Chapter 15

TRAUMATIC BRAIN INJURY IN THE MILITARY POPULATION

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INTRODUCTION

Traumatic brain injury (TBI) is a very significant public health issue and the leading cause of death and disability in young people. The Centers for Disease Control and Prevention (CDC)[1] estimates that 1.4 million individuals sustain a TBI in the United States annually, with 50,000 deaths. About 80,000 to 90,000 individuals suffer permanent disability. The monetary cost to society is almost $50 billion annually when treatment costs, lost wages, disability, and death are considered.[2,3] Even more significant, at its most severe, TBI robs individuals of important aspects of their relationships, well-being, and happiness.

Service in the military, which includes both rigorous and often dangerous training, and exposure to the combat environment, places individuals at greater risk than the general population. Surprisingly, young adult men (the group with the highest rate of TBI in the civilian population), have about the same rate of TBI as young women in the military, a figure that underscores the inherent risk in service.[4] However, Ivins and colleagues,[5] in a recent paper examining trends in TBI-related hospitalizations in the active duty US Army during the 1990s, reported that the Army's TBI-related hospitalization rates decreased for all severity levels, both sexes, and all age categories during that decade. The paper also indicated that in the first half of the 1990s, many of the Army's adjusted TBI-related hospitalization rates, including the overall rate, were higher than the rates for US civilians 17 to 49 years of age. In the second half of the 1990s, most of the Army's adjusted TBI-related hospitalization rates, including the overall rate, were lower than civilian rates, with a 75% reduction overall. These decreases resulted in a relative improvement in the Army's TBI-related hospitalization rates over civilian rates by the late 1990s. This may be related to successful educational efforts or other factors.

Somewhat more difficult to quantify is the effect of TBI on military readiness. Because some potential consequences of TBI include slowed reaction, reduced speed of cognitive processing, and mood changes, the effects of even transitory symptoms could have implications for the deployability or fighting effectiveness of the service member. Additionally, there is the risk that the TBI, especially at the milder end of the spectrum, will be unrecognized. This chapter will describe (*a*) what TBI is, (*b*) how severity is determined, (*c*) common consequences of the injury, and (*d*) some treatment strategies. Furthermore, the identification and management of TBI in a military operational setting will be discussed. Although the entire spectrum of brain injury severity will be discussed, the focus of the chapter is on those with mild TBI (mTBI), as that is the population that is most likely to come to the attention of the military behavioral health provider. Additionally, the overlap of typical postconcussive symptoms with symptoms of mood, anxiety, or other disorders may make referral to such providers probable when an individual with such symptoms of unclear etiology is recognized.

The current conflicts in Iraq (Operation Iraqi Freedom) and Afghanistan (Operation Enduring Freedom) are different than past wars in terms of the survival rates of those injured. The current wounded-to-killed ratio in Iraq is more than 9:1,[6] compared to less than 3:1 in Vietnam and Korea, and approximately 2:1 in World War II.[7] This increased survival of wounded personnel is related to numerous factors including advanced in-theater medical care and superb protective equipment. With these new survival rates come increased numbers of those who may have experienced a TBI. Because the most common injury mechanism in the current conflict is blast, there are possibilities for TBI either through direct blast effect or secondary or tertiary blast effects. It is essential for the healthcare provider to be aware of the possibility that an injured service member may also have sustained a TBI. In many cases, this identification is early after the injury. There is greater potential, however, for more delayed recognition of such an injury, especially if the TBI is at the milder end of the severity spectrum. These "silent injuries" may have implications for functioning over the short or long term, and may affect recovery and rehabilitation of other more visible injuries.

ETIOLOGY AND DIAGNOSIS OF TRAUMATIC BRAIN INJURY

TBI is described as either penetrating or closed. A penetrating brain injury occurs when a foreign object or bone penetrates the dura surrounding the brain. (In the military setting, the object is most commonly a bullet or fragment.) In a closed TBI, penetration does not occur, but forces acting on the head cause damage to the brain. Although there is some variability in the definition of TBI, especially at the milder end of the spectrum, most accepted definitions (CDC,[8] American Congress of Rehabilitative Medicine [ACRM],[9] American Psychiatric Association [APA],[10] and World Health Organization [WHO][11]) have common elements. The Defense and Veterans Brain Injury Center (DVBIC) defines mTBI in a military operational setting as an

injury to the brain caused by an external force, with either an acceleration or deceleration mechanism (or both in some instances), from an event such as a blast, fall, direct impact, or motor vehicle accident. This trauma causes an alteration in mental status, typically resulting in the temporally related onset of symptoms such as headache, nausea, vomiting, dizziness or balance problems, fatigue, insomnia or other sleep disturbances, drowsiness, sensitivity to light or noise, blurred vision, and difficulty remembering or concentrating.

This operational definition of mTBI was established in 2006 by a workgroup of experts in the field of military operational medicine and TBI.[12] They drew from widely accepted definitions, such as those already mentioned (CDC, ACRM, APA, and WHO), as well as the National Athletic Trainers' Association position statement on management of sport-related concussion,[13] and the Prague sports concussion guidelines,[14] incorporating common criteria. These established definitions endorse biomechanical forces as the cause of concussion that results in an alteration of consciousness (AOC), including loss of consciousness (LOC), posttraumatic amnesia (PTA) or retrograde amnesia, or being dazed or confused at the time of the injury. An important aspect of this new operational definition of TBI is that LOC is not a required characteristic of concussion. That is, a service member does not have to have an LOC to have sustained a concussion or mTBI (these terms are used interchangeably). The group acknowledged the continued usefulness of these parameters and adopted them with a few changes. Variations from the established definitions included adding common combat-related mechanisms such as exposure to blast as a possible mechanism of injury, as well as adding a comprehensive list of TBI-related symptoms.

