ISSN 1553-9768 Summer 2008 Volume 8, Edition 3

Journal of Special Operations Medicine

A Peer Reviewed Journal for SOF Medical Professionals

Volume 8, Edition 3 / Summer 08

Journal of Special Operations Medicine

ISSN 1553-9768

IN THIS EDITION: UPDATED TACTICAL COMBAT CASUALTY CARE GUIDELINES

THIS EDITION'S FEATURE ARTICLES:

Dedicated to the Indomitable Spirit & Sacrifices of the SOF Medic

Mild Traumatic Brain Injury: Situational Awareness for Special Operations Medical Providers

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Financial Disclosure

The authors of *Mild Traumatic Brain Injury: Situational Awareness for Special Operational Medical Providers;* Robert D. Forsten, DO, LTC, MC; Richard J. Roberts, PhD; Charles Stewart, MD, FACEP, FAAEM; Benjamin E. Solomon, MD, LTC, MC and Mark R. Baggett, PhD, LTC, MS has indicated that, within the past year, they have had no significant financial relationship with a commercial entity whose product/services are related to the topic/subject matter.

Objectives

1) Physicians will select from a range of guidelines to optimize diagnoses, treatment and referral for patients with mTBI resulting in optimum health and functioning.
2) Nurses will be cognizant of mTBI signs and symptoms and provide care in order to maximize health and functioning.

Abstract

Compared with risk for the civilian population, the likelihood of sustaining a traumatic brain injury (TBI) is substantially higher in United States Special Operations Command (USSOCOM) personnel due to a high operations tempo in training and through multiple deployments in the Global War on Terrorism (GWOT). Although penetrating trauma to the head is easier to identify, more attention needs to be paid by Special Operations Forces (SOF) medical personnel to the signs and symptoms of this "invisible" injury, some of which may occur weeks to months after suspected mild traumatic brain injury (mTBI). The term "mild" when describing TBI can be misleading because blast-exposed personnel can be severely compromised cognitively and emotionally. Labeled by some as the "signature wound" of the GWOT, mTBI continues to be a growing health concern of our individual servicemembers, in addition to commanders, medical providers, families, veterans' organizations, and communities. Since every blast-wave is different depending on type of explosive material and reflecting surface as well as individual differences and situational factors (e.g., pre-morbid intelligence; sitting in vehicle versus head exposed in gun turret), it is impossible to determine which individual, exposed to a blast, will manifest persistent problems weeks to months later following the acute blast. The mechanism of injury following a blast is often not as clear as it may first seem. A servicemember can first be hit with a blast overpressure, then thrown with force against an object, and then be hit in the head by flying debris. Servicemembers may be exposed to one or numerous traumas or blasts and not develop symptoms, or have symptoms that range from mild to severe that last for days, months, or even a lifetime. This article selectively reviews current literature about mTBI and highlights etiology, evaluation, and treatment options for SOF medical providers. What is currently known about the physics of blast-waves and the four major modes of injury following concussion-blasts will be summarized. To complicate the diagnostic picture, Soldiers can have co-morbid post-traumatic stress disorder (PTSD) either from circumstances of the blast injury or develop PTSD from a combat event unrelated to the blast injury. This article discusses these diagnostic changes.

Key Words: mild traumatic brain injury, mTBI, post-traumatic stress disorder, PTSD, blast injury, closed head injury, Operation Iraqi Freedom, OIF, Operation Enduring Freedom, OEF, improvised explosive device, IED.

Introduction

The goal of this paper is to educate SOF providers to identify, treat, case-manage, and refer SOF patients who experience symptoms of mTBI in order to maximize their physical and emotional health so that they may return to active duty. In turn, these medical providers also need to educate their SOF personnel, commanders, and family members with regard to mTBI. If a servicemember is too injured or dysfunctional to continue on active duty, SOF providers may need to provide the liaison for that individual for a medical evaluation board and follow-up care with the Veterans Administration (VA).

When servicemembers are first treated on the battlefield for severe physical trauma, seemingly less severe brain injuries may be initially overlooked. As should be their primary concern, medical providers in the battle space first focus on Trauma Combat Casualty Care. Penetrating (open) or moderate-to-severe closed injury with respiratory or hypovolemic compromise is usually recognized in theatre and treated accordingly at Roles Two and Three. The Army literature is no longer using Levels or Echelons of Care and has adopted the NATO terminology of Roles with some modifications; the documents provide the explanations. A review of the levels of medical treatment will not be discussed in this paper; however, interested readers can review this information at Joint Publication 4-02, Health Service Support, October 2006 or FM 4-02.2, Medical Evacuation, May 2007. Higher Roles of care (usually in Germany or in CONUS) continue evaluation and treatment of moderate, severe, and penetrating brain injury that has already been identified.

