladder program (timed voids every two hours).
 - Limit fluid intake prior to sex.
 - Empty bladder just before sex.
 - Have a urinal, soap, washcloth, water, and towel nearby for urgency problems.
- Inappropriate sexual comments or touching to individuals not their partner. It helps to understand why brain injured patients may make unwanted sexual comments. A change in physical functioning often brings fear of loss of sexual/physical competence. It’s on their minds. Cortical control of emotion is often lessened, lability and impulsivity result. Patients with TBI often misperceive or misinterpret the social situation. They may

have difficulty understanding consequences. They often want nurturance, support, affection, friendship, or company, but sexualize it instead.

- Recommendation. Direct confrontation to the patient. Ascribe positive motivation to the patient's action, but redirect with what's appropriate. Reflecting back reality is the physician's role; there are social consequences to sexual inappropriateness.
- Example of a possible response: "I know that you are grateful for the care and understanding you get from me (ascribing positive motivation to the patient's action, even if not apparent), we are not in a social situation, but in a professional one. You need me to work with you, and I can work with you only if you speak to me with courtesy and respect. We are not in a social situation."

2. Why are the family members, particularly spouses of patients, so complaining and angry?

Family members project negative emotions onto physicians for many different reasons:

- Family members feel they need energy to cope with a crisis, and anger activates people.
- The family member is in danger of collapsing in despair, so they get angry to activate themselves.
- Anger is often delayed from the acute setting, when coping with disaster, emotional suppression is paramount. These suppressed feelings come out in outpatient settings because "now it's safe to express how overwhelmed we feel".
- TBI often challenges families assumptions about how the world works. We all hold some false beliefs about the world, such as:
 - Life's fair. This is untrue. In dealing with unfairness, it helps to change the frame of reference of the family member. For example: Everyone who is alive today has beaten the odds. The odds are 100,000,000 to 1 that a particular sperm would fertilize the egg, which resulted in a particular individual. Those are the odds we all win at conception. After we are born, everything else is gratis, icing on the cake. This is offered as an alternative viewpoint to family members who feel cheated that the patient didn't get his/her fair share of good health and long life with any untoward events.
 - Spiritual beliefs that bad things that happen to us are punishment for sin. Families will state, "But he's good, why did this happen?" It is helpful to point out that suffering is not a punishment, surely innocent infant/children suffer and are not to blame.

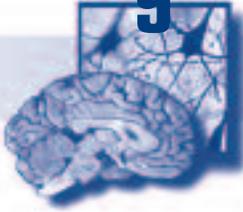
- Displaced anger at the patient for bringing on the TBI, “I told him not to drink and drive”. Often this anger cannot be expressed at the patient who is too vulnerable, so it is often displaced onto the nearest authority figure, the physician.
- The belief that causal events are simple. If I can control A, then B will surely happen. If I can make you give him the right drug, his memory will return. This is a belief in simple, one factor causality.

How to respond to angry family members:

- Never respond in anger, not even in clipped tones.
- Give lots of information to the family member about TBI, it binds anxiety. Have them record the information, to review at a later time.
- Determine what is fair for the patient — and stick to it. Being a broken record is often helpful, this means repeating your position, not in anger.
- Playing dumb can be a useful stance, “ I’m confused, at first I understood you to say that he’s impulsive and can’t be controlled, then you said he’s unable to self-start, can you give me some examples of what you mean?”
- Always ask for specific examples. Family members don’t speak in medical terminology and they may mean something quite different than you do when they say, “he’s depressed”.
- If a family member is constantly attacking you verbally, demanding predictions and prognoses to questions that cannot be answered, recognize that they have difficulty with ambiguity. Acknowledge that not having clear answers is hard, but you have given them all that you can. If they continue to question you, answer questions with a question. This stops the placing of blame on the physician, and places the tolerance of ambiguity where it belongs, back with the family.
- When pressured by a family’s anger, delay by taking time to answer. ”I can understand why you might think that. I’ll have to think about that, I’ll get back with you later.” This allows you not to answer in anger.
- Consider suggesting that the spouse seek counseling. Acknowledging that care giving is an incredible load for anyone to bear, that anyone would need some stress reduction and support. Have the names of some professionals that are familiar with head injury. The BIA maintains such a list, as does the National Caregivers Alliance. Definitely have literature available on local support groups for family members.

Point to Remember

- **Dealing with angry and frustrated family members is stressful; listen and provide educational information in a direct, matter-of-fact, consistent manner**



Driving Issues after TBI

Learning Objectives

1. Describe the effects of physical, cognitive, and neurobehavioral TBI sequelae on driving abilities.
2. State how medications can affect driving abilities.
3. Describe the components of a pre-driving assessment.

Introduction

Driving is an essential activity of daily living for many individuals and is one determinant of level of independence. Individuals lacking this mobility are deprived of meaningful educational and vocational opportunities and reduced ability to participate in recreational, social, cultural, and community events. They are also deprived of full and independent completion of basic survival skills and self-sufficiency.

Driving requires a complex set of skills and abilities, including mobility, vision, and visual-motor coordination, as well as multiple cognitive and neurobehavioral abilities and functions. It is well known that individuals with TBI have varying degrees of physical impairments, cognitive dysfunctions, and neurobehavioral problems that can impede the integrated operation of neuropsychological and physical capacities for driving. However, disabled veterans, including those undergoing rehabilitation, are entitled by Public Law 93-538 to have the opportunity to pursue a return to driving.

It has been estimated that 38% to 78% of individuals with traumatic brain injury seek and obtain driving privileges after their injury (Katz et al., 1990; Oliver et al., 1996; Priddy et al., 1990). A more recent study suggests that individuals with TBI who successfully pass a driving evaluation and complete a comprehensive driving program are able to reintegrate into the driving community with minimal difficulty (Schultheis et al., 2002). Thus, it is the physician's responsibility to determine when and how to return the individual to driving. Only after an understanding of the impact of the individual's physical, cognitive, and neurobehavioral factors on driving has been attained can a plan for training and treatment be developed that will result in safe driving.

Effects of Physical Impairments

Occulomotor dysfunction including diplopia, hemianopsia, and reduced vision can impact driving capabilities following TBI. Therefore, it is essential to perform a visual acuity test and visual field evaluation periodically during the recovery stage. Some individuals are also known to have auditory or vestibular dysfunction, causing hearing impairment and vertigo/dizziness, which might also interfere with driving performance. Refer to Section 4 for details about visual and vestibular dysfunction.

Motor disturbances, such as paresis of the limbs, spasticity, and ataxia can obviously interfere with the visual-motor coordination and motor skills requisite for driving. It is thus essential to evaluate the person's motor performance and range of motion prior to a driving evaluation. Upper extremity steering strength and range of motion are frequently included in pre-driving evaluations.

Primary care physicians may wish to consult ophthalmology or physical medicine and rehabilitation to assist with evaluation of these dysfunctions.

Effects of Cognitive Impairments

Because driving places high demands on the brain's ability to integrate a number of cognitive functions, possession of driving knowledge and driving skills are insufficient to guarantee safe driving performance. Cognitive impairments commonly seen after TBI can influence driving performance in several ways.

Arousal and Attention

Following TBI, individuals often suffer from inattention. Inattention includes impaired sustained attention and divided attention, hemispatial neglect, and slowed information processing speed — all of which can compromise driving capabilities.

Executive Abilities

Changes in executive abilities as a result of TBI were presented in Section 5. Dysfunction in mental flexibility, sequencing ability, monitoring, and allocation of cognitive resources, and problem solving can impede the individual's driving performance. These dysfunctions may be expressed as difficulties in navigation planning, the ability to handle unexpected road conditions, the ability to consider multiple decision alternatives and their consequences, and the ability to perform multiple simultaneous tasks.

Visuo-spatial Perception and Skilled Motor Abilities

The ability to see accurately, to judge depths and speed, and to perceive changes in the environment are important for safe driving. Impairments in

visuo-spatial perception, visual scanning ability, perception of focus, spatial relations, color perception, and perceiving figure-ground relationships can obviously interfere with driving performance and safety.

Effects of Neurobehavioral Dysfunction

Neurobehavioral changes frequently include impulsivity, aggression, lack of insight, and substance abuse. These behaviors can lead to risky driving behaviors, which may endanger the safety of the driver, passengers, pedestrians, and other vehicles and property.

Other Factors Affecting Driving Capabilities

Seizures

The overall incidence of epilepsy among individuals with severe TBI is estimated to be 17.2% (Annegers & Cohen, 2000). A seizure during driving can impose a great threat to public safety. Some investigators have shown a 40% increased risk in serious injuries due to automobile accidents in this population (Taylor et al., 1996). However, there is no common nation-wide law that restricts driving for patients with epilepsy. Most states require people with epilepsy to be seizure-free for a certain period, ranging from 3 to 12 months. Other states have more flexible restrictions, basing the required seizure-free period on clinical factors (Krauss et al., 2001). Thus, it is the responsibility of the physician to help determine when their patients may drive, with the intent being to protect the public and patient safety optimally while permitting patients with controlled seizures to drive.

Medications

Psychotropic medications, commonly prescribed for individuals who have sustained a TBI, include benzodiazepine, neuroleptics, narcotics, hypnotic sedatives and barbiturates, and antidepressant medications such as SSRIs and tricyclics (see Section 6 for more details on pharmacological treatments). The medications can influence driving capabilities by: affecting visual abilities; causing fatigue, drowsiness, and slowed reaction time; and decreasing attention and multitasking abilities (Hopewell, 2002). Other agents, frequently prescribed in the TBI population to treat general medical problems, may also affect the above driving abilities (e.g., b-blockers, anti-convulsants, sedating antihistamines, etc.) or interact with the primarily “psychotropic” medications. As always, physicians should be aware of potential interactions and the effects of multiple prescribed medications and monitor or assess side effects.

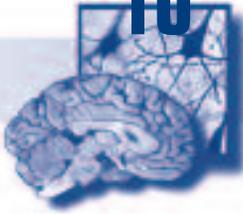
Pre-driving assessment

There is no universally accepted standardized driving evaluation for individuals who have sustained a TBI. Current pre-driving assessment and training methods typically include neuropsychological evaluation, behind-the-wheel evaluations, reaction time measures, and assessment of visual abilities including acuity, night vision, and depth perception (Schultheis, 2000). However, neuropsychological test performance has been found to have highly variable predictive power 0.20 to 0.95 (Schultheis, 2000). Computer-based driving simulators create a safe means of replicating actual driving conditions including hazardous situations (emergency vehicles, surprise pedestrians), and poor weather and lighting conditions (rain, glare) that would be difficult to achieve otherwise. But further research is needed to verify a driving simulator's reliability and validity in predicting driving capabilities.

Driver Rehabilitation Programs

A driver rehabilitation program for individuals who have cognitive and/or physical deficits secondary to brain injury should include a pre-driving assessment, a behind-the-wheel assessment, education, and training. These components are necessary to ensure that individuals are safe to operate a motor vehicle on public roads. This multi-faceted rehabilitation approach is initiated by referral from a physician, who has determined that the patient is medically stable and able to receive this comprehensive evaluation.

VA has 39 Driver Rehabilitation Centers nationwide. Information on the program and location of centers is available at <http://www1.va.gov/health/rehab/dvrcnters.html>.



VA TBI System of Care

Learning Objectives

1. Describe the TBI continuum of care within the VA.
2. Identify the VA Brain Injury Network of Care and its components.
3. Describe the unique benefits available to veterans within the VA as well as within other federal, state, and community agencies.
4. Identify the exceptional, life long issues that impact the brain injured veteran and caregiver.