The majority of TBI experts in this workgroup agreed that symptoms are common following mTBI, but the presence of these symptoms is not mandatory to establish the diagnosis of mTBI. That is, although concussion to the brain may have occurred, it does not always result in self-reported symptoms. In some circumstances there may be measurable changes in functioning or performance, such as increased latency of response on measures of reaction time, even in the absence of changes noticeable to the injured individual.[15] It should be noted that wide consensus among the members of the workgroup supported the inclusion of an AOC in the definition of mTBI, including reports of feeling "dazed and confused" after a traumatic event. There are instances, both in the sports literature and military arena, of individuals who were involved in a traumatic event but did not report any AOC, yet these patients subsequently developed symptoms consistent with concussion, with onset temporally related to the event. Given these cases, a conservative approach might be that those individuals involved in events with an associated high risk for TBI, who report subsequent symptoms, be evaluated further. However, without supporting diagnostic evidence, these individuals do not meet the criteria for having sustained an mTBI. More research is needed to better characterize this group.[13]

Closed-TBI severity is characterized by duration of length of LOC and PTA, and initial, postresuscitation Glasgow Coma Scale (GCS) score (Table 15-1). Those individuals who meet these criteria as having sustained an mTBI, but have positive findings on imaging of the brain, are classified as having suffered a TBI of moderate severity, as these individuals are known to have outcomes similar to those who meet criteria for moderate TBI on the basis of length of coma or duration of LOC.[16,17] It is important to note that these designations describe the severity of the brain injury itself, and do not necessarily describe clinical outcomes or functionality in the future. Although there is greater chance for persisting problems as injury severity increases, many patients at the more severe end of the spectrum can have good outcomes, while some patients who were initially diagnosed as having a "mild" TBI go on

TABLE 15-1

TRAUMATIC BRAIN INJURY DESCRIPTION

Severity	GCS*	LOC	PTA
Mild	13–15	< 20 min–1 h	< 24 h
Moderate	9–12	1–24 h	24 h– < 7 days
Severe	3–8	> 24 h	> 7 days

GCS: Glasgow Coma Scale
LOC: loss of consciousness
PTA: posttraumatic amnesia
*The Glasgow Coma Scale (GCS) is the most commonly used scale to determine the severity of a brain injury. It must be noted, however, that a GCS score signifies the patient's "best" response. A patient could have a serious deficit that is not indicated by the GCS. Also, the score does not indicate the amount of stimulation required to get the patient to score at that level. A severe brain-injured patient usually has a GCS score of 3–8 and presents with a significant neurological deficit. The lowest GCS score is 3 (no neurological functioning). A patient with a severe brain injury also will experience loss of consciousness for more than 1 hour. Posttraumatic amnesia (PTA) refers to the loss of memory of events immediately following the injury. A typical question to ask to assess for PTA is, "What is the first thing you remember after your injury?" If the first memory occurred more than 24 hours after the injury, then, by definition, the patient has suffered a moderate-to-severe traumatic brain injury.

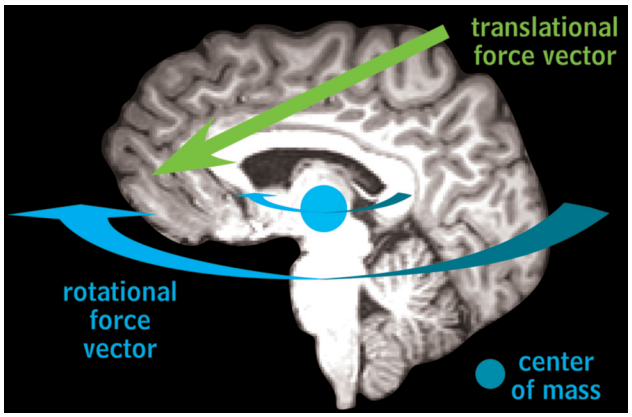

Figure 15-1. The physical forces exerted on the brain during most of the events that can cause traumatic brain injury are reasonably well understood. Both linear (green) and rotational (blue) forces can arise. The brain can be physically displaced within the skull. It can also be rotated or twisted. Rotational forces make the lower-density tissues (eg, cerebral cortex) move more quickly than the higher-density tissues (eg, subcortical white matter), resulting in stretching and shearing forces.

to have seemingly catastrophic changes in personal, social, and occupational functioning. Persistent postconcussional syndromes are discussed more fully later in this chapter.

Forces That Cause Traumatic Brain Injury

Traumatic brain injury may arise from many causes, including accidents, assaults, falls, and exposure to explosions. The physical forces exerted on the brain during most of the events that can cause TBI are reasonably well understood (Figure 15-1). The brain can be physically displaced within the skull by linear forces. It can also be rotated or twisted by angular or rotational forces. These forces make the lower-density tissues, particularly the outer layer of the brain (the cerebral hemispheres), move more quickly than the higher-density tissues that make up the core of the brain. They can also twist the brain around its central axis. Both types of movement result in stretching and shearing forces within the brain. Explosion-related brain injury is a new area of investigation. Although it is not yet proven that the changes in pressure that characterize the blast wave directly injure the brain as they do other parts of the body (eg, air-filled organs such as the lungs, tympanic membrane, and abdominal viscera), preliminary evidence suggests that this can occur.[18] It is clear that both the blast wave and the blast wind propel objects, including people, with sufficient power to cause TBI due to both linear and angular forces.

Vulnerable Areas and Injury Evolution

The most common primary injuries in TBI are traumatic axonal injury (TAI), contusion (bruising), and subdural hemorrhage. Movement of the brain within the skull can tear the surface veins that bridge from the brain to the dural venous sinus, resulting in a traumatic subdural hemorrhage (Figure 15-2).[19] The most common locations are the frontal and parietal convexities on the same side as the injury.[20] Subdural hemorrhage is crescent shaped on neuroimaging and conforms to the cerebral surface.[19,20] Its spread is limited by the dural reflections, and it rarely crosses the midline. When the brain moves within the skull enough to impact bone,