This article will focus on the neurobehavioral sequelae of mTBI. In this context, the word "mild" refers only to the acute forces (e.g. blunt-force trauma or blast-exposure) that caused the closed-head trauma and to the acute symptoms experienced by the combatant. Serious symptoms can persist or develop weeks to months after a seemingly "mild" instance of closed-head trauma for several reasons. First, acute problems are frequently overlooked due to the presentation on injury — either serious or life-threatening symptoms take precedence after the servicemember experiences a brief period of loss of consciousness and then returns to duty. Second, SOF personnel are highly motivated to perform their duties and to "improvise, adapt, and overcome," factors that potentially interfere with completing a mis-

sion. Therefore, they paradoxically may try to minimize or even deny symptoms to themselves (and care-providers) in order to cope with those very same symptoms. Third, the effects of new symptoms may only fully manifest when warriors return to the home-front and are confronted by multiple duties and challenges that are not necessarily present during a mission (e.g., family life, making appropriate social decisions, managing finances, etc.) In this regard, family members, close friends in the same unit, or supervisors may be invaluable collateral informants when a service-member is clearly struggling but denying symptoms. Fourth, serious symptoms and persistent dysfunction can and does occur in the context of normal neurological exams, normal EEGs, and normal MRI and CT scans following blast-exposure. Finally, if patients have sustained frontal lobe damage they may not be fully aware of their new deficits and limitations due to lack of insight into their own daily functioning.

Due to the advances in battlefield medicine (particularly medical techniques and evacuation) as well as body armor used by military personnel, mortality has significantly declined in current operations in the GWOT compared to past conflicts. Individuals who may have died in previous wars due to injury to the head, neck, and upper extremities are now surviving at much higher rates.[1] However, the same body armor that saves life can contribute to brain injury. Because each blast-wave differs (depending on type of explosive material and reflecting surface as well as individual differences in brains in terms of neuroanatomy, density of neurons in specific regions, etc.), it is impossible at this time to determine which individual will develop clinically significant problems after mTBI due to blast-exposure. A patient may be exposed to one or numerous blasts yet not develop symptoms, or have symptoms that range from mild to severe that last for days, months, or over a lifetime.

The exact proportion of troops who have mTBI is not known, although it has been reported as high as 18% in news articles citing Army medical officials. Many troops and VA patients have reportedly developed "persistent post-concussive syndrome (PCS)," characterized by common symptoms such as irritability, memory problems, headache, tinnitus, and difficulty concentrating.[2] As a result, the Department of Defense (DoD) and the Department of Veterans Affairs have implemented population screening procedures for mTBI. Despite these steps, little is known about the epidemiology of mTBI due to blast-exposure and its association with adverse health effects.[3] Furthermore, there is still considerable debate regarding the brain-related versus psychosocial factors that may contribute to persistent PCS following blunt-force trauma.[4]

Background and Significance

As of April 30, 2008, 31,848 servicemembers have been wounded in action in OIF/OEF. Thirty-two percent of the most seriously injured requiring medical evacuation from theater to Walter Reed Army Medical Center had a documented TBI. Over 90% of combat-related TBI are closed-head injuries with most servicemembers sustaining an mTBI.[5] Eighty-eight percent of military personnel treated at Role (Echelon) II medical units in Iraq had been injured by improvised explosive devices (IED)s or mortars. Many (47%) of these injuries involved the head. Similarly, 97% of the injuries to one Marine unit in Iraq were due to explosions (65% IEDs, 32% mines).[6] The Centers for Disease Control and Prevention estimates that 5.3 million Americans currently have a long-term or lifelong need for help to perform activities of daily living as a result of TBI, and 40% of those hospitalized for TBI had at least one unmet need for services one year after injury. The most frequent unmet needs were: improving memory and problem solving; managing stress and emotional upsets; controlling temper; and improving one's job skills.[7]

Considered against the backdrop of thousands of years of human evolution, brain injuries due to high speed motor vehicle crashes, explosive IED blasts, and bullet wounds are relatively new phenomena. This primitive protection provided by the combination of the skull, three membranes surrounding the brain, and the cerebrospinal fluid (CSF) has not had sufficient time to respond to evolutionary pressures to protect human beings from the technological advances of modern warfare during the last two centuries. At best, the covering of the cerebral hemispheres affords us modest protection against minor falls or being hit in the head by wooden clubs and small rocks wielded by other human beings, rather than the forces generated by extreme blast over-pressures due to roadside bombs or by high speed collisions with windshields, telephone poles, or other vehicles.[8]

In 1915, "shell shock" was initially conceptualized as due to the effects of a neurological lesion, the result of powerful compressive forces (mostly from artillery and mines). However, doubts soon arose about the contribution of direct cerebral trauma to shell shock, and some expressed the view that the symptoms were more psychological than organic in origin, even to the extent of characterizing them as "traumatic neuroses." Servicemembers who developed somewhat

similar symptoms miles from the front or were never exposed to blast injuries only confused the picture for WWI medical providers. This caused military doctors and patients to believe that shell shock was environmentally or contextually determined and the way in which healthcare and compensation were organized served to reinforce both symptoms and disability. Vigorous debate ensued between various schools of thought that led to a series of novel managerial interventions designed to limit what had become an epidemic of patients and war pension claims.[9] Over 90 years later, this vigorous debate continues academically and politically, but we are far closer in the identification, understanding, evaluation, and management of mTBI related to blast-exposure, as well as co-morbid post-traumatic stress disorder (PTSD) and differentiating between the two. Increased awareness and much additional research should aid in understanding these complex phenomena related to the GWOT and ultimately lead to improved treatment for servicemembers.