Introduction

The Department of Veterans Affairs (VA) recognizes traumatic brain injury as a priority condition for health care services. It is one of the Special Disability Populations subject to VA's congressionally mandated Capacity Report. The VA is required to maintain its capacity to provide specialized care for veterans sustaining a brain injury. TBI is also designated as one of its' Special Emphasis Programs for which national performance measures are in place. A long-standing national agreement between the Department of Defense (DoD) and VA provides for transfer of active duty personnel who have incurred a brain injury to VA medical centers (VAMCs) for care.

The VA established a system of care with the goal of assisting active duty military and veterans sustaining a brain injury in progressing along the continuum of care (**Table 1**, next page) toward achieving optimal clinical outcomes. This section provides an overview of the different levels of care that may be needed when providing for individuals with a TBI. It includes a brief history of the development of the TBI Network of Care, the structure, management of referrals, and the roles and functions of the designated sites. Due to the catastrophic nature of brain injury, maximizing available resources can be crucial. Sources of assistance within VA as well as state and local entities are provided to assist health care providers, survivors, and families.

Providing a Continuum of Care

Persons having sustained a traumatic brain injury may require different levels of TBI-specific and other supportive care throughout their lives. Ideally, patients follow a typical treatment course in which they initially receive acute medical, proceed to the acute level of rehabilitation, and progress to more independent settings such as home with an outpatient program. However, some patients due to the severity of their brain injury and/or other medical issues would not benefit from acute rehabilitation or cannot tolerate such an intensive approach. Unfortunately, some patients do not progress and will not benefit from intensive rehabilitation. These patients will likely require long-term care (see Appendix A) that is offered in the least restrictive setting possible.

Ongoing follow-up assessment of the TBI survivor is key to managing sequelae and preventing development of secondary conditions. A TBI survivor is vulnerable to life events that typically occur and effect general health status. Something that may seem relatively insignificant can potentially result in a dramatic change in the ability to care for oneself. Quite likely, it is the primary care professionals that will be following the TBI survivor for the long-term. Consulting with TBI specialists will assist in providing appropriate ongoing care.

Components of a Continuum of Care for TBI	
Trauma Care	<ul style="list-style-type: none"> • Emergency Room/Trauma Center • ICU
Specialized Acute Inpatient Rehabilitation	<ul style="list-style-type: none"> • High Intensity Rehabilitation (3-5 hours per day) • Patient actively participates
Sub-Acute Rehabilitation	<ul style="list-style-type: none"> • Lower Intensity Rehabilitation (< 3 hours per day) • Ventilator Care • Coma Care
Post-Acute Rehabilitation	<ul style="list-style-type: none"> • Outpatient • Day Treatment • Home Care
Community Re-entry	<ul style="list-style-type: none"> • Transitional Living • Independent Living • Vocational Rehabilitation • Supportive Employment
Extended Care	<ul style="list-style-type: none"> • Skilled Nursing Facility • Neurobehavioral Management • Assisted Living • Adult Day Care • Respite Care

Table 1

Point to remember:

- **The needs of survivors are unique and may require intensive long-term management and support beyond acute rehabilitation**

Development of a VA/DoD TBI Network of Care

Prior to 1992, the VA used multiple approaches to provide specialty care across the continuum for veterans and active duty members having sustained a traumatic brain injury. Physical Medicine and Rehabilitation Services with a special interest in this condition acquired additional knowledge and skill to enhance their capability of providing TBI-specific medical care and rehabilitation. However, this specialized care was not integrated throughout the VA's health care system.

In February 1992 the Defense and Veterans Head Injury Program (DVHIP) was established and funded by Congressional appropriation. The mission charge to the program was:

“The purpose of this project is to seek to define optimal care and rehabilitation utilization for patients with TBI...It is intended that this effort facilitate the referral of TBI patients among DoD and DVA facilities.” (E. Mendez, ASD, DoD Health Affairs, Dec. 1991)

In 2002, DVHIP was restructured as The Defense and Veterans Brain Injury Center (DVBIC). DVBIC has become proactive since its inception, developing multiple new programs that will enable patients evaluated in the DVBIC to have multiple treatment options at each site. The program helps to ensure that all military and veterans personnel with traumatic brain injury receive TBI-specific evaluation, treatment, and follow-up. Clinical care and research is conducted at both military and VA sites that allow DVBIC to complete innovative single and multi-center trials to inform future clinical care and treatment strategies. Research conducted by the DVBIC has helped define optimal care for survivors of TBI.

The DVBIC is currently comprised of DoD, VA, and civilian specialized brain injury sites:

- Walter Reed Army Medical Center - Washington, DC Center Headquarters
- Naval Medical Center San Diego - San Diego, CA
- Wilford Hall Medical Center - Lackland Air Force Base, TX
- James A. Haley Veterans Hospital - Tampa, FL
- Minneapolis Veterans Affairs Medical Center - Minneapolis, MN
- Veterans Affairs Palo Alto Health Care System - Palo Alto, CA
- Hunter McGuire Veterans Affairs Medical Center - Richmond, VA
- Virginia Neurocare, Charlottesville, VA

For additional information contact the DVBIC Headquarters 1-800-870-9244 or visit their Web site at <http://www.dvbic.org>

The DVHIP formed the nidus for further development of TBI services within the VA. Additional VAMCs were identified who were willing to support and provide components of the TBI continuum of care. They became known as Network TBI Centers. The original four VA DVHIP sites became known as Lead Centers because they provide the full spectrum of TBI care. VA Under Secretary for Health officially recognized this system of care in an Information Letter issued in February 1997.

Each of the participating medical centers has a designated TBI coordinator who facilitates patient care, expedites facility transfers, and community placement. A National TBI Coordinator position was established within the Rehabilitation Strategic Healthcare Group to monitor and maintain this system of care.

Point to remember:

- DVHIP (now DVVIC) established the original structure for a coordinated system of Brain Injury Care for the VA and DoD.

VA TBI Network of Care: Major Roles and Functions

VHA Brain Injury Rehabilitation Sites

TBI Lead Centers (4)

- Located in tertiary care medical centers
- Accredited in Brain Injury Medical Rehabilitation by the Commission on Accreditation of Rehabilitation Facilities (CARF)
- Dedicated TBI Interdisciplinary Treatment Teams
- Provide the full spectrum of rehabilitation services
- Accept admissions nationwide, including active duty military
- Focus is on rehabilitation in the acute and early post-acute phase
- Assist other health care providers with assessment and treatment planning
- Identify federal, state, and community resources

TBI Network Centers (18)

- CARF accredited in Comprehensive Inpatient Medical Rehabilitation
- Provide components of specialized care
- Staff have an interest in TBI rehabilitation
- Usually located closer to the patient's home
- Coordinate continued rehabilitation after discharge
- Follow the patient in their catchment area for the long-term duration of service needs
- Provide care coordination
- Identify federal, state, and community resources

TBI Associate Network Centers (5)

- Provide TBI-specific case management
- Assist with coordination of care
- Identify federal, state, and community resources
- Provide education to survivors and families
- Outsource services as needed
- Follow the patient in their catchment area for the long-term duration of service needs

Point to remember:

- **All VA TBI Lead centers offer specific comprehensive acute Brain Injury Rehabilitation. The scope of care at the Network and Associate Network Centers varies from site to site.**

Consultation and Referrals

To facilitate management of referrals and coordination of care across the system, the TBI Network of Care is organized regionally (**Table 2**). The TBI Lead Centers, having the most expertise and experience, consult and guide other programs in their region to best meet the needs of the survivor and family:

For VISN's	TBI Lead Center	Phone Contact
1, 2, 3, 4, 5, and 6	Richmond VAMC	(804) 675-5332
7, 8, 9, 16, and 17	Tampa VAMC	(813) 972-2000, ext. 6185
10, 11, 12, 15, and 23	Minneapolis VAMC	(612) 725-2000, ext. 3562
18, 19, 20, 21, and 22	Palo Alto HCS	(650) 493-5000, ext. 66150

Table 2

VHA TBI Network

Lead Centers

James A. Haley Veterans Hospital – Tampa, FL
Minneapolis VAMC – Minneapolis, MN
Veterans Affairs Palo Alto Health Care System – Palo Alto, CA
Hunter Holmes McGuire VAMC – Richmond, VA

Network Centers

VAMC Albuquerque, NM	VAMC Augusta, GA	VAMC Bay Pines, FL
VAMC Boston, MA	VAMC Denver, CO	VAMC Hines, IL
VAMC Houston, TX	VAMC Indianapolis, IN	VAMC Knoxville, IA
VAMC Little Rock, AR	VAMC Long Beach, CA	VAMC Martinsburg, WV
VAMC Northport, NY	VAMC San Juan, CA	VAMC Seattle, WA
VAMC Tuscaloosa, AL	VAMC Tuskegee, AL	VAMC West Los Angeles, CA

Associate Centers

VAMC Albany, NY	VAMC Cincinnati, OH	VAMC Pittsburgh, PA
VAMC Temple, TX	VAMC Wichita, KS	

For additional assistance, contact **Gretchen C. Stephens**, VA National Traumatic Brain Injury Coordinator at (804) 675-5597 or e-mail at gretchen.stephens@med.va.gov. National program information and points of contact are updated and available at <http://www1.va.gov/health/rehab/index.htm>.

To facilitate decision making for referrals and coordination of care across the system, a [TBI Referral Algorithm \(Appendix B\)](#) was developed and implemented by the TBI Network case managers and coordinators.

Point to remember:

- **Contact a TBI Lead Center for assistance with TBI care planning. TBI Case Management helps ensure continuity of care**

Resources and Outreach

It is important to be aware of all resources that may be available to veterans and their families dealing with the impact of traumatic brain injury. These programs and services may be a valuable source of assistance.

Health Care Benefits: Maximizing Resources

Upon enrollment, veterans are placed in one of VA's Enrollment Priority Groups (<http://www.va.gov/elig/Priority%20Groups.htm>) 1 through 8, with 1 being the highest priority group. Veterans in enrollment group 5 or higher, having sustained a brain injury may qualify for the Priority 4 group through a formal Catastrophic Disability Evaluation. The provider, the veteran, or his/her representative may request the evaluation. Contact the Enrollment Office at a local VA health care facility for information on how to request an evaluation. Moving to Priority 4 enrollment group does not eliminate responsibility for co-payments as a result of means testing.

Active duty, retired service members, and retired reservists may be eligible for health care under the military health system. Information regarding eligibility and benefits under TRICARE can be found at:

<http://www.tricare.osd.mil/>.

Veterans covered under other insurance providers may be able to obtain TBI-specific services closer to their home.

Point to remember:

- Identifying a veteran as catastrophically disabled may help to maximize VA health care benefits.

Veterans Benefits Administration (VBA)

Vocational Rehabilitation and Employment (VR&E) Program

Veterans who have a **service-connected disability** that was incurred or aggravated in service are potentially eligible for vocational rehabilitation services. VR&E Vocational Rehabilitation Counselors in the VBA Regional Offices will determine if a veteran is entitled and demonstrates a need for vocational rehabilitation services in order to prepare for employment. (Appendix C). Information on location of VBA Regional offices is available at: http://www.vba.va.gov/bln/vre/regional_offices.htm. (The printable Vocational Rehabilitation application form (VA-1900) is located at http://www.vba.va.gov/bln/vre/self_serv.htm).

Independent Living Program

Independent living generally refers to the empowering of individuals to take control of their own lives, live autonomously, and participate fully in society. Independent living services (Appendix D) are provided through the Department of Veterans Affairs Vocational Rehabilitation and Employment Program. These services are intended to improve the ability of a veteran to function more independently within his/her family and/or community. Independent living services do not require a program goal of return to work. The printable application form (VA-1900) is located at http://www.vba.va.gov/bln/vre/self_serv.htm. Additional information is available at <http://www.vba.va.gov/bln/vre/ilp.htm>.