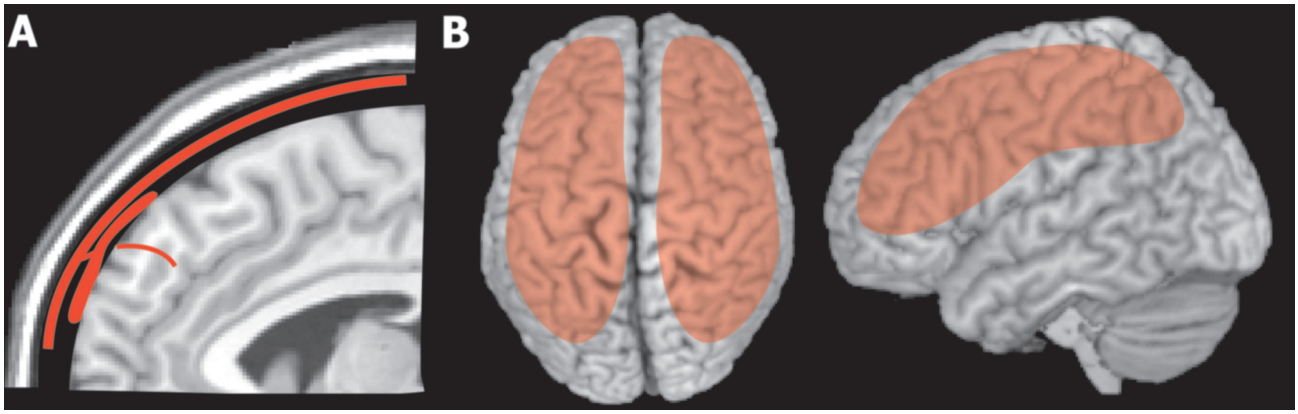

Figure 15-2. Traumatic subdural hemorrhage occurs when the brain moves within the skull enough to tear the vessels that bridge from the brain surface to the dural venous sinus (**a**). The most common locations are the frontal and parietal convexities on the same side as the injury (**b**).

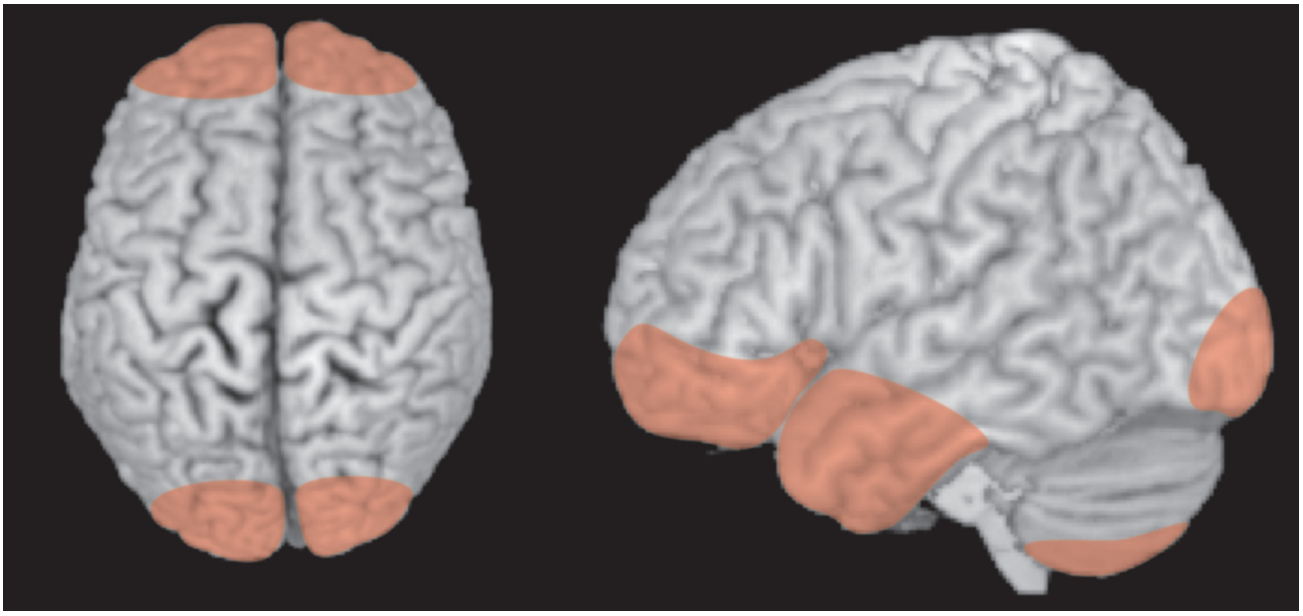

Figure 15-3. Contusions occur when the brain moves within the skull enough to impact bone, causing bruising. The most common locations are the superficial gray matter of the inferior, lateral, and anterior aspects of the frontal and temporal lobes, with the occipital poles or cerebellum less often involved.

it can cause contusion of the brain parenchyma (Figure 15-3). The most common locations are the superficial gray matter of the inferior, lateral, and anterior aspects of the frontal and temporal lobes; the occipital poles or cerebellum are less often involved.[20]

The most likely injury to occur in mTBI is TAI, also called diffuse axonal injury. Certain areas of the brain are particularly vulnerable to TAI (Figure 15-4). One is the corticomedullary (gray matter–white matter) junction, particularly in the frontal and temporal lobes. Areas of very concentrated white matter, such as the corpus callosum and internal capsule, are also quite vulnerable. Finally, the deep gray matter and upper brainstem are also frequent sites of TAI.[20] TAI is the result of shearing, stretching, or angular forces pulling on axons and small vessels.[21]

The old view of TAI was that these forces produced mechanical tearing of axons. Although this can occur, it is now considered to be unusual. The present understanding is that TAI is a progressive injury.[22,23] Stretching of the axon and its enclosing myelin sheath results in increased permeability, allowing an influx of calcium. This, in turn, triggers events that loosen the normally tight myelin sheath in the vicinity of the injury and also cause damage to the axon's cytoskeleton (a complex network of microtubules and neurofilaments that form the internal supporting structure for neurons).

The integrity of the cytoskeleton is essential for many vital functions, including transport of substances from the cell body out to the axon and dendrites

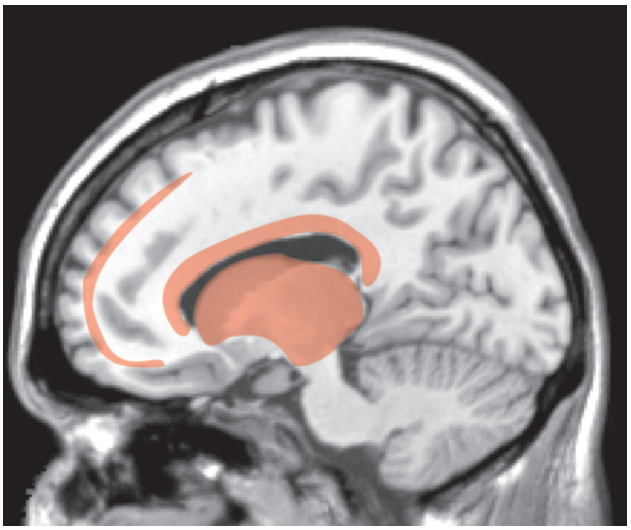

Figure 15-4. Traumatic axonal injury (TAI) results when shearing, stretching, or angular forces pull on axons and small vessels. Impaired axonal transport leads to focal axonal swelling and (after several hours) may result in axonal disconnection. The most common locations are the corticomedullary junctions (particularly frontotemporal), internal capsule, deep gray matter, upper brainstem, and corpus callosum.