In Fiscal Year 2007, the Congressionally Directed Medical Research Programs' Psychological Health and Traumatic Brain Injury (PH/TBI) allotted $150 million and $151 million for future research for mTBI and PTSD respectively. Key priorities of the PH/TBI Research Program are to complement ongoing DoD efforts to ensure the health and readiness of our military forces and to support the Department of Defense Psychological Health and Traumatic Brain Injury Center of Excellence in its efforts to advance and spread PH/TBI knowledge, enhance clinical and management approaches, and facilitate other vital services to best serve the needs of military families impacted by PH problems and or TBI.[10]

Etiology

For the purpose of this paper, we will focus on TBI caused by explosive force caused by IEDs, rockets, land mines, breaching operations, etc. However, within USSOCOM, personnel also experience head trauma by other means: hand to hand combative training, airborne and air assault operations, demolitions training, .50 caliber weapons firing, shallow water blackout with subsequent asphyxiation, falls, and motor vehicle accidents on and off duty (list not all inclusive). Blast injuries are divided into four types, and in many cases personnel sustaining a mild, moderate, severe, or penetrating TBI suffer three to four types of blast injuries (in some cases all four). Primary blast injuries are caused by the direct effect of the blast wave or primary injuries which will be discussed in more detail below; secondary injuries are caused by other objects that are accelerated by the explosive waves, i.e., penetrating trauma from the explosive device (shrapnel) and subsequent surrounding structure (wood, glass, rocks, concrete, etc.); tertiary injuries are caused by movement of the victim being thrown or structural collapse (fractures and penetrating injury as well as open head fractures and closed head trauma from coup-contrecoup injuries); quaternary injury results from burns, asphyxia from being buried in debris, or exposure to toxic inhalants. One implication of the concepts of secondary and tertiary blast-exposure injuries is that there is often some ballistic or blunt-force component to the head following blast-concussion due to IEDs.

Shock wave blasts from IEDs, rocket-propelled grenades, and land mines are the leading cause of mTBI for active duty military personnel in combat zones. In prior military conflicts, TBI was present in roughly 14 to 20% of surviving casualties. Reports indicate 12,274 servicemembers have sustained a TBI in OIF and OEF as of March 24, 2007 but that number could grow much higher.[11] Currently, we lack the data to establish a dose-response curve for individuals with multiple blast-exposures and to predict the potential effects of such variables as inter-blast interval or the interactive effects of both blunt-force trauma and blast-exposure for a single given individual.

Primary blast injuries can be characterized as barotrauma caused from either significantly high over-pressurization or under-pressurization relative to atmospheric pressure following an explosion. Body organs are damaged by pressure changes at air-fluid interchanges due to the high-frequency stress wave and low-frequency shear wave. Over-pressure or under-pressure waves predominate depending on the characteristics and location of the blast. This blast wave causes a wide range of physical problems on the human body to include: ruptured tympanic membranes, pulmonary damage and air embolization, rupture of hollow viscera, ruptured orbital globe, and brain injuries caused by concussion (coup-contrecoup injuries due to blast wave), as well as barotraumas caused by acute gas embolism.[13,14]

An explosion is an event that occurs when a substance rapidly releases energy and produces a large volume of gaseous products. High explosive, thermobaric, and nuclear detonations all provide this change in potential energy to kinetic injury in a very short period of time. The extreme compression of molecules by this change in energy creates bands of locally high pressure, the blast wave which moves outwards from the epicenter of the blast. These blast waves travel faster than the speed of sound. Blast products – gas, particles, and debris of the container and items in proximity to the ex-

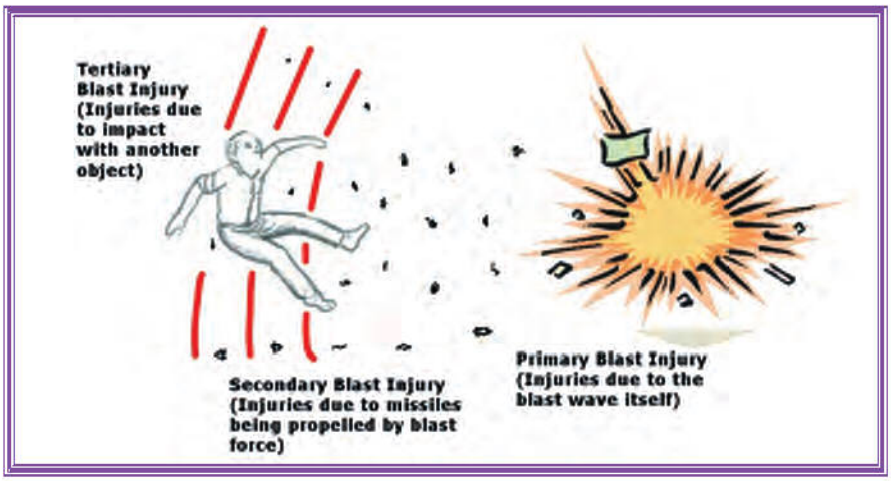

Figure 1. Illustration of primary, secondary, and tertiary injury from blast; quaternary not pictured (illustration by Charles Stewart M.D.)

plosive (including human remains) also spread outwards, but travel much more slowly. When a high explosive detonates, it is converted almost instantaneously into a gas at very high pressure and temperature. For example the major ingredient in Composition C4 (Cyclotrimethylenetrinitramine or RDX [Royal Demolition Explosive]) can generate an initial pressure of over four million pounds per square inch (4x10E6 PSI). The temperature can be as high as 3,000 degrees Celsius — more than twice that generated by a conventional explosive. The blast wave can travel at approximately 10,000 feet per second. Body armor provides a false sense of security during an explosive detonation. The body armor does protect the victim from shrapnel and to a lesser extent, objects picked up and flung by the blast wave, but it also provides a reflecting surface that can concentrate the power of the explosion as the blast wave reflects off of the armor front and back. Since the bulk of injuries from an explosive device are from secondary objects flung by the blast wave, the advantages of body armor outweigh the risk of enhancement of the blast wave. The medical provider should not assume that body armor will protect the victim from an explosion-related injury.[14]