Point to remember:

- **Service-connected veterans with TBI should apply for Chapter 31 benefits through VR&E for Independent Living Services**

Other VA Programs

VA has a number of special needs programs that in some instances may be appropriate for persons with brain injuries. These programs are not geared specifically for the individual who sustained a TBI; therefore, the individual may need to be functioning at a fairly high level, e.g., independent in activities of daily living and does not require close supervision.

Homeless Veterans Program

The VA's specialized homeless veterans treatment programs offer a variety of programs and services such as:

- Domiciliary Care for Homeless Veterans
- Supported Housing Program
- Comprehensive Homeless Centers

Additional information about these programs and others is available at <http://www.va.gov/homeless>.

Veterans Industry

Veterans Industries is a vocational rehabilitation program of the Department of Veteran Affairs that subcontracts with many and diverse industries. They provide temporary and permanent staffing for information technology, manufacturing, warehousing, construction, office support, retail, and the services delivery industry. They also provide outsource support in assembly, packaging, sorting, grading, reclaiming, and recycling.

Veterans Industries programs develop an individual rehabilitation plan for each veteran. And, depending on the specific program location, Veterans Industries provides a wide range of support services to the veteran. These include:

1. Transitional work experience
2. Sheltered work therapy
3. Career development/job placement and follow-along services
4. Employment education and job readiness training
5. Vocational assessment (situational)
6. Transitional residence (residential rehabilitation)
7. Vocational counseling and case management

Additional information is available at <http://www.va.gov/vetind>.

Point to remember:

- **There are other programs within VA that are not specifically geared to persons with Brain Injury, but may be a viable resource depending on the needs and abilities of the veteran**

Other non-VA Resources and Programs

The Brain Injury Association (BIA)

The BIA partners with many federal, state, and other agencies to promote awareness, education, prevention, public policy, and research related to brain injury. BIA is an excellent resource for health care providers, families, and survivors. Their Web site (<http://www.biausa.org>) contains educational materials, links to state chapters, support groups, and many other resources. They also maintain a toll-free Family Help line 1-800-444-6443.

TBI State Grant Programs

A number of states across the country have received federal grants from the Health Resources and Services Administration (HRSA) in conjunction with the 1996 Traumatic Brain Injury Act. These grants are specifically for providing access and development of programs and services for survivors and families. The Web site contains information about these funded programs: <http://www.tbitac.org>.

State Vocational Rehabilitation Departments

Every state offers vocational rehabilitation services for individuals with disabilities. Vocational Rehabilitation is a joint federal-state program that is administered by each state, so the available services vary from state to state. Services may include assessment/evaluation, job training, and assistance with job placement, supported employment services, and independent living services. In some cases, Vocational Rehabilitation agencies also may

pay for rehabilitative services at private programs, technical/vocational training (at technical schools), or post-secondary education.

Point to remember:

- **There are Federal, State, and local organizations outside of VA that can provide a variety of resources to persons with brain injury**

Life Care Management Considerations

In addition to medical care for their brain injury, veterans and their caregivers may need assistance with a variety of non-medical issues that can significantly impact their functioning and managing in the community setting. These may include but are not limited to:

Guardianship

When a patient is felt to lack the capacity and or judgment to make decisions regarding themselves or personal affairs the state may assign a guardian. The guardian can be a family member, other interested party or professional guardian chosen by the family or state. States differ on mechanisms of determination and assignment. Court fees may apply.

Fiduciary

In instances where a veteran is felt to lack the capacity or judgment to handle their VA monies, a fiduciary may be assigned through the local VA Regional Office. The fiduciary may be a family member or professional fiduciary. The fiduciary only manages VA monies such as compensation or pension benefits.

Representative Payee

In instances where it is felt that a person lacks the capacity to manage their Social Security benefits, the Social Security Administration (SSA) may assign someone as their representative payee. This can be a family member, other interested party or a guardian. For further information visit the SSA Web site at <http://www.ssa.gov>.

Advance Directives

Legal documents such as a Living Will, Durable Power of Attorney for Health Care, and Health Care Surrogate are collectively known as Advance Directives. Advance Directives can only be completed by an individual who at that time has the capacity to understand the content and consequences. Advance directives only go into effect when a person cannot communicate their wishes. If advance directives do not exist, the legal next of kin or guardian will be asked to make health care and end of life decisions for a patient.

Life Care Planning

A Life Care Plan is a tool to help manage the individual with a brain injury, over his/her life expectancy and to manage resources and finances. A Life Care Plan should be prepared by a person who is a Certified Life Care Planner with advanced life care planning, training and experience in traumatic brain injury, case management, or a medical care expert. The International Academy of Life Care Planners offers credentialing.

Veteran Service Organizations

AMVETS - <http://www.amvets.org>

Provides information and counseling to veterans on matters such as education, disability compensation, employment, hospitalization, pension, and other benefits.

VETERANS OF FOREIGN WARS (VFW) - <http://www.vfw.org>

Advocates for and with veterans of foreign wars. Provides counseling services and educational programs to veterans.

DISABLED AMERICAN VETERANS (DAV) - <http://www.dav.org>

Provides counseling services to veterans with disabilities and advocates on behalf of the veterans.

PARALYZED VETERANS OF AMERICA (PVA) - <http://www.pva.org>

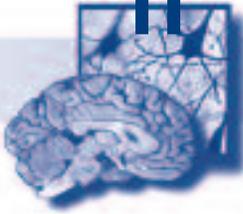
Provides services to veterans with disabilities on matters such as service-connected disability compensation, loan guarantees, medical care, educational benefits, government insurance, and benefit claims and appeals.

Client Assistance, Protection, and Advocacy

Each state offers Client Assistance Programs (CAP) and Protection and Advocacy (P & A) Systems. (Appendix E). These programs offer valuable resources to people with traumatic brain injury and their families who meet eligibility criteria. In the event an individual is not found eligible for services, the CAP or P & A System will provide assistance to locate other resources.

Point to remember:

- **Non-medical issues such as financial and legal concerns may be brought up by a patient/care giver in the ambulatory care setting and may impact the patient's overall health. It is important for the primary care provider to be aware of these issues and of the resources that can help.**



References

- AAN. (1997). Practice parameter: the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology*, 48, 581-585.
- Adams, J. H., Doyle, D. et al., (1989). Diffuse axonal injury in head injury: definition, diagnosis, and grading. *Histopathology* 15, 49-59.
- Adams, R., & Victor, M. (1985). *Craniocerebral Trauma. Principles of Neurology*. New York, McGraw-Hill.
- Ahmed, I., & Fujii, D. (1998). Posttraumatic Psychosis. *Seminars in Clinical Neuropsychiatry*, 3, 23-33.
- Akinwuntan, A. E., Feys, H., DeWeerd, W., Pauwels, J., Baten, G., & Strypstein, E. (2002). Determinants of driving after stroke. *Archives of Physical Medicine Rehabilitation*, 83, 334-41.
- Alexander, M. P. (1992). Neuropsychiatric correlates of persistent postconcussive syndrome. *Journal of Head Trauma Rehabilitation*, 7, 60-69.
- Alexander, M. P. (1995). Mild traumatic brain injury: pathophysiology, natural history, and clinical management. *Neurology*, 45, 1253-60.
- Alves, W., Macciocchi, S. N. et al., (1993). Postconcussive symptoms after uncomplicated mild head injury. *Journal of Head Trauma Rehabilitation*, 8, 48-59.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC: Author.
- Annegers, J. F., & Coan, S. P. (2000). The risks of epilepsy after traumatic brain injury. *Seizure*, 9, 453-7.
- Arciniegas, D. B., Topkoff, J., & Silver, J. M. (2000). Neuropsychiatric aspects of traumatic brain injury. *Current Treatment Options in Neurology*, 2, 160-186.
- Auerbach, S. (1989). The pathophysiology of traumatic brain injury. *Physical Medicine and Rehabilitation State of the Art Review: Traumatic Brain Injury*. Horn, L., and Cope, D. Philadelphia, Hanley, and Belfus. 3, 1-11.
- Barth, J. T., Macciocchi, S. N. et al., (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery*, 13, 529-533.

- Batchelor, J., Harvey, A. et al., (1995). Stroop colour word test as a measure of attentional deficit following mild head injury. *The Clinical Neuropsychologist*, 9, 180-186.
- Beard, C. M., Kokmen, E., O'Brien, P. C. et al., (1995). The prevalence of dementia is changing over time in Rochester, Minnesota. *Neurology*, 45, 75-79.
- Bennett, T. L., & Raymond, M. J. (1997). Emotional consequences and psychotherapy for individuals with mild brain injury. *Applied Neuropsychology*, 4, 55-61.
- Ben-Yishay, Y., & Prigatano, G. P. (1990). Cognitive remediation. Rosenthal, M., Bond, M. R. (Eds.). *Rehabilitation of the adult and child with traumatic brain injury* (2nd ed.), 393-409.
- Bigler, E. D. (2001). Structural and Functional Neuroimaging of Traumatic Brain Injury. McDevitt, J. (ed). *Traumatic Brain Injury*, 349-359.
- Binder, L. M. (1986). Persisting symptoms after mild head injury: A review of the postconcussive syndrome. *Journal of Clinical and Experimental Neuropsychology*, 4, 323-346.
- Binder, L. M. (1997). A review of mild head trauma. Part II: Clinical implications. *Journal of Clinical and Experimental Neuropsychology*, 19, 432-57.
- Binder L. M., Rohling, M. L., & Larrabee, G. L. (1997). A review of mild head trauma, part I: meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, 19, 432-457.
- Blumbergs, P. C., Scott, G. et al., (1994). Staining of amyloid precursor protein to study axonal damage in mild head injury. *The Lancet*, 344, (8929), 1055-6.
- Blumbergs, P. C., Scott, G. et al., (1995). Topography of axonal injury as defined by amyloid precursor protein and the sector scoring method in mild and severe closed head injury. *Journal of Neurotrauma*, 12, 565-572.
- Bohnen, N., Jolles, J., & Twijnstra, A. (1992). Neuropsychological deficits in patients with persistent symptoms six months after mild head injury. *Neurosurgery*, 30, 692-6.
- Bohnen, N., & Jolles, J. (1992). Neurobehavioral aspects of postconcussive symptoms after mild head injury. *Journal of Nervous and Mental Disease*, 180, 683-692.
- Bohnen, N., Jolles, J. et al., (1995). Late neurobehavioral symptoms after mild head injury. *Brain Injury*, 9, 27-33.
- Bohnen, N., VanZutphen, W. et al., (1994). Late outcome of mild head injury: results from a controlled postal survey. *Brain Injury*, 8, 701-708.
- Borcuk, P. (1995). Predictors of intracranial injury in patients with mild head injury. *Annals of Emergency Medicine*, 25, 731-736.