(axoplasmic transport). Breakdown of the cytoskeleton disrupts transport of materials within the axon. Material collects proximal to the injury, leading to focal axonal swelling. If the swelling continues to progress, the axon eventually detaches. The proximal end seals and continues to swell, leading to the formation of the classic axonal retraction ball. The distal portion of the axon undergoes Wallerian degeneration, a process that can take several months in humans.[24] (This account is based primarily on the study of heavily myelinated, large-diameter axons, such as those that make up the majority of the corpus callosum and internal capsule.)

Growing evidence suggests that the injury cascade may be quite different in other populations of axons (eg, small-diameter, unmyelinated axons) and vulnerable areas (eg, injuries close to cell bodies, as occurs at the corticomedullary junction and in deep gray matter).[24] This has important implications both for neuropathological identification of TAI and for potential therapeutic targets.[24] Secondary processes can cause further brain injury.[24–27] For example, there may be a widespread release of glutamate (an excitatory neurotransmitter), with the potential for lethal overstimulation of neurons (excitotoxicity). Release of blood into brain tissue has toxic effects, including the possibility of triggering cerebral vasospasm, which increases the risk for ischemia.

DETERMINING SEVERITY OF TRAUMATIC BRAIN INJURY

Structural Neuroimaging

TAI is characterized by multiple small injuries, often widely dispersed in the brain rather than clustered. As a result, its identification on structural neuroimaging can be quite challenging. Although magnetic resonance imaging (MRI) is more sensitive than computed tomography in detecting this type of brain injury, even MRI is often negative.[20,22,28–31] Gradient-echo MRI is especially useful because it can visualize even very small areas of hemorrhage, as often occur in conjunction with axonal injury.[32–36] This type of MRI is sensitive to alterations in magnetic susceptibility. Presence of blood within tissue creates a local magnetic field disturbance, causing a decrease in signal intensity in the area containing blood. Areas of TAI not containing hemorrhage are better seen on T2*-weighted spin-echo MRI, where they will appear bright. Particularly useful is the FLAIR (fluid attenuated inversion recovery) sequence, in which the normally high signal intensity of cerebrospinal fluid is suppressed, making lesions near the ventricles much easier to identify.[37] A newer method of MRI that shows great promise for improved imaging of TAI is diffusion tensor imaging, in which the image is made sensitive to the speed and direction of water diffusion.[38,39] In gray matter and fluid, water diffuses at the same speed in all directions (isotropic diffusion). In white matter, water diffuses much more quickly along the length of axons (fibers, tracts) than across them (anisotropic diffusion). Thus, diffusion tensor imaging provides a way to examine the structural integrity of the white matter.

Combat-Related Traumatic Brain Injury

There is increasing evidence that combat-related TBIs are a frequent occurrence. Approximately one half of combat-related injuries, many a result of explosions, involve the head or neck.[40,41] Several studies from the DVBIC document the presence of TBI in soldiers returned from Afghanistan or Iraq.[42–45] Common postconcussive symptoms included headache (47%), irritability or aggression (45%), difficulty with memory (46%), and difficulty with concentration (41%).[43] Studies of active duty soldiers suggest that the majority of brain injuries would be classified as mild, as indicated by either no or only brief LOC.[44,46] In most cases these less severe injuries would not have required medical evacuation.[45]

It is well known that mTBI in civilians is underrecognized by both medical personnel and patients, resulting in significant underreporting.[47,48] A similar situation exists in the military in that combat-related mTBI may often be unrecognized by both medical personnel and service members.[44,46,49] DVBIC has recently released a new assessment tool—the Military Acute Concussion Evaluation (MACE)—to facilitate identification and evaluation of postconcussive symptoms following combat-related brain injuries. The literature on concussion strongly supports the need to fully evaluate anyone experiencing alteration in mental status (eg, dazed, confused, "saw stars," LOC) as a result of exposure to conditions that can injure the brain.[26] Of particular importance is evaluation of memory, as presence of PTA appears to be associated with higher rates of cognitive difficulties during the first few weeks after injury.[49]

Postconcussive symptoms may include altered consciousness (drowsiness, confusion, lethargy), headache, amnesia, nausea or vomiting, fatigue, irritability, restlessness, auditory or vestibular disturbances (balance problems, ringing in the ears, dizziness, hearing changes, sensitivity to noise), visual disturbances (blurred vision, sensitivity to light, double vision), gait

abnormalities, and problems sleeping.[50,51] Although most patients recover well from mTBI, the rate of recovery is highly variable. During this period of healing individuals are likely to perform below their normal level on both physical and cognitive tasks. Evidence also suggests that the brain is more vulnerable to reinjury during this period. Therefore, in the context of active combat, premature return to duty increases risks for both the individual and the team.[26]

Many mild brain injuries will not become evident until after the service member returns home. Although the majority of people recover well following mTBI, a significant minority do not.[25] Common problems in the chronic phase following TBI include headaches, dizziness, fatigue, sleep disturbances, spasticity, vestibular impairments, visual difficulties, personality changes, and cognitive or emotional deficits. Some studies indicate that as many as one third of these patients may have psychiatric symptoms within the first year postinjury.[52–54]

COMMON COGNITIVE SEQUELAE

An examination of cognitive dysfunction following TBI must take into account myriad factors, including severity and nature of the injury (focal vs diffuse), time since injury, motivation of the subject, and other injuries. In the case of mTBI, there is agreement that there are short-term cognitive consequences of the injury, affecting various aspects of attention, speed of processing, and other cognitive domains. Even in the absence of self-reported cognitive dysfunction or other symptoms, decrements in reaction time have been reported.[14] The longer-term cognitive consequences from mTBI are less clear.