During explosions, high pressure gases rapidly expand from the original volume and generate a marked pressure wave – the "blast wave" that moves outward in all directions. The result is a sudden shattering blow on the immediate surroundings. Furthermore, a blast-wave that would cause only modest injury in the open can be lethal if the victim is in a confined area (e.g., a basement) or near a reflecting surface such as a solid wall or a building (or body armor). If the pressure wave is near a solid barrier (e.g., a narrow alleyway), the pressure exerted at the reflecting surface may be many times that of the incident blast-wave.[15] In mTBI, neurological deficits seen days to weeks after blast exposure may be caused by "microshearing" of axons and dendrites and/or their synapses generalized throughout the brain due to this blast wave (further research in this area is warranted). However, axonal stretching of neurons that survive may also play a part in the development of brain dysfunction.[16]

There is simply no question that body armor provides substantial protection against projectile injury. What has not yet been quantified is the contribution of the armor to primary blast injury. The potential reflection of blast waves by armor may be particularly important in the genesis of traumatic brain injury, since the helmet can act as a focusing reflector of waves, with the brain at the center point of the focus.

Co-morbidity

PTSD, mood, and substance use disorders frequently co-occur with mTBI or persistent PCS head trauma and can significantly complicate diagnosis, treatment, and the recovery process. Post-TBI symptoms such as poor sleep, poor memory/concentration, and irritability are common in PTSD, mTBI, and major depressive disorder (which may be triggered by either PTSD or mTBI).

PTSD has been found to be strongly associated with mTBI, as well as blast-exposure and participation in combat. It was also demonstrated that mTBI was associated more with loss of consciousness versus altered consciousness or being "dazed" or having one's "bell rung." In a nonrandom sample of two Army brigades likely to be representative of servicemembers serving in ground-combat units in Iraq, 15% of servicemembers reported an injury during deployment that involved loss of consciousness or altered mental status and thus, by definition, incurred at least one mTBI. This 15% was significantly more likely to report high combat exposure and a blast mechanism of injury than others servicemembers in the study who reported other types of injuries. Forty-four percent who reported loss of consciousness met criteria for PTSD, compared with 27.3% of those with altered mental status, 16.2% of those with other injuries, and 9.1% of those with no injuries. In this study, the association between mTBI and

PTSD remained significant after combat experiences had been controlled for.[3] However, given this data, providers should also be hesitant to lump all symptoms under only a TBI diagnosis. Feeling disoriented or not remembering injuries or blasts can also occur in the context of PTSD due to the psychological process of severe dissociation. It is important to note that military personnel with mTBI have a greater risk for many types of health-related problems but should not necessarily be led to believe that they have sustained a brain-injury that will result in permanent behavioral or cognitive changes. "Normalization" of symptoms, recognizing post-traumatic stress reactions, supportive care, and reassurance from care-providers may minimize the unnecessary attribution of normal stress reactions to minimal or nonexistent neuropathology and facilitate resilience after mTBI.[4]

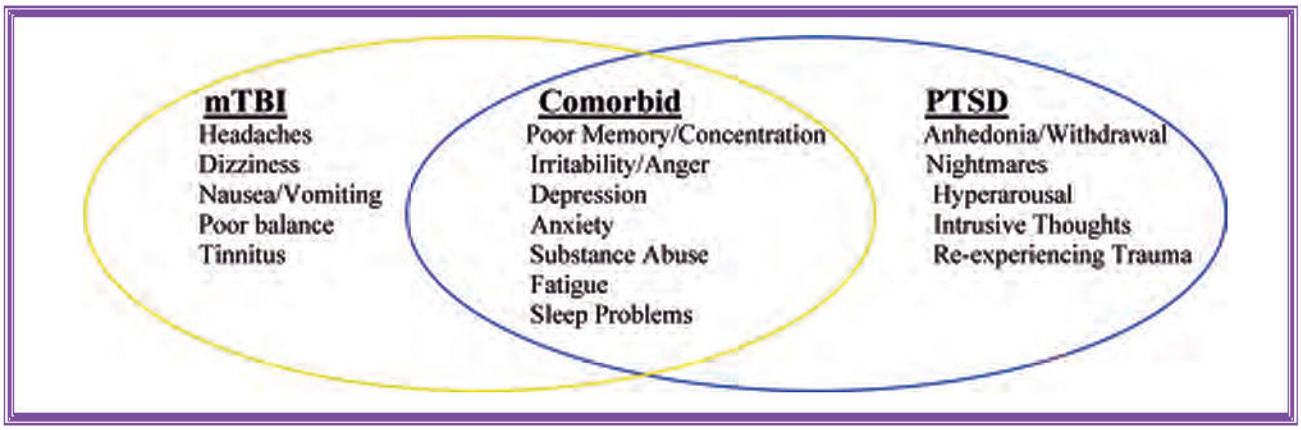

Figure 2. Symptoms common to both mTBI and PTSD

Preventive Measures

Protective factors that prevent and reduce PTSD or in some cases symptoms following a mTBI are routinely seen in SOF personnel. These include psychological assessment in many personnel, above average intelligence, increased training, high unit morale, esprit de corps and camaraderie, maturity, and studies from SERE school that show SOF personnel are more stress hardy (biologically) in terms of dealing with acute and long term stress.[17,18,19] The possibility exists that although deployments in this population are more frequent than conventional forces, deployments are in shorter duration and may be protective in terms of individual psychological and physical "resetting." Another interesting phenomenon reported in the literature is that heat acclimation (chronic exposure to moderate heat) can also provide resistance to TBI.[20] Stress inoculation in terms of training and conditioning also plays an important role in the overall outcome of the development of mTBI and PTSD. However, it is important to recognize that markedly elevated levels of cortisol and other glucocorticoids are associated with intense combat-related stress. Some evidence exists to suggest that high levels of stress may damage sensitive brain tissues (e.g., hippocampus) or disrupt the healing processes in brain tissue following mTBI.