- Bourgeois, J. A., Behadir, N., & Minjares, S. (2002). Donepezil for cognitive deficits following traumatic brain injury: a case report. *Journal of Neuropsychiatry and Clinical Neurosciences*, 14, 463-64.
- Braakman, R., Gelpke, G. et al., (1980). Systematic selection of prognostic features in patients with severe head injury. *Neurosurgery*, 6, 362-370.
- Bray, R. M., & Marsden, M. E. (2000). Trends in substance use among US military personnel: The impact of changing demographic composition. *Substance Use & Misuse*, 35, 949-969.
- Brooke, M. M., Patterson, D. R., Questad, K. A., Cardenas, D., & Farrel-Roberts, L. (1992). The Treatment of Agitation During Initial Hospitalization After Traumatic Brain Injury. *Archives of Physical Medicine and Rehabilitation*, 79, 917-921.
- Brooke, M. M., Questad, K. A., Patterson, D. R., & Valois, T. A. (1992). Driving evaluation after traumatic brain injury. *American Journal of Physical Medicine and Rehabilitation*, 71, 177-182.
- Bryant, R., & Harvey A. G. (1999). Postconcussive symptoms and posttraumatic stress disorder after mild traumatic brain injury. *Journal of Nervous and Mental Disease*, 5, 302-305.
- Cameron, K. L., Yunker, C. A. et al., (1999). A standardized protocol for the initial evaluation and documentation of mild brain injury. *Journal of Athletic Training*, 34, 34-42.
- Challman, T. D., & Lipsky, J. J. (2000). Methylphenidate: its pharmacology and uses. *Mayo Clinic Procedures*, 75, 711-21.
- Chestnut, R., Ghajar, J. et al., (2000). Early indicators of prognosis in severe traumatic brain injury. *Journal of Neurotrauma* 17, 557-627.
- Chutka, D. S. et al., (1995). Drug prescribing for elderly patients. *Mayo Clinic Procedures*, 70, 685.
- Cicerone, K. D., & Kalmar, K. (1995). Persistent postconcussion syndrome: the structure of subjective complaints after mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 10, 1-17.
- Cifu, D. X., Kreutzer, J. S., Marwitz, J. H., Rosenthal, M., Englander, J., & High, W. (1996). Functional outcomes of older adults with traumatic brain injury: a prospective, multicenter analysis. *Archives of Physical Medicine & Rehabilitation*. 77, 883-888.
- Close, J., Ellis, M. et al., (1999). Prevention of falls in the elderly trial (PROFET): a randomized controlled trial. *The Lancet*, 353, 93.
- Cohen, H. (1994). Vestibular Rehabilitation Improves Daily Life Function. *American Journal of Occupational Therapy*, 48, 919-925.
- Control, C. F. D. (1999). *Traumatic Brain Injury in the United States: A Report to Congress*, Centers for Disease Control. 2001.

- Culotta, V. P., Sementilli, M. E., Gerold, K., & Watts, C. C. (1996). Clinicopathological heterogeneity in the classification of mild head injury. *Neurosurgery*, 38, 245-50.
- Deb, S., Lyons, I., & Koutzoukis, C. (1999). Neurobehavioural symptoms one year after a head injury. *British Journal of Psychiatry*, 174, 360-365.
- Definition of mild traumatic brain injury. (1993). *Journal of Head Trauma Rehabilitation*, 8, 86-87.
- DiGallo, A., Barton, J., & Parry-Jones, W. (1997). Road traffic accidents: Early psychological consequences in children and adolescents. *British Journal of Psychiatry*, 170, 358-362.
- Dikmen, S., McLean, A. et al., (1986). Neuropsychological and psychosocial consequences of minor head injury. *Journal of Neurology, Neurosurgery, & Psychiatry*, 49, 1227-32.
- Dixon, C. E., Taft, W. C. et al., (1993). Mechanisms of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8, 1-12.
- Drubach, D. A., Kelly, M. P., Winslow, M. M., & Flynn, J. P. (1993). Substance abuse as a factor in the causality, severity, and recurrence rate of traumatic brain injury. *Maryland Medical Journal*. 42, 989-993.
- Eisenberg, H., & Levin, H. (1989). Computed tomography and magnetic resonance imaging in mild to moderate head injury. Levin, H., Eisenberg, H., and Benton, A. (Eds). In *Mild Head Injury*. New York: Oxford University Press, 133-141.
- Englander, J., & Cifu, D. X. (1999). The older adult with traumatic brain injury. In Rosenthal, M., Griffith, E. R., Kreutzer, J. S., & Pentland, B. (eds): *Rehabilitation of the adult and child with traumatic brain injury*. 3rd ed. Philadelphia, FA Davis, 453-470.
- Englander, J., Hall, K., Simpson, T., & Chaffin, S. (1992). Mild traumatic brain injury in an insured population: Subjective complaints and return to employment. *Brain Injury*, 6, 161-6.
- Ewing, R., McCarthy, D. et al., (1980). Persisting effects of mild head injury observable during hypoxic stress. *Journal of Clinical Neuropsychology*, 2, 147-155.
- Falk, N. S., & Aksionoff, E. B. (1992). The primary care optometric evaluation of the traumatic brain injury patient. *Journal of the American Optometric Association*, 63, 547-553.
- Fann, J. R., Uomoto, J. M., & Katon, W. J. (2001). Cognitive improvement with treatment of depression following mild traumatic brain injury. *Psychosomatics*, 42, 48-54.
- Fann, J., Uomoto, J. et al., (2000). Sertraline in the treatment of major depression following mild traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences*, 12, 226-232.

- Fitzgerald, D. C. (1996). Head Trauma: Hearing Loss and Dizziness. *Journal of Trauma*, 40, 488-496.
- Fox, D., Lees-Haley, P. et al., (1995). Base rates of postconcussive symptoms in health maintenance organization patients and controls. *Neuropsychology*, 9, 606-611.
- Fox, D., Lees-Haley, P. et al., (1995). Post-concussive symptoms: Base rates and etiology in psychiatric patients. *The Clinical Neuropsychologist*, 9, 89-92.
- Furman, J. M., & Cass, S. P. (1999). Benign positional vertigo. *New England Journal of Medicine*, 341, 1590-6.
- Gasquoine, P. G. (1997). Postconcussion symptoms. *Neuropsychology Review*, 7, 77-85.
- Gennarelli, T. (1987). Cerebral concussions and diffuse brain injuries. *Head Injury*. P. Cooper. Baltimore, Williams, and Wilkins, 108-124.
- Gianutsos, R., Ramsey, G., & Perlin, R. (1987). Rehabilitative optometric services for survivors of brain injury. *Archives of Physical Medicine and Rehabilitation*, 69, 573-578.
- Gizzi, M. (1995). The efficacy of vestibular rehabilitation for patients with head trauma. *Journal of Head Trauma Rehabilitation*, 10, 60-77.
- Godbout, A. (1997). Structured habituation training for movement provoked vertigo after severe traumatic brain injury: a single-case experiment. *Brain Injury*, 11, 629-641.
- Goldberg, G. (2001). Mild traumatic brain injury and concussion. *Physical Medicine and Rehabilitation: State of the Art Rev*, 15, 363-398.
- Gouvier, W. D., Maxfield, M. W., Schweitzer, J. R., Horton, C. R., Shipp, M., Neilson, K., & Hale, P. N. (1989). Psychometric prediction of driving performance among the disabled. *Archives of Physical Medicine and Rehabilitation*, 70, 745-750.
- Gouvier, W., Cubic, B. et al., (1992). Postconcussion symptoms and daily stress in normal and head-injured college populations. *Archives of Clinical Neuropsychology*, 7, 193-211.
- Gouvier, W., Uddo-Crane, M. et al., (1988). Base rates of post-concussional symptoms. *Archives of Clinical Neuropsychology*, 3, 273-278.
- Graham, D. I. (1999). Pathophysiological aspects of injury and mechanisms of recovery. In Rosenthal, M., Griffith, E. R., Kreutzer, J. S., and Pentland, B. (eds): *Rehabilitation of the adult and child with traumatic brain injury*. 3rd ed. Philadelphia, 19-41.
- Graham, D., Lawrence, A. et al., (1987). Brain damage in non-missile head injury secondary to high intracranial pressure. *Neuropathology and Applied Neurobiology*, 13, 209-17.

- Gray, B., Ichise, M. et al., (1992). Technetium-99m-HMPAO SPECT in the evaluation of patients with a remote history of traumatic brain injury: a comparison with x-ray computed tomography. *Journal of Nuclear Medicine*, 33, 52-8.
- Gronwall, D. (1989). Cumulative and persisting effects of concussion on attention and cognition. *Mild Head Injury*. Levin, H., Eisenberg, H., and Benton, A. New York, Oxford University Press, 153-162.
- Hagen, C., Malkmus, D., Durham, P., & Bowman, K. (1979). Levels of cognitive functioning. In *Rehabilitation of the head injured adult: Comprehensive physical management*. Downey, CA: Professional Staff Association of Rancho Los Amigos Hospital.
- Hakkarainen, H., & Hakamies, L. (1978). Piracetam in the treatment of postconcussional syndrome. *European Neurology*, 17, 50-5.
- Hamm, R. J., Temple, M. D., O'Dell, D. M., Pike, B.R., & Lyeth B. G. (1996). Exposure to environmental complexity promotes recovery of cognitive function after traumatic brain injury. *Journal of Neurotrauma*, 13, 41-47.
- Hanks, R. A., Temkin, N., Machamer, J., & Dikmen, S. S. (1999). Emotional and behavioral adjustment after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 80, 991-997.
- Hartlage, L. C., Durant-Wilson, D., & Patch, P. C. (2001). Persistent neurobehavioral problems following mild traumatic brain injury. *Archives of Clinical Neuropsychology*, 16, 561-570.
- Haydel, M., Preston, C. et al., (2000). Indications for computed tomography in patients with minor head injury. *New England Journal of Medicine*, 343, 100-5.
- Herdman, S. J. (1990). Treatment of vestibular disorders in traumatically brain-injured patients. *Journal of Head Trauma Rehabilitation*, 5, 63-76.
- Hopewell, C. A. (2002). Driving assessments issues for practicing clinicians. *Journal of Head Trauma Rehabilitation*, 17, 48-61.
- Hsiang, J., Yeung, T. et al., (1997). High-risk mild head injury. *Journal of Neurosurgery*, 87, 234-238.
<http://www.nanonline.org/nandistance/mbti/modules/suppl/driving.html>.
- Jacobs, A., Put, E. et al., (1994). Prospective evaluation of technetium-99m-HMPAO SPECT in mild and moderate traumatic brain injury. *Journal of Nuclear Medicine*, 35, 942-947.
- Jagoda, A. S., Cantrill, S. V., Wears, R. L., Valadka, A., Gallagher, E. J., Gottesfeld, S. H., Pietrzak, M. P., Bolden, J., Bruns, J. J., Jr., & Zimmerman, R. (2002). American College of Emergency Physicians. Clinical policy: neuroimaging and decision making in adult mild traumatic brain injury in the acute setting. *Annals of Emergency Medicine*. 40, 231-249.
- Jagoda, A., Bolden, J. et al., (2002). Clinical policy: Emergency department management of mild traumatic brain injury (MTBI) in adults. *Annals of Emergency Medicine*, 39.