Iverson and colleagues[55] report data on a group of young adult patients seen in the trauma service of a Pennsylvania general hospital (1991–1994) with mTBIs (based on postinjury GCS scores) who completed neuropsychological testing within the first week postinjury. Those who met severity inclusion criteria and were under age 40 accounted for 484 of the 1,695 total patients. Most were men, with motor vehicle accidents being the most common cause of injury. Most patients (82%) had GCS scores of 15, with the remaining ones having GCS scores of 14. About 56% had known LOC, with about 18% negative, and the rest unclear or unknown. The prevalence of intracranial abnormalities on day-of-injury computed tomography was 11.8%. The rest of these patients had negative (68.6%) or missing (19.6%) results. The patients were split into two groups on the basis of differences on the Galveston Orientation and Amnesia Test (GOAT). The groups were relatively evenly split by scores above 90 and those below (range 0–100). Scores between 90 and 75 are not low enough to indicate frank PTA, but do suggest some ongoing confusion. All those patients were administered a brief (30–45 minutes) but wide-ranging neuropsychological test battery. Overall, the results suggested that when trauma patients are evaluated shortly after an mTBI, the presence of posttraumatic confusion is related to worse short-term neuropsychological outcome in that subjects with lower GOAT scores had significantly worse scores on measures of simple attention, verbal learning, verbal and visual memory, and some aspects of executive functioning than did subjects in the above-90 range on GOAT. Furthermore, brief traumatic LOC (less than 5 minutes) is not related to short-term neuropsychological outcome (there were no significant differences in scores based solely on whether or not there was brief LOC).

Belanger and colleagues[56] conducted a metaanalysis of the relevant literature based on 39 studies involving 1,463 cases of mTBI and 1,191 control cases to determine the impact of mTBI across nine cognitive domains—(1) global cognitive ability, (2) attention, (3) executive functions, (4) fluency, (5) memory acquisition, (6) delayed memory, (7) language, (8) visuospatial skill, and (9) motor functions. The overall effect of mTBI on neuropsychological functioning was moderate (d = .54). However, findings were influenced by cognitive domain, time since injury, patient characteristics, and sampling methods. Acute effects (less than 3 months postinjury) of mTBI were greatest for delayed memory and fluency. In unselected or prospective samples, the overall analysis revealed no residual neuropsychological impairment by 3 months postinjury. In contrast, clinic-based samples and samples including participants in litigation were associated with greater cognitive sequelae of mTBI.

In another metaanalysis focused on the sports concussion literature, the authors reviewed studies from 1970 to 2004 from which 21 studies met inclusion criteria, with 790 cases of mTBI and 2,016 control cases.[57] The overall effect for size of concussion on cognition was 0.49, with delayed memory, memory acquisition, and global cognitive functioning showing the greatest effects acutely. No residual effects were found from the group tested over 1 week postinjury.

Postconcussive Disorder

A WHO analysis of outcome in mTBI concludes that although acute symptoms are common, the vast majority of individuals have good resolution of their

mTBI symptoms by 3 months postinjury and many quite sooner.[58] The authors acknowledge (and this is consistent with clinical practice in both military and civilian settings) that there are individuals who show persistent symptoms. The fourth edition of the *Diagnostic and Statistical Manual for Mental Disorders* proposed diagnostic criteria for a postconcussional disorder in its appendix of provisional diagnostic criteria sets.[10] Diagnosis of a postconcussional disorder required a "significant cerebral concussion" with measured cognitive deficit and the presence of at least three of eight symptoms—(1) fatigue, (2) sleep disturbance, (3) headache, (4) dizziness, (5) irritability, (6) anxiety/depression, (7) personality change, and (8) apathy—with onset after injury and persistence past 3 months. (See Exhibit 15-1 for full criteria for postconcussional disorder.) Criteria have also been included in the *International Classification of Diseases, 10th Revision* (ICD-10).[59] These criteria require a history of TBI and the presence of three of eight symptoms—(1) headache, (2) dizziness, (3) fatigue, (4) irritability, (5) insomnia, (6) concentration problems, (7) memory difficulty, and (8) reduced tolerance of stress, emotion, or alcohol.

Boake et al[60] compared these two diagnostic sets and concluded that there was a large difference between the prevalence of postconcussive syndrome using these two criteria sets, with the ICD-10 criteria being more inclusive. The differences suggest that there could be disagreement in diagnosis depending on which criteria set is used. Furthermore, they concluded that both criteria sets had limited specificity to TBI (if the history of TBI itself was removed as a criteria), as the other criteria could be met after general trauma, whether or not the brain was injured. The authors point out that this finding is supportive of previous CDC recommendations—namely that postconcussional symptoms in themselves are not sufficient to make a diagnosis of mTBI. Iverson and colleagues,[61] in a review article on outcome from mTBI, also report that postconcussion symptoms are common in healthy subjects, those without a history of TBI, and in various patient groups.

The existence of a group of patients with persistent symptoms has been controversial. The scope of the problem itself is difficult to determine (clinical lore has set the figure at 10%–20% although it is likely less than 5% of the total number of individuals who suffer mTBI; see Iverson[55] for a complete discussion). There is also disagreement concerning the cause of these persistent symptoms. Various authors have attributed them to different causes, some believing them to reflect the injury itself, and others attributing these symptoms to a multicausal etiology, with premorbid personality characteristics, social-psychological factors, and exaggeration

EXHIBIT 15-1

RESEARCH CRITERIA FOR POST-CONCUSSIONAL DISORDER

A. History of closed head injury:
- Causing cerebral concussion
- Symptoms include:
  - loss of consciousness
  - posttraumatic amnesia
  - posttraumatic onset of seizuers (less common)

B. Difficulty (based on cognitive evaluation) in:
- attention, such as:
  - concentration
  - shifting focus of attention
  - performing simultaneous cognitive tasks

  or
- memory, such as:
  - learning
  - recalling information

C. Three or more of the following occuring post-trauma (and lasting 3 or more months):
- easy fatigue
- disordered sleep
- headache
- vertigo/dizziness
- irritability/aggression on little or no provocation
- anxiety/depression/affective lability
- changes in personality
- apathy/lack of spontaneity

D. "B" and "C" symptoms:
- have onset following head trauma

  or
- represent a substantial worsening of preexisting symptoms

E. Disturbance causes significant impairment/significant worsening in:
- social/occupational functioning (adults)
- school/academic performance (school-age children)

F. Symptoms do not meet criteria for/not better accounted for by:
- dementia due to head trauma
- another mental disorder such as:
  - amnestic disorder due to head trauma
  - personality change due to head trauma

Adapted with permission from American Psychiatric Association. *Diagnostic and Statistical Manual*. 4th ed. Washington, DC: APA; 2000: 761–762.