While it is likely still several years away, the military has ongoing research to place sensors in every ballistic helmet to measure the effects of blasts and alert medical providers to potential blast injury. In a recent news article, the deputy coordinator for the DoD's Blast Injury Research Program Coordinating Office commented that this technology's first hurtle will be to prove that a sensor reading can be matched to a specific event.[21] Currently, there are systems being field tested by at least two contracting agencies, one of which is already in the theatre of operations in several thousand servicemembers.

Evaluation

The military has instituted the Military Acute Concussion Evaluation (MACE) for use on the battlefield. Instructions for its use are available at http://www.dvbic.org/cms.phpp=Medical_care. This is a post-injury assessment measure and is designed for use fairly immediately following a TBI. There are no data to support its use beyond the acute injury period, although it may have sensitivity to persistent cognitive deficits after the first week. Key points to remember about the MACE are that the cutoff score for possible mTBI is 25 (although scores of 26 or 27 call for follow-up), and the MACE should be administered within 24

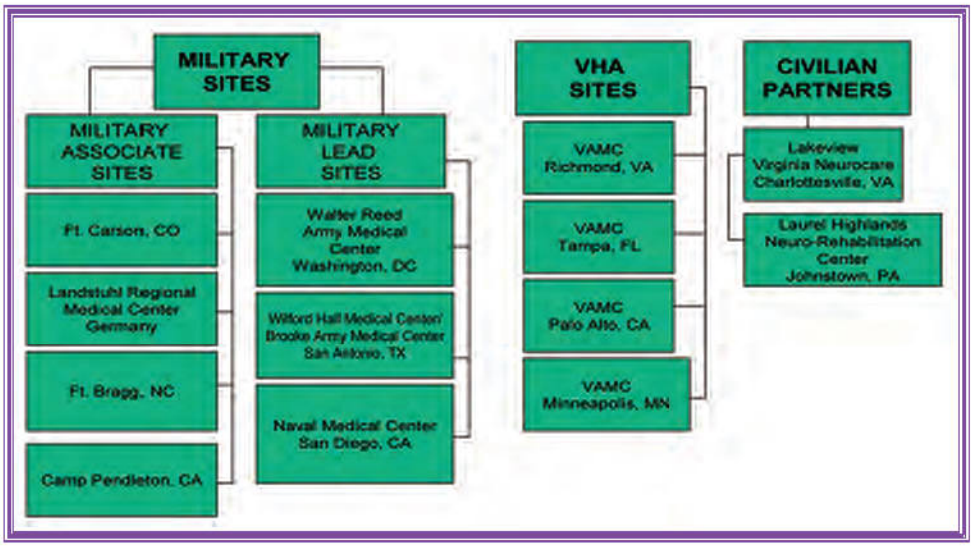

Figure 3. Organization of the Defense and Veterans Brain Injury Center locations.[24] The DVBIC is composed of seven military facilities, four VAMCs, and two civilian community reentry programs. Initiatives can be found on the center's website at http://www.dvbic.org.

hours of blast-exposure or blunt-force head trauma. Serial exams may be of use to coordinate return to duty or further evaluation and treatment. Although the majority of patients with mTBI recover quickly with minimal intervention, there is a subset that develops lingering symptoms that interfere with social and occupational functioning. Since patients with mTBI may not come to clinical attention for a variety of reasons, the purpose of assessment may vary slightly based on the timing of presentation following injury (Defense and Veterans Brain Injury Center: Updated mTBI Clinical Guidance).[22]

Most patients seeking medical care in a primary care setting are seen using the primary symptom-based approach where the medical provider treats the patient according to the most prominent symptoms. The patient informs the treating provider of an ailment causing some level of distress, discomfort, or dysfunction and the provider decides on a treatment to resolve the ailment. Tests are ordered and possibly a referral to a specialist. However, if a new symptom arises or becomes problematic, this process is repeated and can be time consuming leading to frustration for the patient and provider as well as missed diagnoses. For most medical patients, the primary system-based approach to patient care works; however, it has been shown to delay care when treating TBI. For TBI and blast-related polytrauma, focusing on the mechanism of injury approach, rather than solely on primary symptoms, can create a more comprehensive and integrated program of care. Patients have a case-manager assigned for screening, are evaluated by a psychiatrist and/or neurologist, and are referred to multiple specialists for evaluation such as a psychiatrist, neuropsychologist, speech pathologist, audiologist, rehabilitation therapist and/or social worker based on individual needs.[23] This in-depth screening leads to a comprehensive treatment plan with higher rates of resolution for TBI. This model is currently used to treat blast related trauma including TBI at the Veterans Health Administration's Polytrauma Rehab Centers, DVBICs and Walter Reed's Psychiatry Continuity Service for the treatment of PTSD.