- Jenkins, A., Teasdale, G., Hadley, M. D., Macpherson, P., & Rowan, J. O. (1986). Brain lesions detected by magnetic resonance imaging in mild and severe head injuries. *The Lancet*, 2(8504), 445-446.
- Jennett, B., Teasdale, G. et al., (1979). Prognosis of patients with severe head injury. *Neurosurgery*, 4, 238-239.
- Jorge, R. E., Robinson, R. G., Arndt, S. V., Starkstein, S. E., Forrester, A. W., & Geisler, R. (1993). Depression Following Traumatic Brain Injury: a 1 year longitudinal study. *Journal of Affective Disorders*, 27, 233-243.
- Judd, T. (1989). Assessment and intervention for major symptoms of brain damage. Workshop presented in Managua, Nicaragua. Cited from secondary source (Sohlberg and Mateer, 1989).
- Kaelin, D. L., Cifu, D. X., & Matthies, B. (1996). Methylphenidate effect on attention deficit in the acutely brain-injured adult. *Archives of Physical Medicine and Rehabilitation*, 77, 6-9.
- Karzmark, P., Hall, K., & Englander, J. (1995). Late-onset post-concussion syndrome after mild head injury: The role of premorbid, injury-related, environmental, and personality factors. *Brain Injury*, 9, 21-26.
- Katz, R. T., Golden, R. S., Butter, J. et al., (1990). Driving safety after brain damage: follow-up of twenty-two patients with matched controls. *Archives of Physical Medicine and Rehabilitation*, 71, 133-137.
- Kay, T., Newman, B. et al., (1992). Toward a neuropsychological model of functional disability after mild traumatic brain injury. *Neuropsychology*, 6, 371-384.
- Kay, T., Harrington, D. E. et al., (1993). Definition of Mild Traumatic Brain Injury. *Journal of Head Trauma Rehabilitation*, 8, 86-87.
- Kelly, J. (2002). The diagnosis and management of concussion. Presentation, Pacific Coast Brain Injury Conference, Vancouver, Canada, October.
- Kersel, D. A., Marsh, N. V., Havill, J. H., & Sleigh, J. W. (2001). Psychosocial functioning during the year following severe traumatic brain injury. *Brain Injury*, 15, 683-696.
- King, N. (1996). Emotional, neuropsychological, and organic factors: Their use in the prediction of persisting postconcussion symptoms after moderate and mild head injuries. *Journal of Neurology, Neurosurgery, & Psychiatry*, 61, 75-81.
- Kline, A. E., Massucci, J. L., Marion, D. W., & Dixon, C. E. (2002). Attention of working memory and spatial acquisition deficits after a delayed and chronic bromocriptine treatment regimen in rats subjected to traumatic brain injury by controlled cortical impact. *Journal of Neurotrauma*, 19, 415-25.
- Kline, A. E., Yan, H. Q., Bao, J., Marion, D. W., & Dixon, C. E. (2000). Chronic methylphenidate treatment enhances water maze performance following traumatic brain injury in rats. *Neuroscience Letters*, 25, 163-66.

- Kolakowsky-Hayner, S. A., Gourley, E. V. 3rd., Kreutzer, J. S., Marwitz, J. H., Cifu, D. X., & Mckinley, W. O. (1999). Pre-injury substance abuse among persons with brain injury and persons with spinal cord injury. *Brain Injury*, 13, 571-581.
- Kolb, B., & Gibb, R. (1991). Environmental enrichment and cortical injury: Behavioral and anatomical consequences of frontal cortex lesions. *Cerebral Cortex*, 1, 189-98.
- Korteling, J. E., & Kaptein, N. A. (1996). Neuropsychological driving fitness tests for brain-damaged subjects. *Archives of Physical Medicine and Rehabilitation*, 77, 138-46.
- Kraus, J. F., & Nourjah, P. (1988). The Epidemiology of mild, uncomplicated brain injury. *Journal of Trauma*, 23, 1637-1643.
- Kraus, M. F. (1995). Neuropsychiatric sequelae of stroke and traumatic brain injury: the role of psychostimulants. *International Journal of Psychiatry Medicine*, 25, 39-51.
- Krauss, G. L., Ampaw, L., & Krumholz, A. (2001). Individual state driving restrictions for people with epilepsy in the US. *Neurology*, 57, 1780-5.
- Lal, S., Merbtiz, C. P., & Grip, J. C. (1988). Modification of function in head-injured patients with Sinemet. *Brain Injury*, 2, 225-33.
- Larrabee, G. J. (1997). Neuropsychological outcome, post concussion symptoms and forensic considerations in mild closed head injury. *Seminars in Clinical Neuropsychiatry*, 2, 196-206.
- Larrabee, G. J. (1999). Current Controversies in Mild Head Injury, in *The Evaluation and Treatment of Mild Traumatic Brain Injury*. Edited by Varney, N. R., and Roberts, R. J., Mahwah, New Jersey, Lawrence Erlbaum Associates, 327-346.
- Lees-Haley, P., & Brown, R. (1993). Neuropsychological complaint base rates of 170 personal injury claimants. *Archives of Clinical Neuropsychology*, 8, 203-209.
- Levin, H. S., Benton, A. L., & Grossman, R. G. (1982). *Neurobehavioral Consequences of Closed Head Injury*. New York, Oxford University Press.
- Levin, H. S., Amparo, E. et al., (1987). Magnetic resonance imaging and computerized tomography in relation to the neurobehavioral sequelae of mild and moderate head injuries. *Journal of Neurosurgery*, 66, 706-13.
- Levin, H. S., Mattis, S., Ruff, R. M. et al., (1987). Neurobehavioral outcome following minor head injury: three-center study. *Journal of Neurosurgery*, 66, 234-243.
- Levin, S., O'Donnell, V. et al., (1979). The Galveston Orientation and Amnesia Test: a practical scale to assess cognition after head injury. *Journal of Nervous and Mental Disease*, 167, 675-84.
- Lewin, I. (1992). *The Cost of Disorders of the Brain*. The National Foundation for the Brain. Washington, D.C.

- Liebert, M. A. (2000). Role of Antiseizure Prophylaxis Following Head Injury. *Journal of Neurotrauma*, 17, 549-552.
- Luis, C. A., Vanderploeg, R. D. et al., in press.
- Macciocchi, S. N., Barth, J. T. et al., (1996). Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery*, 39, 510-4.
- Marsh, H. V., & Smith, M. D. (1995). Post-concussion syndrome and the coping hypothesis. *Brain Injury*, 9, 553-562.
- Masanic, C. A., Bayley, M. T., VanReekum, R., & Simard, M. (2001). Open label study of donepezil in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 82, 896-901.
- Max, W., MacKenzie, E. J., & Rice, D. P. (1991). Head injuries: costs and consequences. *Journal of Head Trauma Rehabilitation*, 6, 76-91.
- Maxwell, W., Povlishock, J. et al., (1997). A mechanistic analysis of nondisruptive axonal injury: a review. *Journal of Neurotrauma*, 14, 419-40.
- Mazer, B. L., Korner-Bitensky, N. A., & Sofer, S. (1998). Predicting ability to drive after stroke. *Archives of Physical Medicine and Rehabilitation*, 79, 743-50.
- McAllister, T. W., Saykin, A. J., Flashman, L. A., Sparling, M. B., Johnson, S. C., Guerin, S. J., Mamourian, A. C., Weaver, J. B., & Yanofsky, N. (1999). Brain activation during working memory 1 month after mild traumatic brain injury: A functional MRI study. *Neurology*, 53, 1300-1308.
- McAllister, T. W., Sparling, M. B., Flashman, L. A., Guerin, S. J., Mamourian, A. C., & Saykin, A. J. (2001). Differential working memory load effects after mild traumatic brain injury. *NeuroImage*, 14, 1004-1012.
- McCauley, S. R., Boake, C., Levin, H. S., Contant, C. F., & Song, J. X. (2001). Postconcussional disorder following mild to moderate traumatic brain injury: Anxiety, depression, and social support as risk factors and comorbidities. *Journal of Clinical and Experimental Neuropsychology*, 23, 792-808.
- McCrea, M., Kelly, J. P. et al., (1997). Standardized assessment of concussion in football players. *Neurology*, 48, 586-8.
- McDowell, S., Whyte, J., & D'Esposito, M. (1998). Differential effect of a dopaminergic agonist on prefrontal function in traumatic brain injury patients. *Brain*, 121, 1155-64.
- McGowan, J. C., Yang, Y. H. et al., (2000). Magnetization transfer imaging in the detection of injury associated with mild head trauma. *American Journal of Neuroradiology*, 21, 875-80.
- McLean, A. Jr., Cardenas, D. D., Burgess, D., & Gamzu, E. (1991). Placebo controlled study of pramirecetam in young males with memory and cognitive problems resulting from head injury and anoxia. *Brain Injury*, 5, 375-380.

- McLean, A., Temkin, N. R., Dikmen, S., & Wyler, A. R. (1983). The behavioral sequelae of head injury. *Journal of Clinical Neuropsychology*, 5, 361-376.
- Meythaler, J. M., Depalma, L., Devivo, M. J., Guin-Renfroe, S., & Novack, T. A. (2001). Sertraline to improve arousal and alertness in severe traumatic brain injury secondary to motor vehicle crashes. *Brain Injury*, 15, 321-31.
- Meythaler, J. M., Peduzzi, J. et al., (2001). "Current concepts: Diffuse axonal injury—associated traumatic brain injury." *Archives of Physical Medicine and Rehabilitation*, 82, 1461-1471.
- Miller, H. (1961). Accident neurosis. *British Medical Journal*, 1, 919-925.
- Mittenberg, W., DiGiulio, D. V., Perrin, S., & Bass, A. E. (1992). Symptoms following mild head injury: Expectation as aetiology. *Journal of Neurology, Neurosurgery, and Psychiatry*, 55, 200-204.
- Mittenberg, W., & Strauman, S. (2000). Diagnosis of mild head injury and the postconcussion syndrome. *Journal of Head Trauma Rehabilitation*, 15, 783-791.
- Mittenberg, W., Tremont, G., Zielinski, R., Rayls, K., & Fichera, S. (1996). Cognitive behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology*, 11, 139-145.
- Mittenberg, W., Zielinski, R., & Fichera, S. (1993). Recovery from mild head injury: A treatment manual for patients. *Psychotherapy in Private Practice*, 12, 37-52.
- Morton, M. V., & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: A literature review and recommendations. *Brain Injury*, 9, 81-92.
- Newcombe, F., Rabbitt, P. et al., (1994). Minor head injury: pathophysiological or iatrogenic sequelae? *Journal of Neurology, Neurosurgery, and Psychiatry*, 57, 709-16.
- Nickels, J. L., Schneider, W. N., Dombovy, M. L., & Wong, T. M. (1994). Clinical use of amantadine in brain injury rehabilitation. *Brain Injury*, 8, 709-18.
- Oliver, J. H., Ponsford, J. L., & Curran, C. A. (1996). Outcome following traumatic brain injury: a comparison between 2 and 5 years after injury. *Brain Injury*, 10, 841-848.
- Ommaya, A. K., Ommaya, A. K., Dannenberg, A. L., & Salazar, A. M. (1996). Causation, incidence, and costs of traumatic brain injury in the U.S. military medical system. *Journal of Trauma-Injury, Infection & Critical Care*, 40, 211-217.
- Ommaya, A., Salazar, A. et al., (1996). Outcome after traumatic brain injury in the U.S. military medical system. *Journal of Trauma-Injury, Infection, & Critical Care*, 41, 972-975.
- Ommaya, A., Salazar, A. et al., (1999). Defense and Veterans Head Injury Program: A model injury registry. *Military Medicine Atlas of Injuries in the U.S. Armed Forces (Suppl)*, 7, 17-21.