(either conscious or unconscious) playing a role.

The Relationship Between Posttraumatic Stress Disorder and Traumatic Brain Injury

Posttraumatic stress disorder (PTSD) can result from highly stressful experiences, such as being in combat or being injured in combat. Hoge and colleagues[62] reported that for all groups surveyed after their deployment, there was a strong relationship between intensity of combat experiences (killing the enemy, being shot at) and exposure to traumatic combat-related events (handling dead bodies, knowing someone who was killed), and the prevalence of PTSD. Among service members in OIF, the prevalence of PTSD increased with the number of firefights during deployment (with increases to 19.3% for those involved in more than five firefights). The rates of PTSD were significantly associated with having been wounded or injured (odds ratio for those deployed to Iraq, 3.27; odds ratio for those deployed to Afghanistan, 2.49). This is consistent with the findings of Koren et al[63] in a small but well-designed study looking at rates of PTSD in injured Israeli war veterans. That study clearly indicated that bodily injury is a risk factor for PTSD, with odds of developing PTSD following traumatic injury approximately eight times higher than following injury-free emotional trauma. The authors suggest that even this rather high figure might be an underestimate of the rate because 35% of these injured combat veterans had refused to participate in the study.

Controversy exists, however, regarding the rate and risk factors for PTSD following TBI. Bombardier and colleagues[64] report a cumulative rate of 11.3% in a mixed sample of those with TBI ranging from mild to severe over a 6-month period. Furthermore, their findings were consistent with those of previous studies suggesting that more severe TBI may be protective with regard to the development of PTSD, that is, the AOC or LOC associated with TBI may lessen the individual's ability to reexperience the trauma.[65] Risk factors for developing PTSD following TBI include having less education, feeling terrified or helpless, and having major depression.[64]

In-Theater Management

The in-theater management of TBI depends on its severity. (There are guidelines recently released on the management of more severe combat TBI,[66] but in-depth discussion of these guidelines is beyond the scope of this chapter.) The clinical management of the mTBI patient in a military occupational setting was addressed in the 2006 DVBIC workgroup. A graphical representation of the level-I practice guideline is shown in Figure 15-5. This guideline relies on the use of the MACE (see Exhibit 15-2 for full instrument), a tool developed by the DVBIC. The MACE has both a history and evaluation component. The history component can confirm the diagnosis of mTBI after establishing that a trauma has occurred and during the course of this traumatic event, the service member experienced an AOC. An AOC can be defined on a continuum from "dazed and confused," to not remembering the injury, to an LOC. The evaluation component, designed to be easily used by medics and corpsmen, can be administered within 5 minutes. It utilizes the Standardized Assessment of Concussion[67] to preliminarily document neurocognitive deficits in four cognitive domains: (1) orientation, (2) immediate memory, (3) concentration, and (4) delayed recall.

The Relationship Between Substance Abuse and Traumatic Brain Injury

The relationship between TBI and substance use and abuse is an important one that presents a number of complexities for understanding their interrelatedness. Intoxication is in itself a risk factor for TBI. That is, TBI can result from unintentional alcohol-related causes such as motor vehicle accidents or falls while intoxicated. More intentional alcohol-related TBI can result from other causes such as assault while intoxicated (both on the part of the victim and perpetrator), mate-related abuse, and direct self-harm such as suicide attempts.[1] Savola, Niemelä, and Hillbom[68] investigated the relationship of different patterns of alcohol intake to various types of trauma. They examined the alcohol consumption in a series of 385 consecutive trauma admissions. On admission, 51% of the patients had alcohol in their blood. Binge drinking was the predominant (78%) drinking pattern of alcohol intake, and assaults, falls, and biking accidents were the most frequent causes of trauma. Dependent alcohol drinking and binge drinking were found to be significantly more common among patients with head trauma than in those with other types of trauma. The relative risk for head injury markedly increased with increasing blood alcohol levels.

A TBI can also exacerbate previous substance abuse or lead to behavioral and personality changes that could lead to alcohol or drug misuse. Prior history of a substance abuse disorder is a risk factor for greater morbidity[69] and excessive use following TBI.[70] Substance-use disorders following TBI adversely affect neuropsychological functioning, subjective well-being, employment, and involvement with the criminal justice system.[71–74]