Deployed care providers can find clinical practice guidelines for the assessment and treatment of acute mTBI at http://www.pdhealth.mil/downloads/clinical_practice_guideline_recommendations.pdf for the three roles (levels) of care in theatre. The algorithms should not be interpreted as a substitute for sound clinical judgment. Operational and tactical considerations may in some instances override the CPG.[25]

Common symptoms following blast-exposure or blunt-force trauma include:

- difficulty organizing daily tasks
- blurred vision or eyes tiring easily
- headaches
- ringing in the ears
- feeling sad, anxious or listless
- easily irritated or angered
- feeling tired all the time
- feeling light-headed or dizzy
- trouble with memory, attention or concentration
- more sensitive to sounds, lights, or distractions
- impaired decision-making or problem-solving
- difficulty inhibiting behavior or behaving impulsively
- slowed thinking, moving, speaking, or reading
- easily confused, feeling easily overwhelmed
- change in sexual interest or behavior (Force Health Protection and Readiness Policy and Programs: Combat Trauma Quick Facts).[26]

Table 1. Traumatic Brain Injury[27]

Mild TBI	Moderate TBI	Severe TBI
Normal Structural Imaging	Normal or abnormal structural imaging	Normal or abnormal structural imaging
LOC = 0-30 minutes*	LOC > 30 min and < 24 hours	LOC > 24 hours
AOC = a moment up to 24 hours	AOC > 24 hours. Severity based on other criteria	
PTA = 0-1 day	PTA > 1 and < 7 days	PTA > 7 days

LOC -Loss of consciousness
AOC- Alteration in consciousness
PTA- Post-traumatic amnesia
*An inconsistency currently exists between published guidance and the published V codes for mild TBI when loss of consciousness is between 30 and 59 minutes. Until this consistency is resolved, Service and military medical personnel are to report in the attached format using criteria published above.[19]

Other symptoms commonly seen are alteration in sense of smell and taste, involuntary muscle tightness or stiffness, weakness in one side of the body, seizures, problems with perception and direction, increased need for simple/concrete directions, untriggered mood swings, and loss of one's social network resulting in isolation (Defense and Veterans Brain Injury Center: Signs and Symptoms of Traumatic Brain Injury located at www.dvbic.org).[28]

A recent study of 682 blast victims in Iraq revealed that the ear was the organ most vulnerable to blast over-pressure. The overall incidence of tympanic perforation was 35% in this population, and this type of injury was a significantly associated with loss of consciousness (and by definition at least mTBI). SOF medical providers treating perforated tympanic-membranes need to have a high index of suspicion for comorbid mTBI and should routinely screen for neurological symptoms with the MACE.[29] However, petechiae in the oropharynx may be more sensitive in the diagnosis of blast injury and subsequent mTBI and therefore, medical providers should routinely include this in their examination for suspected head trauma.[30]

Formal neuropsychological testing is ideally conducted six to twelve months post-injury when most of the cognitive improvement following an mTBI has already occurred. Neuropsychological testing is generally conducted prior to the initiation of formal cognitive rehabilitation in order to guide the treatment plan. However, testing may be done anytime in assessment and management of mTBI. Neuropsychological testing may also be useful following the completion of a rehabilitation plan to evaluate outcomes. Treatment need not be delayed while awaiting neuropsychological testing.[22]

Functional neuroimaging [(functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and single photon emission computed tomography (SPECT)] has been shown to be more useful and provide greater specificity in the evaluation of mTBI than structural neuroimaging with MRI or computed tomography (CT), as demonstrated by associations between brain activation and clinical outcomes.[31] Unfortunately, many treating locations do not have access to facilities that provide these studies. The most common types of non-penetrating traumatic brain injury are diffuse axonal injury, contusion, and subdural hemorrhage. When considering radiological work-up, MRI is more sensitive than CT in detecting diffuse axonal injury, however most cases of mTBI appear normal on CT and MRI. T2 weighted magnetic resonance (MR) images, especially fluid attenuated inversion recovery (FLAIR) images, are best for visualizing non-hemorrhagic lesions. Previous studies have shown that common locations of injury seen on scans would explain the symptoms of mTBI (memory, mood, attention, concentration, etc.). The most common locations for diffuse axonal injury are the corticomedullary (gray matter-white matter) junction (particularly frontotemporal), internal capsule, deep gray matter, upper brainstem, and corpus callosum. The most common locations for contusions 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. The most common locations for subdural hemorrhage are the frontal and parietal convexities.[6] Recently, it has been suggested that diffusion tensor imaging (DTI) with MRI may ultimately prove valuable for demonstrating neuronal abnormalities in the acute phase of mTBI.[32]

fMRI relies on magnetic properties of hemoglobin to create images of blood flow to the brain. Unlike CT and MRI, fMRI detects abnormal brain function, not structure. Patients are scanned while performing a cognitive task such as remembering a number. Increased regional neuronal activity results in increased blood flow and a change in the ratio of oxyhemoglobin to deoxyhemoglobin. This altered ratio appears as a bright area of activation, which often is

superimposed over a surface-rendered projection of the brain. Although there is no radiation exposure, the patient must be highly cooperative for the exam; however, this method shows the best correlation to clinical outcome compared to other radiological modalities.[33]

Lastly, secondary to increased impulsivity commonly seen after head trauma especially to the frontal lobe, it is recommended that screening for increased alcohol and other substance use disordered be performed. A good screening tool for alcohol is the alcohol use disorders identification tool or AUDIT.[34]