- Passineau, M. J., Green, E. J., & Dietrich, W. D. (2001). Therapeutic effects of environmental enrichment on cognitive function and tissue integrity following severe traumatic brain injury in rats. *Experimental Neurology*, 168, 373-384.
- Pettus, E., Christman, C. et al., (1994). Traumatically induced altered membrane permeability: Its relationship to traumatically induced reactive axonal change. *Journal of Neurotrauma*, 11, 507-522.
- Plenger, P. M., Dixon, C. E., Castillo, R. M., Frankowski, R. F., Yablon, S. A., & Levin, H. S. (1996). Subacute methylphenidate treatment for moderate to moderately severe traumatic brain injury: a preliminary double-blind placebo-controlled study. *Archives of Physical Medicine and Rehabilitation*, 77, 536-40.
- Povlishock, J., Becker, D. et al., (1983). Axonal change in minor head injury. *Journal of Neuropathology and Experimental Neurology*, 42, 225-242.
- Powell, T. J., Collin, C., & Sutton, K. (1996). A follow-up study of patients hospitalized after minor head injury. *Disability and Rehabilitation: An International Multidisciplinary Journal*, 18, 231-237.
- Priddy, D. A., Johnson, P., & Lam, C. S. (1990). Driving after a severe head injury. *Brain Injury*, 4, 267-272.
- Prigatano, G. P. (1989). Bring it up in milieu: Toward effective traumatic brain injury rehabilitation interaction. *Rehabilitation Psychology*, 34, 135-144.
- Prigatano, G. P. (1999). *Principles of Neuropsychological Rehabilitation*. New York: Oxford University Press.
- Prigatano, G. P., & Schacter, D. L. (1991). *Awareness of Deficit After Brain Injury: Clinical and Theoretical Issues*. Oxford University Press, Oxford.
- Rao, V., & Lyketsos, C. (2000). Neuropsychiatric sequelae of traumatic brain injury. *Psychosomatics*, 41, 95-103.
- Report of the Sports Medicine Committee. (1990 (Revised 1991)). *Guidelines for the management of concussion in sports*, Colorado Medical Society.
- Rimel, R., Giordani, B. et al., (1981). Disability caused by minor head injury. *Neurosurgery*, 9, 221-228.
- Rosenthal, M., Kreutzer, J. S., Griffith, E. R., & Pentland, B. (1999). *Rehabilitation of the Adult and Child with Traumatic Brain Injury*, Third Edition. F.A. Davis, Philadelphia.
- Rothweiler, B., Temkin, N. R., & Dikmen, S. S. (1998). Aging effect on psychosocial outcome in traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 79, 881-887.
- Ruff, R. M., & Barth, J. T. (1999). *Neuropsychological Testing Considerations in Mild TBI*, in *Mild Traumatic Brain Injury*, CD-ROM, NAN Distance Program, Drexel University.

- Rugg-Gunn, F., Symms, M. et al., (2001). Diffusion imaging shows abnormalities after blunt head trauma when conventional magnetic resonance imaging is normal. *Journal of Neurology, Neurosurgery, and Psychiatry*, 70, 530-3.
- Rutherford, W. H., Merrett, J. D., & McDonald, J. R. (1979). Symptoms at one year following concussion from minor head injuries. *Injury*, 10, 225-230.
- Rutherford, W. (1989). Postconcussion symptoms: Relationship to acute neurological indices, individual differences, and circumstances of injury. *Mild head injury*. Levin, H., Eisenberg, H., and Benton, A. New York, Oxford University Press, 217-228.
- Ryan, L. M., Gouvier, W. D. et al., (1998). Predictors of postconcussion symptoms in mild head injury. *Archives of Clinical Neuropsychology*, 13, 147.
- Salazar, A., & Warden, D. (1999). Traumatic Brain Injury. *Scientific American Medicine*. New York, Scientific American, 11, 1-8.
- Sandel, M. E., Bell, K. R., & Michaud, L. J. (1998). Brain injury rehabilitation. 1. Traumatic brain injury: prevention, pathophysiology, and outcome prediction. *Archives of Physical Medicine & Rehabilitation*, 79 Suppl 1, S3-S9.
- Sarapata, M., Herrmann, D., Johnson, T., & Aycok, R. (1998). The role of head injury in cognitive functioning, emotional adjustment and criminal behavior. *Brain Injury*, 12, 821-842.
- Schanke, A-K., & Sundet, K. (2000). Comprehensive driving assessment: neuropsychological testing and on-road evaluation of brain injured patients. *Scandinavian Journal of Psychology*, 41, 113-121.
- Scheiman, M. (2002). Understanding and managing visual deficits: A guide for occupational therapists, 2nd edition. Slack Inc, Philadelphia, PA.
- Schneider, W. N., Drew-Cates, J., Wong, T. M., & Donbovy, M. L. (1999). Cognitive and behavioral efficacy of amantadine in acute traumatic brain injury: an initial double-blind placebo-controlled study. *Brain Injury*, 13, 863-72.
- Schultheis, M. T. (2000). Driving after Brain Injury - In National Academy of Neuropsychology web course Mild Traumatic Brain Injury - Module XIV.
- Schultheis, M. T., Matheis, R. J., Nead, R., & DeLuca, J. (2002). Driving behaviors following brain injury: self-report and motor vehicle records. *Journal of Head Trauma Rehabilitation*, 17, 38-47.
- Shumway-Cook, A. (1992). Rehabilitation of vestibular dysfunction in traumatic brain injury. *Physical Medicine and Rehabilitation Clinic*, 3(2), 355-368.
- Silver, J. M., Yudofsky, S. C., & Hales, R. E. (1994). *Neuropsychiatry of Traumatic Brain Injury*. Edited by Silver, J. M., Yudofsky, S. C., & Hales, R. E., Washington, D.C., American Psychiatric Press, Inc.
- Skoog, I. et al., (1993). A population-based study of dementia in 85-year-olds. *New England Journal of Medicine*, 328, 153.

- Smith, D., Meaney, D. et al., (1995). New magnetic resonance imaging techniques for the evaluation of traumatic brain injury. *Journal of Neurotrauma*, 12, 573-7.
- Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attentional training program. *Journal of Clinical and Experimental Neuropsychology*, 9, 117-130.
- Sohlberg, M. M., & Mateer, C. A. (1989). Training use of compensatory memory books: A three stage behavioral approach. *Journal of Clinical & Experimental Neuropsychology*, 11, 871-891.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Attention Process Training*. Wake Forest, Lash & Associates.
- Sohlberg, M. M., & Mateer, C. A. (1989). *Introduction to Cognitive Rehabilitation: Theory and Practice*. The Guilford Press, NY.
- Sohlberg, M. M., & Mateer, C. A. (2001). *Cognitive rehabilitation: An integrative neuropsychological approach*. Guilford Press, New York.
- Sosin, D. M., Sniezek, J. E., & Thurman, D. J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury*, 10, 47-54.
- Stablein, D., Miller, J. et al., (1980). Statistical methods for determining prognosis in severe head injury. *Neurosurgery*, 6, 243-248.
- Staplin, L., Lococo, K., & Sim, J. (1992). *Traffic maneuver problems of older drivers: final technical report*. FHWA Report FHWA-RD-92-092. McLean Virginia: Federal Highways Administration.
- Statler, K. D., Jenkins, L. W. et al., (2001). The simple model versus the super model: Translating experimental traumatic brain injury research to the bedside. *Journal of Neurotrauma*, 18, 1195-1206.
- Strich, S. J., & Oxon, D. M. (1961). Shearing of nerve fibers as a cause of brain damage due to head injury. *Lancet* (August), 443-449.
- Suchoff, I., Ciuffreda, K., & Kapoor, N. (Eds) (2001). *Visual and vestibular consequences of acquired brain injury*. Santa Ana, CA: Optometric Extension Program Foundation Inc.
- Suchoff, I., Kapoor, N., Waxman, R., & Ference, W. (1999). The occurrence of ocular and visual dysfunctions in an acquired brain-injured patient sample. *Journal of the American Optometric Association*, 70, 301-8.
- Szymanski, H., & Linn, R. (1992). Review of the postconcussion syndrome. *International Journal of Psychiatry in Medicine*, 22, 357-375.
- Taverni, J. P., Seliger, G., & Lichtman, S. W. (1998). Donepezil medicated memory improvement in traumatic brain injury during post-acute rehabilitation. *Brain Injury*, 12, 77-80.
- Taylor, J., Chadwick, D., & Johnson, T. (1996). Risk of accidents in drivers with epilepsy. *Journal of Neurology, Neurosurgery, and Psychiatry*, 60, 621-7.

- Thurman, D. J., Alverson, C., Dunn, K. A. et al., (1999). Traumatic brain injury in the United States: A public health perspective. *Journal of Head Trauma Rehabilitation*, 14, 602-615.
- Tinetti, M. E., Baker, D. I., McAvay, G., Claus, E.B., Garrett, P. et al., (1994). A multifactorial intervention to reduce the risk of falling among elderly people living in the community. *New England Journal of Medicine*, 331, 821-7.
- Vanderploeg, R. D., Curtiss, G., & Luis, C. A. (2001, August). Resolving Controversies in Mild TBI: Neuropsychological, Psychosocial, and Psychiatric Outcomes. Symposium at the Annual Convention of the American Psychological Association, San Francisco, CA.
- Warden, D. L., Bleiberg, J., Cameron, K. L., Ecklund, J., Walter, J., Sparling, M.B., Reeves, D., Reynolds, K. Y., & Arciero, R. (2001). Persistent prolongation of simple reaction time in sports concussion. *Neurology*, 57, 524-526.
- Warden, D., Sparling, M. et al., (in press). Military Textbook Chapter: Mild Traumatic Brain Injury.
- Washington Headquarters Services, Directorate for Information Operations and Reports, US Department of Defense. (n.d.). Selected manpower statistics: Fiscal year 2000. Retrieved May 29, 2003 from <http://web1.whs.osd.mil/MMID/PUBS.HTM>.
- Watanabe, T. K., & Sant, M. O. (2001). Common Medical Complications of Traumatic Brain Injury. *Traumatic Brain Injury*. McDeavitt, J. (ed), 283-294.
- Weidmann, K., JTL, W. et al., (1989). SPECT cerebral blood flow, MR imaging, and neuropsychological findings in traumatic head injury. *Neuropsychology*, 3, 267-281.
- Weinberg, R. M., Auerbach, S. H., & Moore, S. (1987). Pharmacologic treatment of cognitive deficits: a case study. *Brain Injury*, 1, 57-59.
- Whyte, J., Rosenthal, M., & Zuccarelli, L. A. (2000). Altered cellular anatomy and physiology of acute brain injury and spinal cord injury. *Critical Care Nursing Clinicians of North America*, 12, 403-11.
- Whyte, J., Hart, T., Schuster, K., Fleming, M., Polansky, M., & Coslett, H. B. (1997). Effects of methylphenidate on attentional function after traumatic brain injury. A randomized, placebo-controlled trial. *American Journal of Physical Medicine and Rehabilitation*, 76, 440-50.
- Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery*, 27, 422-428.
- Wong, P. P., Dornan, J., Schentag, C. T., Ip, R., & Keating, M. (1993). Statistical profile of traumatic brain injury: a Canadian rehabilitation population. *Brain Injury*, 7, 283-294.
- World Health Organization (1992). *International statistical classification of diseases and related health problems (10th ed.)*. Geneva, Switzerland: Author.

- Wrightson, P., & Gronwall, D. (1981). Time off work and symptoms after mild head injury. *Injury*, 12, 445-454.
- Wroblewski, B., Glenn, M. B., Cornblatt, R., Joseph, A. B., & Suduikis, S. (1993). Protriptyline as an alternative stimulant medication in patients with brain injury: a series of case reports. *Brain Injury*, 7, 353-62.
- Yablon, S., & Dostrow, V. (2001). Post Traumatic Seizures and Epilepsy. *Traumatic Brain Injury*. McDeavitt, J. (ed), 301-318.
- Yudofsky, S. C., & Hales, R. E. (2002). *The American Psychiatric Publishing Textbook of Neuropsychiatry and Clinical Neurosciences (4th Edition)*. Edited by Yudofsky, S. C., and Hales, R. E., Washington, D.C., American Psychiatric Publishing, Inc.
- Zhu, J., Hamm, R. J., Reeves, T. M., Povlishock, J. T., & Phillips, L. L. (2000). Post injury administration of L-deprenyl improves cognitive function and enhances neuroplasticity after traumatic brain injury. *Experimental Neurology*, 166, 136-52.