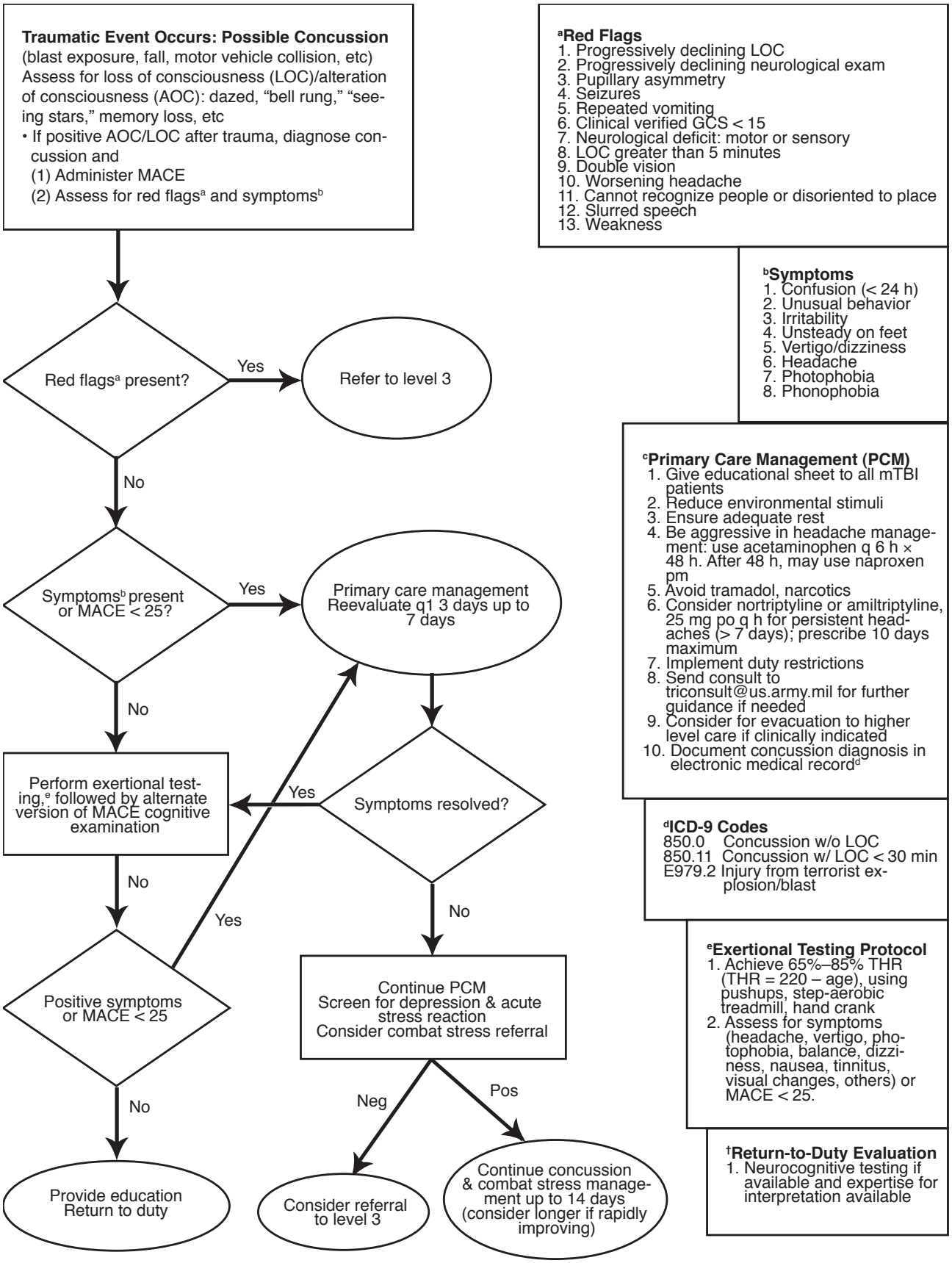
Traumatic Event Occurs: Possible Concussion
(blast exposure, fall, motor vehicle collision, etc)
Assess for loss of consciousness (LOC)/alteration of consciousness (AOC): dazed, "bell rung," "seeing stars," memory loss, etc
• If positive AOC/LOC after trauma, diagnose concussion and
(1) Administer MACE
(2) Assess for red flags[a] and symptoms[b]
Red flags[a] present?
Yes
Refer to level 3
No
Symptoms[b] present or MACE < 25?
Yes
Primary care management
Reevaluate q1 3 days up to 7 days
No
Perform exertional testing,[e] followed by alternate version of MACE cognitive examination
Yes
Symptoms resolved?
No
Yes
No
Positive symptoms or MACE < 25
No
Provide education
Return to duty
Continue PCM
Screen for depression & acute stress reaction
Consider combat stress referral
Neg
Pos
Consider referral to level 3
Continue concussion & combat stress management up to 14 days (consider longer if rapidly improving)
[a]Red Flags
1. Progressively declining LOC
2. Progressively declining neurological exam
3. Pupillary asymmetry
4. Seizures
5. Repeated vomiting
6. Clinical verified GCS < 15
7. Neurological deficit: motor or sensory
8. LOC greater than 5 minutes
9. Double vision
10. Worsening headache
11. Cannot recognize people or disoriented to place
12. Slurred speech
13. Weakness
[b]Symptoms
1. Confusion (< 24 h)
2. Unusual behavior
3. Irritability
4. Unsteady on feet
5. Vertigo/dizziness
6. Headache
7. Photophobia
8. Phonophobia
[c]Primary Care Management (PCM)
1. Give educational sheet to all mTBI patients
2. Reduce environmental stimuli
3. Ensure adequate rest
4. Be aggressive in headache management: use acetaminophen q 6 h × 48 h. After 48 h, may use naproxen pm
5. Avoid tramadol, narcotics
6. Consider nortriptyline or amiltriptyline, 25 mg po q h for persistent headaches (> 7 days); prescribe 10 days maximum
7. Implement duty restrictions
8. Send consult to triconsult@us.army.mil for further guidance if needed
9. Consider for evacuation to higher level care if clinically indicated
10. Document concussion diagnosis in electronic medical record[d]
[d]ICD-9 Codes
850.0 Concussion w/o LOC
850.11 Concussion w/ LOC < 30 min
E979.2 Injury from terrorist explosion/blast
[e]Exertional Testing Protocol
1. Achieve 65%–85% THR (THR = 220 – age), using pushups, step-aerobic treadmill, hand crank
2. Assess for symptoms (headache, vertigo, photophobia, balance, dizziness, nausea, tinnitus, visual changes, others) or MACE < 25.
[†]Return-to-Duty Evaluation
1. Neurocognitive testing if available and expertise for interpretation available

Figure 15-5 (left page). Initial management of concussion in a deployed seting. Defense and Veterans Brain Injury Center decision algorithm for battlefield mild traumatic brain injury at a Level I setting. Definitive assessment and care is given by providers to include a more detailed assessment, management recommendations, and consideration for evacuation to a higher level of care.

AOC: alteration of consciousness
GCS: Glascow Coma Scale
HS: at bedtime
ICD-9: International Statistical Classification of Diseases, 9th edition
LOC: loss of consciousness
MACE: Military Acute Concussion Evaluation
mTBI: mild traumatic brain injury
po: by mouth
q: every
THR: target heart rate
w/: with
w/o: without

Reproduced from: US Army Institute of Surgical Research. Joint Theater Trauma System Web site. Available at: http://www.usaisr.amedd.army.mil/cpgs/mTBIDplydSet0811.pdf. Accessed December 15, 2009.

SYMPTOM TREATMENT

Symptom treatment for mTBI can be discussed from four areas: (1) pharmacologic management, (2) educational interventions, (3) rest/return-to-duty decisions, and (4) targeted therapies. An extensive discussion of the pharmacologic interventions in TBI is beyond the scope of this chapter. The reader is referred to reviews of the evidence for various pharmacologic interventions.[45,73–76] In general, however, symptomatic treatment strategies can be the most effective, including regulation of sleep through pharmacologic and nonpharmacologic strategies,[77] headache management,[78] pain management,[79] and treatment of depression.[80] Treatment of all of these has been associated with improved quality of life or outcomes.