Management

As mission dictates, servicemembers who fall below cut off scores on the MACE or experience persistent problems with cognitive or sensory function (e.g., confusion, memory lapses, hearing loss, or blurred vision that does not resolve) should be pulled from the fight or placed on a one-week profile for limited duty at a minimum. Personnel should be screened at a minimum for neuropsychological symptoms after two to three months to ensure adequate documentation of symptoms, as the majority of cases or persistent PCS resolve after three months. Concussion research from sports medicine, primarily from boxing and football, suggested that returning an athlete to the field or boxing too early may put them at greater risk of having another concussion. Unfortunately, due to the nature of blast injury there is no clear timeline on return to duty for military personnel. The sequelae of each injury and prognosis will be based on the factors we have discussed above. Instead, return to duty from a military standpoint must be decided by an informed medical care provider based on that individual patient's mTBI, symptoms, and recovery. To promote faster recovery and manage symptoms, personnel should be directed to:

- Get plenty of rest & sleep.
- Increase activity slowly.
- Carry a notebook (write things down if trouble remembering) or use a PDA.
- Establish a regular daily routine to structure activities.
- Do only one thing at a time if easily distracted; turn off the TV or radio while at work.
- Check with someone trusted when making major life decisions.
- Avoid activities that could lead to another brain injury (contact sports, motorcycles, skiing, etc.).
- Avoid alcohol as it may slow healing of the injury.
- Avoid pseudo-ephedrine-containing projects as they may also increase symptoms (check labels on cough, cold, allergy, and diet medications).
- Avoid excessive use of over-the-counter sleeping aids (they can slow thinking and memory).[26]

Using medications to manage post-TBI syndromes is difficult and controversial due to lack of evidence in most studies, as well as the possibility of unintended side-effects worsening symptoms or creating new ones (e.g. decreased libido due to SSRI antidepressants). The general rule is to start slow at low dose and monitor closely side-effects and benefit of target symptoms. Unfortunately, no standard regimen exists, but there are numerous case studies and anecdotal evidence for medications to alleviate symptoms related to mood, sleep, and memory. No strong evidence exists that drugs are effective for mTBI-related neurobehavioral disorders, although weak evidence shows such drug classes, such as psychostimulants (dextroamphetamine, methylphenidate), can reduce symptoms of apathy, inattention, and slowness as well as improve impulsivity, memory, and concentration. Smaller studies of anticonvulsants used in treating post-TBI showed that valproic acid might improve behavioral control and decrease aggression, and it did not worsen performance on neuropsychological testing; carbamazepine reduced agitation in seven TBI patients and reduced anger in eight of ten others; gabapentin caused paradoxical effects in two TBI patients; and lamotrigine improved agitation in one TBI patient.[35] Both valproic acid and carbamazepine require periodic blood tests, and thus their use prevents an individual servicemember being deployable to an active combat zone. As stated, there is little data on medication efficacy and side-effects with co-morbid TBI. Medications discussed below are used/avoided as discussed based on known efficacy/side-effect profile both from on-label and from studied off-label use.

Headache is a remarkably common symptom of TBI. In part this is true because of the nature of TBI – an injury to the head which houses the brain is likely to cause headache. Indeed the headache of concussion is one of the most common initial symptoms. In the TBI clinic at Ft Bragg, most servicemembers with TBI suffered TBI weeks, months, or even years prior to presentation. Most report headaches that meet criteria for chronic daily headache beginning within weeks of the event that produced the TBI. Careful history and exam elicits multiple symptoms and signs of muscle involve-

ment in head and neck. Often these signs and symptoms have been present and worsening for months.

Initially, treatment should be conservative, consisting of a triptan, an anti-depressant with at least class C evidence for efficacy in headaches and a sleep-aid such as Ambien. Later, more aggressive therapy can be initiated such as pulsed steroids (dexamethasone 8mg BID x four to five days every other week), naproxen 500mg TID, baclofen 20mg TID, a proton pump inhibitor such as omeperazole, and ice packs to neck and occiput muscles BID to QID. Patients are taught specialized stretches and referred for physical therapy, chiropractic, and massage therapy as well. Three types of anti-inflammatory medications are used as are conservative therapies such as stretching and massage along with and specialized therapies such as chiropractic and physical therapy. The military would refer to this hit-hard-and-all-at-once technique as the "combined arms approach."

With chronic daily headache comes the opportunity for analgesic overuse syndrome (rebound headache). This is seen frequently in this patient population. Treatment is by standard guidelines: two months of analgesic "washout" during which no analgesics except celecoxib and/or steroids are used; celecoxib and steroids are not usually associated with overuse syndrome. A rescue medication such as a triptan may be used sparingly.

A prophylactic medication is often indicated for the resolution of chronic daily headache. For many servicemembers with TBI and even mild cognitive impairment, medications with sedating effects such as amitriptyline have an effect out of proportion to dose, causing increased cognitive slowing and impairment enough to significantly impact ADLs as reported separately by patients and spouses. In those without cognitive impairment, all those with proven efficacy (such as amitriptyline, topirimate) are effective in TBI without undue side-effects.

Low-dose, slowly titrated topirimate, despite its cognitive side-effects, has proven quite effective in the Ft Bragg TBI clinic for headache resolution when titrated over four weeks from 25mg QHS to 50mg BID. Nortriptyline has also been effective, titrated over four to five weeks from 25mg QHS to 100-125mg QHS.