A

Appendix A: Long-Term Care

Long-Term Care

Long-term care is no longer an automatic referral to a nursing home. Today, long-term care has come to mean living with a brain injury in the least restrictive environment best suited to meet the individuals' needs. The individuals' physical, emotional, and behavioral needs, along with severity of injury (both physical and cognitive) determine which option is best. There are many options available for long-term living. Ideally, a placement facility will be familiar with the complexities of brain injury. If it is not possible to locate a facility familiar with brain injury, staff will need to educate the facility staff to help ensure a successful placement. Here are some alternative options:

- a. **Living in an independent apartment or private home** — This option allows the individual to remain in familiar surroundings and to live as independently as possible. The key to success is finding the right people to serve as supportive help or companion caregivers. If families are unable to provide this care there may be agencies in the community that can provide this service, or make recommendations, or contact the State Department of Vocational Services or Human Services. Funding is usually through private funds or community resources. **Respite Care** is an invaluable resource to caregivers and patients, offering a break from each other and allowing the caregiver time to themselves. A companion or sitter may provide respite in home. It may be provided outside of the home in an adult day care or assisted living or nursing facility, depending on the individuals needs. Respite care is time limited, ranging from a few hours per day, a week, or short-term placements. Funding is usually through private funds, but may also be provided through some private insurance plans or community resources. **Day Programs** may also be utilized to help patients remain in the home by giving them meaningful, engaging, structured activities during the day while the caregiver is at work. Many VA Medical Centers offer respite care and day programs.
- b. **Cooperative supported apartments** are available in some communities by local agencies. These apartments are for persons with brain injury who can live without much supervision. Usually

- a caseworker or case manager monitors the individual. Local Centers for Independent Living should have information on these facilities. Funding is usually through private funds or community resources.
- c. **Group homes are another option for long-term living.** These homes are usually licensed by the State Department of Health or Social Services. In group homes, the person engages in social and some leisure activities. Funding is usually through private funds or community resources.
 - d. **Community-based residential treatment centers** – these are larger community based facilities that provide rehabilitation and other therapeutic activities. They are staffed around the clock with trained staff and the facilities are licensed by the appropriate state agency. Funding during rehabilitation may be through private funding, insurance benefits, or community resources. Funding beyond the rehabilitation phase is usually covered by private funds.
 - e. **Assisted living facilities** – these are facilities that provide 24-hour supervision and are licensed by the state. Services vary but may include assistance with daily living activities, meal preparation, medication monitoring, transportation, and leisure activities. Therapy and nursing services are not usually provided but may be contracted or obtained through community, Medicaid, or insurance benefits.
 - f. **Skilled nursing facilities** – these state licensed facilities provide round the clock nursing care and supervision for persons with complex health and medical needs including vent care. They may provide subacute care or long-term (custodial) care. Cost for subacute care is usually covered through insurance or Medicare benefits or private funds. Long-term (custodial care) is usually funded through private funds or Medicaid.

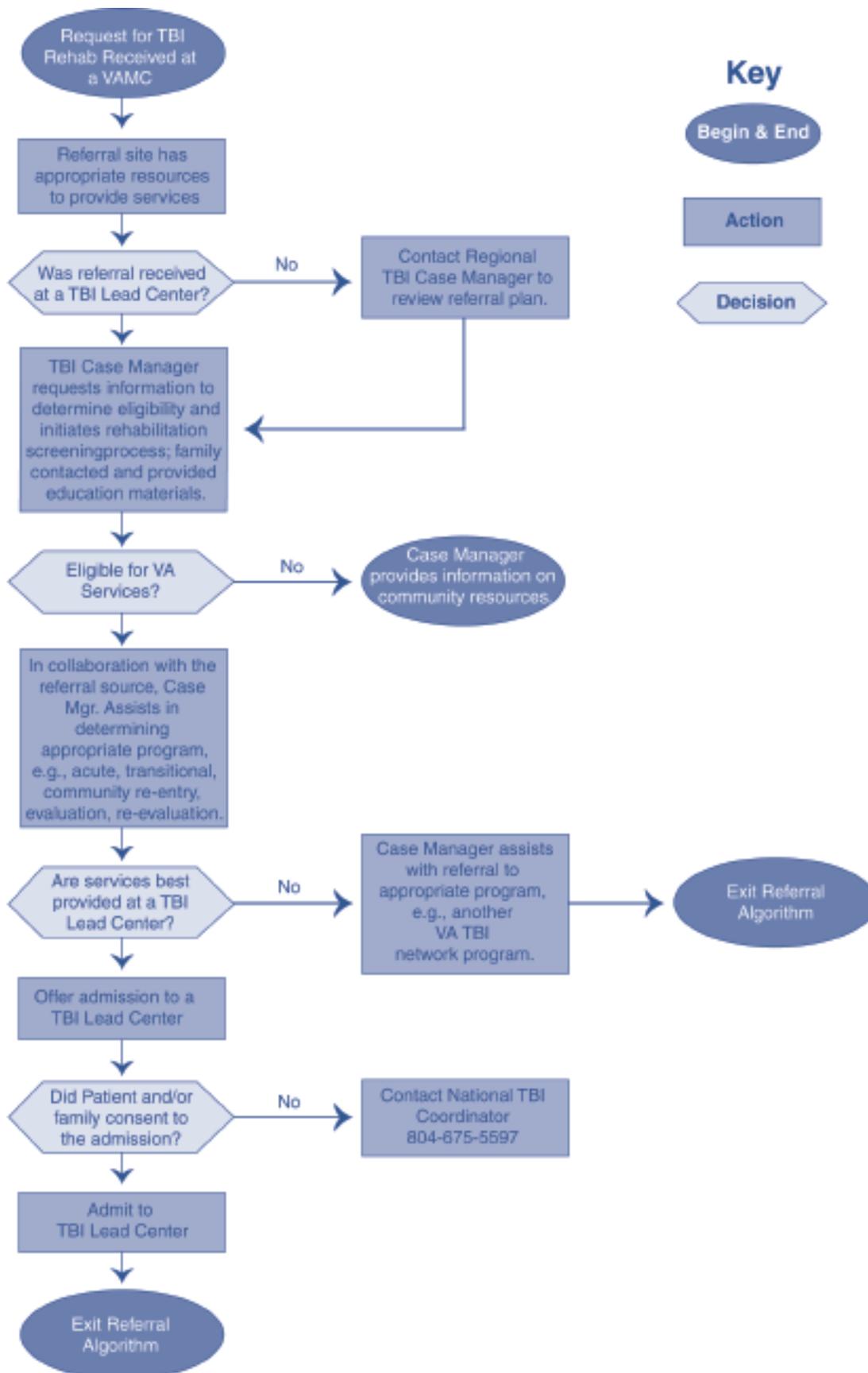
B

Appendix B: TBI Referral Algorithm

The TBI Referral Algorithm original is also located at the following Web site:

http://www.va.gov/health/rehab/tbi_referral_algorithm.doc

See Page 148 for full view of Algorithm



C

Appendix C: VBA Vocational Rehabilitation and Employment (VR&E) Program

Eligible veterans with disabilities may receive rehabilitation services through VBA's VR&E program, which may include an extended evaluation, independent living services, educational or vocational training, employment services, or a combination of any of these. If necessary, training may take place in a college or university, technical school, on-the-job training or in a specialized rehabilitation program (for individuals with severe disabilities). VA pays for the costs of tuition, books, supplies, and equipment and may pay for other special services (e.g., transportation, tutorial assistance, adaptive equipment, services). The VA may supply a monthly stipend in addition to compensation benefits received by the veteran. Throughout the program, the veteran will receive assistance in locating and obtaining a suitable job.

Veterans who are rated unemployable by the VA due to a service-connected disability may request an evaluation by VR&E, and if entitled, may participate in a program of rehabilitation services and receive assistance in securing employment under the VR&E program. Should the veteran then secure employment; his/her unemployability rating is protected from reduction until he/she has worked continuously for 12 months.

Additional information on VR&E is available at <http://www.vba.va.gov/bln/vre/index.htm>.

D

Appendix D: VBA VR&E Independent Living Eligibility and Services

The VR&E Program offers Independent Living (IL) services for veterans who meet the eligibility and entitlement requirements for the VR&E program, who demonstrate an IL need and who do not currently have employment as a goal.

Eligibility

- Must have a discharge other than dishonorable.
- Must have a compensable service-connected disability.

Entitlement

An evaluation by VBA's Vocational Rehabilitation and Employment (VR&E) Program will determine entitlement to Vocational Rehabilitation services which may include Independent Living services.

Program Services

The services, which may be provided to program participants and their families, include but are not limited to:

1. Evaluation of independent living potential;
2. Training in independent living skills;
3. Attendant care;
4. Health maintenance programs;
5. Identification of appropriate housing accommodations;
6. Reader services for those with visual impairments;
7. Interpreter services for those with hearing impairments; and
8. Special transportation assistance.

E

Appendix E: Client Assistance, Protection and Advocacy

Client Assistance Programs and Protection and Advocacy Systems investigate, negotiate, and mediate solutions to problems expressed by persons with developmental disabilities or mental illness, or applicants/clients of programs funded under the Rehabilitation Act, their families or agency representatives.

These programs also offer technical assistance to attorneys, government representatives, and service providers. They also provide legal counsel and litigation services to eligible individuals who are unable to attain adequate legal services in their communities. Training for advocates, consumers, volunteers, professionals, and others is also provided.

Client Assistance Programs and Protection and Advocacy Systems are listed in the Brain Injury Association's *National Directory of Brain Injury Rehabilitation Services* in the state resources section. For more information, contact the National Association of Protection and Advocacy Systems at (202) 408-9514. You can also search on the Internet using key words "Client Assistance Program."

Client Assistance Program

Client Assistance Programs (CAP) provides services to individuals seeking or receiving services under the Rehabilitation Act, (including federally funded vocational rehabilitation and independent living services).

The goal of CAP services is to help people with disabilities obtain information and access to the array of services available through programs, projects, and facilities funded under the Rehabilitation Act. CAP is administered by the U.S. Department of Education, Office of Special Education and Rehabilitation Services, Rehabilitation Services Administration.

Protection and Advocacy for Persons with Developmental Disabilities (PADD)

Protection and Advocacy Systems for Persons with Developmental Disabilities (PADD) were established to provide advocacy services to people

with developmental disabilities. Developmental disabilities are defined by the federal government as chronic and attributable to mental and/or physical impairment, which is evident prior to age 22. Such disabilities tend to be life-long and result in substantial limitations in three or more of the following major life activities: self-care, receptive and expressive language, learning, mobility, self-direction, and the capacity for independent living and/or economic self-sufficiency. An interagency project, PADD is administered by the Administration on Developmental Disabilities, the Center for Mental Health Services, Rehabilitation Services Administration, and the National Institute on Disability and Rehabilitation Research.

Protection and Advocacy for Individuals with Mental Illness (PAIMI)

The Protection and Advocacy for Individual persons with Mental Illness (PAIMI) was established to protect the rights of persons with mental illness under federal and state statutes, and investigate allegations of abuse and neglect of individuals residing in facilities. Individuals eligible for services must reside in facilities that provide 24-hour care and treatment, or have been discharged from such a facility within the previous 90 days. Programs represent individuals in certain situations involving prisons and jails, and transportation to and from facilities. The PAIMI system is administered by the U.S. Department of Health and Human Services, Center for Mental Health Services.

Protection and Advocacy for Individual Rights (PAIR)

The Protection and Advocacy Program for Individual Rights (PAIR) was established as a national program under the Rehabilitation Act in 1993. Through the PAIR programs, P & A systems nationwide protect and advocate for services to persons with disabilities who are not eligible for PADD or PAIMI programs, or whose issues do not fall within the jurisdiction of the Client Assistance Program. PAIR is administered by the U.S. Department of Education, Office of Special Education and Rehabilitative Services, Rehabilitation Services Administration (RSA).