Educational Interventions

Educational and psychological therapies have also proven effective in mTBI. Mittenberg et al[81] compared two mTBI groups: a treatment group (n = 29) and a control group (n = 29). The treatment group received a printed manual and met with a therapist prior to hospital discharge to review (*a*) the nature and incidence of expected symptoms, (*b*) the cognitive-behavioral model of symptom maintenance and treatment, (*c*) techniques for reducing symptoms, and (*d*) instructions for gradual resumption of premorbid activities. The control group received routine hospital treatment and discharge instructions. After 6 months, the treated patients reported significantly shorter average symptom duration (33 days vs 51 days) and significantly fewer symptoms at follow-up. The conclusion was that brief, early psychological interventions are effective in reducing the incidence of postconcussive symptoms. Ponsford and colleagues[82] have shown similar results in a group of individuals with mTBI. Those who were seen at 1 week postinjury and given informational material reported fewer symptoms overall and were significantly less stressed at 3 months after the injury than a group that did not receive the same education. A number of educational materials effective for individuals with brain injuries and their families are available at a number of sources including the Defense and Veterans Brain Injury center Web site (www.dvbic.org/cms.php?p=Education).

Rest and Return-to-Duty Issues

Other palliative interventions such as bed rest have been shown to have some efficacy in treating postconcussive symptoms over the short term (eg, decreased dizziness), but have not proven to have long-term outcomes better than individuals who did not get such rest.[83] Return-to-duty issues in the military operational setting are addressed in the algorithm above (see Figure 15-5). Most of the decisions are based on the resolution of self-reported TBI symptoms as well as the integration of clinical data based on testing a service member to see if symptoms may return when physically stressed.

SUMMARY

In modern warfare, TBI is a common occurrence that has significant implications for the health and welfare of the troops, as well as overall fighting effectiveness. Early identification of less obvious (usually milder) TBI is important, as is a basic understanding of when individuals may be treated in situ and ways to maximize recovery. Even in peacetime, there are concerns about TBI because it occurs at high rates in the military. It is essential, therefore, that healthcare providers and the fighting force both have a basic understanding of the need for prevention of these injuries, and early identification when they do occur.

EXHIBIT 15-2

MILITARY ACUTE CONCUSSION EVALUATION FORM

Military Acute Concussion Evaluation (MACE)

Defense and Veterans Brain Injury Center

Patient Name: _____

SS#: _____-_____-_____**Unit:** _____

Date of Injury: _____/_____/_____

Time of Injury: _____

Examiner: _____

Date of Evaluation: _____/_____/_____

Time of Evaluation: _____

History: (I – VIII)

I. **Description of Incident**

Ask:

a) What happened?

b) Tell me what you remember.

c) Were you dazed, confused, "saw stars"?

❒ Yes ❒ No

d) Did you hit your head? ❒ Yes ❒ No

II. **Cause of Injury** (Circle all that apply):

1) Explosion/Blast 4) Fragment

2) Blunt object 5) Fall

3) Motor Vehicle Crash 6) Gunshot wound

7) Other _____

III. **Was a helmet worn?** ❒ Yes ❒ No

Type _____

IV. **Amnesia Before: Are there any events just BEFORE the injury that are not remembered? (Assess for continuous memory prior to injury)**

❒ Yes ❒ No If yes, how long _____

V. **Amnesia After: Are there any events just AFTER the injuries that are not remembered? (Assess time until continuous memory after the injury)**

❒ Yes ❒ No If yes, how long _____

VI. **Does the individual report loss of consciousness or "blacking out"?**

❒ Yes ❒ No If yes, how long _____

VII. **Did anyone observe a period of loss of consciousness or unresponsiveness?**

❒ Yes ❒ No If yes, how long _____

VIII. **Symptoms** (circle all that apply)

1) Headache 2) Dizziness

3) Memory Problems 4) Balance problems

5) Nausea/Vomiting 6) Difficulty Concentrating

7) Irritability 8) Visual Disturbances

9) Ringing in the ears 10) Other _____

Examination: (IX – XIII)

Evaluate each domain. Total possible score is 30.

IX. Orientation (1 point each)

Month:	0	1
Date:	0	1
Day of Week:	0	1
Year:	0	1
Time:	0	1

Orientation Total Score ____/5

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This form may be copied for clinical use.

(**Exhibit 15-2** *continues*)

Exhibit 15-2 *continued*

Military Acute Concussion Evaluation (MACE)

Defense and Veterans Brain Injury Center

X. Immediate Memory:

Read all 5 words and ask the patient to recall them in any order. Repeat two more times for a total of three trials.

(1 point for each correct, total over 3 trials)

List	Trial 1	Trial 2	Trial 3
Elbow	0 1	0 1	0 1
Apple	0 1	0 1	0 1
Carpet	0 1	0 1	0 1
Saddle	0 1	0 1	0 1
Bubble	0 1	0 1	0 1
Trial Score			

Immediate Memory Total Score _____/15

XI. Neurological Screening

As the clinical condition permits, check

Eyes: pupillary response and tracking

Verbal: speech fluency and word finding

Motor: pronator drift, gait/coordination

Record any abnormalities. **No points are given for this.**

XII. Concentration

Reverse Digits: (go to next string length if correct on first trial. Stop if incorrect on both trials.) 1 pt. for each string length.

4-9-3	6-2-9	0	1
3-8-1-4	3-2-7-9	0	1
6-2-9-7-1	1-5-2-8-5	0	1
7-1-8-4-6-2	5-3-9-1-4-8	0	1

Months in reverse order:

(1 pt. for entire sequence correct)

Dec-Nov-Oct-Sep-Aug-Jul

Jun-May-Apr-Mar-Feb-Jan 0 1

Concentration Total Score ____/5

XIII. Delayed Recall (1 pt. each)

Ask the patient to recall the 5 words from the earlier memory test (Do NOT reread the word list.)

Elbow	0	1
Apple	0	1
Carpet	0	1
Saddle	0	1
Bubble	0	1

Delayed Recall Total Score ____/5

TOTAL SCORE _____/30

Notes: _____

_____

_____

_____

_____

_____

_____

Diagnosis: (circle one or write in diagnoses)

No concussion

850.0 Concussion without

Loss of Consciousness (LOC)

850.1 Concussion with

Loss of Consciousness (LOC)

Other diagnoses _____

_____

McCrea, M., Kelly, J. & Randolph, C. (2000). *Standardized Assessment of Concussion (SAC): Manual for Administration, Scoring, and Interpretation.* (2nd ed.) Waukesa,WI: Authors.

Defense & Veterans Brain Injury Center
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