F Appendix F: Additional Links

http://vaww.va.gov/health/rehab/TBI_Case_Managers.htm

<http://vaww.va.gov/health/rehab/SpecPrograms.htm>

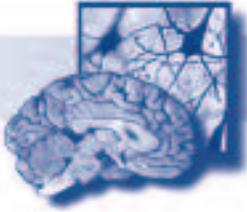
http://vaww.va.gov/health/rehab/TBI_REFERRAL_ALGORITHM.doc

<http://www.driver-ed.org>

[http://www.guideline.gov/summary/summary.aspx?doc_id=3121&nbr=2347
&string=brain+AND+injury](http://www.guideline.gov/summary/summary.aspx?doc_id=3121&nbr=2347&string=brain+AND+injury)

[http://www.guideline.gov/summary/summary.aspx?doc_id=3122&nbr=2348
&string=brain+AND+injury](http://www.guideline.gov/summary/summary.aspx?doc_id=3122&nbr=2348&string=brain+AND+injury)

http://www.cdc.gov/ncipc/pub-res/tbi_toolkit/physicians/mtbi/index.htm



CME Exam

- 1 The single most common cause of TBI is:
 - a. Falls
 - b. Firearms
 - c. Motor vehicle accidents
 - d. Sports injuries
2. Which of the following is **NOT** a defining characteristic of mild TBI?
 - a. Any alteration in mental state at the time of the accident
 - b. Glasgow Coma Scale score of 13-15
 - c. Indication that a physically traumatic event occurred
 - d. Positive CT or MRI findings
3. Individuals with a moderate to severe traumatic brain injury who are having trouble with attention, completing projects they start, and thinking as efficiently and effectively as before may have sustained damage to the:
 - a. Frontal lobes
 - b. Hypothalamic-pituitary axis
 - c. Occipital lobes
 - d. Parietal lobes
4. The most commonly used scale to quantify initial severity of brain injury is the:
 - a. American Academy of Neurology guidelines for grading level of concussion
 - b. Glasgow Coma Scale (GCS)
 - c. Post-concussion Symptom Checklist
 - d. Rancho Los Amigos Scales of Functioning
5. Which of the following is **NOT** a potential indication for an initial CT scan of the head following a mild TBI?
 - a. Age < 50 years
 - b. Drug or alcohol intoxication at time of injury
 - c. Emesis
 - d. Headache

6. What percentage of individuals who suffer MTBI experience significant ongoing symptoms or problems after 3 months?
 - a. Less than 5%
 - b. 10% to 30%
 - c. 50%
 - d. 75%
7. Which of the following is **NOT** true about “second impact syndrome”?
 - a. It can occur if individual suffers a second concussion, even 3 years after a previous concussion
 - b. It is a rare but often deadly condition
 - c. It may occur if an individual experiences two or more mild TBIs within a brief period of time
 - d. The length of period of rest following a first concussion necessary to avoid a “second impact syndrome” is currently unknown
8. Which of the following is **NOT** true?
 - a. At Rancho Levels I and II, rehabilitation goals include increasing the level of responsiveness and avoiding complications such as contractures
 - b. At Rancho Level IV and above, patients can actively participate to some degree in various rehabilitation therapies
 - c. Patients at Rancho Levels VI and below remain either somewhat confused or unresponsive
 - d. By Rancho Level VIII patients are entirely back to normal
9. Which of the following is **NOT** true about post-TBI Spasticity?
 - a. Spasticity can also lead to pain syndromes and skin breakdown
 - b. Spasticity is best treated on an outpatient basis through primary care clinics
 - c. Spasticity must be distinguished from other causes of resistance to passive movement including anxiety, joint pain, heterotopic ossification, and contractures
 - d. Whenever spasticity develops, a stretching, positioning, and range-of-motion program should be instituted
10. Initial medical assessment in the post-acute or chronic phase of TBI should include all of the following **EXCEPT**:
 - a. A CT or MRI scan of the head
 - b. An assessment of what education the patient has received about TBI and recovery
 - c. An understanding of the patient’s beliefs about brain injury and their beliefs about their long-term prognosis
 - d. Whether or not the patient has pending litigation related to TBI

11. Which of the following is **NOT** true about a neuropsychological evaluation?
 - a. It can assess current cognitive functioning and identify any potential cognitive problems
 - b. It can assess potential emotional and psychological factors that might be affecting current functioning
 - c. It can evaluate limb range of motion, mobility, and balance issues
 - d. It can help assess the impact of secondary factors, such as litigation, on current functioning

12. Which of the following is **NOT** a common medical symptom or problem in the individual with a moderate to severe TBI?
 - a. Dizziness and balance problems, while common after mild TBI, most frequently subside spontaneously
 - b. Headache is the most common pain complaint in this patient population but other pain sources are also frequent
 - c. Post-injury fatigue can have several contributing factors, so a wide range of possible causes should be explored including frustration, depression, and environmental demands
 - d. Spasticity usually emerges for the first time in the post-acute phase of TBI

13. What are the three most common cognitive problems following moderate to severe TBI?
 - a. a) attention/concentration, b) learning and memory, c) executive dysfunction
 - b. a) attention/concentration, b) speech/language, c) executive dysfunction
 - c. a) reasoning, b) abstraction, c) problem-solving
 - d. a) speech/language, b) visuospatial functioning, c) abstraction

14. Which of the following is true about cognitive ability recovery following moderate to severe TBI?
 - a. Although some chronic problems typically remain, in the first few months rapid cognitive improvement is the rule
 - b. Memory almost always recovers to pre-injury levels
 - c. Minor cognitive recovery may happen over the course of years and decades
 - d. Once brain injury has occurred, there is no recovery

15. Which of the following is true regarding cognitive rehabilitation in the individual with a **chronic TBI**?
- A new trial of compensatory cognitive training may be helpful if the environmental or social situation has changed resulting in a decrement in functional independence
 - A trial of cognitive therapy is usually indicated
 - Cognitive therapy is useful for learning and memory difficulties, but otherwise has no utility
 - In the chronic phase of recovery, cognitive therapy is effective in re-establishing lost cognitive abilities
16. The consequences of long-lasting post-TBI behavioral and emotional problems can include all of the following **EXCEPT**:
- Exhaustion of financial resources
 - Higher degree of sociability
 - Increase likelihood of divorce
 - Increased isolation over time
17. Principles guiding pharmacotherapy of emotional and behavior problems following TBI include all the following **EXCEPT**:
- Familiarity with side effects is crucial, and should help guide selection of medication
 - Higher levels of medications are likely needed to control symptoms in those with TBI
 - Medication selection should be symptom-based, not diagnostic category-based
 - Medication should be guided by hypothesis regarding the pathophysiology of the symptoms
18. Which of the following statements is **true**?
- Anticonvulsant medications such as carbamazepine (Tegretol) and Valproate sodium (Depakote) are not effective for the treatment of chronic agitation following TBI
 - Atypical antipsychotics (i.e., risperidone and quetiapine) are very useful for the treatment of chronic agitation following TBI
 - Benzodiazepines are a safe and effective medication for agitation in TBI and have few adverse side effects
 - Medication management of acute and chronic Agitation/Aggression in TBI is identical
19. All of the following are true **EXCEPT**:
- A TBI secondary to being struck as a pedestrian is more in the elderly than in younger adults
 - Falls are the leading cause of TBI in the elderly
 - Individuals over the age of 75 are at a higher risk for TBI than all other adults over the age of 25
 - Men generally account for a higher percentage of TBI than women do, except in individuals over the age of 65

20. In terms of prognosis or outcomes, all the following are true for the elderly **EXCEPT**:
- Chronic premorbid conditions such as cerebrovascular disease, alcoholism, advanced liver or renal disease, or diabetes potentially impede or prolong the recovery process in the elderly
 - The elderly have a more fragile physiologic status, which can result in a more destructive injury
 - The majority of elderly TBI survivors undergoing rehabilitation do not achieve functional improvements
 - The probability of poor outcome increases with advanced age
21. Which of the following explains why many individuals with TBI have limited energy or complain of feeling fatigued?
- Following TBI one's rate of metabolism often slows down, resulting in low energy
 - Their natural day/night cycle is disturbed following TBI resulting in less restful sleep
 - They have difficulty reading facial expressions and hence misperceive social cues
 - They have inhibitory control problems secondary to frontal lobe damage
22. All of the following help explain why many individuals with TBI seem more insensitive, egocentric, or irritable, **EXCEPT**:
- Often they have language impairment in comprehension, such as an aphasia
 - They have difficulty reading facial expressions and hence misperceive social cues
 - They have difficulty with multitasking and hence have trouble keeping up with normal social interchanges
 - They have frontal lobe impairment inhibitory control difficulties
23. Why is apathy, difficulty in moving forward on tasks, or passivity so common following TBI?
- Following TBI, many individuals become lazy and just want others to take care of them
 - Following TBI, one rate of metabolism often slows down, resulting in low energy
 - TBI commonly results in language comprehension problems, so they don't know what others are asking of them
 - The apathy following TBI is a result of both frontal lobe dysfunction and low mental energy

24. All of the following are helpful ways of dealing with anger in family members of those with TBI **EXCEPT**:
- Acknowledge that they are in a difficult situation and that not having clear answers is hard
 - Never respond in anger, not even in clipped tones
 - Respond in a direct, matter-of-fact, consistent manner
 - Try to spend as little time with them as possible to avoid their anger
25. Which of the following is **NOT** a factor in the ability to safely drive post-TBI?
- Impairments in arousal and attention
 - Impairments in executive abilities
 - Impairments in visuospatial abilities
 - Speech/language impairments such as dysarthria or Broca's aphasia
26. All of the following are true about post-TBI seizure problems and driving **EXCEPT**:
- A seizure during driving can impose a great threat to public safety
 - Most states require people with epilepsy to be seizure-free for a certain period, ranging from 3 to 12 months
 - The incidence of a post-TBI seizure disorder is estimated to be around 17%
 - There is a nation-wide law that restricts driving for patients with epilepsy
27. All of the following are true about post-TBI driving assessment **EXCEPT**:
- Computer-based driving simulators create a safe means of replicating actual driving conditions
 - There are no VAMCs with the capacity to perform driving evaluations
 - There is a listing of VAMCs able to perform both driving evaluations and driving rehabilitation listed on the VA Intranet
 - There is no universally accepted standardized driving evaluation for individuals who have sustained a TBI
28. Which best describes the needs of a TBI patient within the continuum of care?
- All brain injury patients needing long-term care require nursing home placement
 - All brain injury patients require acute rehabilitation
 - Brain injury patients have no further needs beyond acute rehabilitation.
 - TBI patients may vary in their progression throughout the continuum and require ongoing, coordinated disease management

29. To help maintain or gain independence the VA offers independent living services. To receive these benefits a veteran:
 - a. Is automatically eligible for this VBA program by virtue of their status as a veteran
 - b. Must be service-connected and submit application for benefits (VA-1900)
 - c. Need not apply, the military makes veterans aware of all possible VA benefits at discharge
 - d. Need not apply, they are eligible by virtue of his, or her service-connected status

30. When the primary care provider sees a veteran with a brain injury it is important to:
 - a. Be aware of the impact of common non-medical issues on overall health
 - b. Discourage the patient from contacting organizations involved in advocacy for the brain injured
 - c. Focus only on the immediate medical complaints of the patient
 - d. Minimize the patient's anxiety by encouraging them to focus on other issues