An abortive headache treatment is often indicated, even in overuse syndrome. A variety has been studied. Midrin and zolmitriptan are generally effective for migraine headache although their use is recommended sparingly. In the Ft Bragg TBI clinic, frovatriptan has shown good efficacy as an abortive therapy even in non-migrainous headaches. Steroid and ketorolac injections can also be used as indicated.

Insomnia is a persistent and difficult-to-treat symptom of TBI. Zolpidem has been used in the Ft Bragg TBI clinic with some success but typically the doses needed are on the high side – zolpidem 10mg or zolpidem CR 12.5mg QHS. The medication is also sedating and can produce amnestic side-effects. There is little data suggesting that amnestic side-effects are contra-indicated in TBI or in TBI w/cognitive impairment but it seems counter-intuitive to prescribe an amnestic to an amnestic patient. Many using zolpidem report daytime somnolence and a "hangover" feeling throughout the early part of the day at these doses; most report a lack of effectiveness at lower doses. In the Ft Bragg TBI clinic, eszopiclone has been tried at different doses with best success with the 1mg formulation: 1 to 2mg QHS; patient may take the remainder of the nightly maximum 3mg for overnight awakenings. Example: Patient takes 2mg to fall asleep at 11 pm. He awakens at three am with difficulty going back to sleep. He takes the remaining 1mg, returns to sleep, wakes three or so hours later to the alarm without the "hangover" sensation.

Attention and concentration are often troublesome issues after TBI. The Ft Bragg TBI clinic has used stimulants approved for attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD) for a limited number of TBI patients with mixed success. Methylphenidate has been titrated from 5mg QAM to 10mg BID; atamoxetine 10mg/day titrated up to 40mg/day; and buproprion in the XL formulation 150mg titrated as high as 450mg/day.

Although there is little data that supports the use of selective serotonin reuptake inhibitors (SSRI) or serotonin-norepinephrine reuptake inhibitors (SNRI) post-TBI, they are commonly used to treat co-morbid mood, PTSD, and sleep disturbance as well as adjunctive therapy for post-concussive headache in TBI patients. Commonly used SSRIs prescribed off-label in this population include fluoxetine, sertraline, paroxetine, citalopram, and escitalopram. Commonly prescribed SNRIs off-label include venlafaxine and duloxetine. Second generation antipsychotics (olanzapine, risperidone, quetiapine, ziprasidone) are also frequently used off-label for mood stabilization, anger outbursts, irritability, sleep disturbance, and agitation. For the individual patient who is diagnosed with both PTSD and mTBI, the prescribing healthcare provider needs to make a decision whether to target the symptoms of PTSD first (e.g., nightmares, hyper-arousal) or address the symptoms of mTBI first and then treat any residual PTSD. We are not aware of any current guidelines to assist clinicians in

Table 2. Concussion Management Grid for treatment of common symptoms.[22]

Symptom Cluster	Presenting Symptoms or Complaints Assess frequency, severity, aggravating factors	Special Assessment related to complaint	Assessment Red Flags And Immediate Referral	Treatment Options by Symptom Cluster **NOTE:** Treat headache, sleep & irritability first as other symptoms often improve with pain control & rest **Inclusion does not imply FDA approved use. See full prescribing information.**
Headache	Headache Sensitivity to light/sound Tinnitus Nausea	**Examine:** Neurologic exam Musculoskeletal exam including cervical spine **Refer:** Any abnormality- 24 hours referral to Neurology	**Neurology referral** Worsening headache Seizures Blackout **Emergency Department (ED)** Fever Stiff neck	**Episodic:** (pm at HA onset, up to 3 days/week): Ibuprofen 600-800 mg.; Naproxen; Triptans, compazine, Phenergan **Chronic Daily Headache (Preventive):** onset ~4 weeks Propranolol 10- 240mg (BP & PTSD effects) Amitriptyline or Nortriptyline 10-100mg q HS (sleep) AED's gabapentin 300-900 mg q HS to BID sodium valproate 500-1500 mg (draw levels) topiramate 25-100mg q day to BID
Vision	Blurry vision Double vision (diplopia) Difficulty reading or focusing	**Examine:** Fundoscopic exam, visual acuity, visual fields Consider fluoresceine exam of cornea if foreign body suspected	**Neurology referral** Papilledema Cranial nerve deficit **Optometry or ophthalmology referral** Evidence of foreign body (FB)	Optometry evaluation- request binocular testing
Balance & Hearing	Dizziness Vertigo Balance difficulties Coordination problems Ringing in the ears	**Examine:** Dix-Hallpike Maneuver, Romberg, Cerebellar function (finger to nose, rapid alternating movement), nystagmus ENT/Audiology- otoscopic exam, bedside hearing test, audiogram if avail. **Administer:** consider Dizziness Handicap Inventory (DHI) normal ≤ 11	**Neurology referral** Lateral abnormality, nystagmus, abnormal Romberg **ED or emergent Neurosurgery referral** CSF leak **ENT referral** Hemotympanum, FB, TM perforation	ENT/Audiology/Vestibular PT referral depending on local resources if Positive Dix-Hallpike- or DHI > 11 or persistent dizziness complaints
Sleep	Fatigue/Loss of energy Difficulty falling asleep Difficulty staying asleep Easily tired Nightmares/sleep walking	**Administer:** Epworth Sleepiness Scale, consider PSQI **Examine:** neck size, airway, height, weight **Evaluate:** sleep routine, medication/supplement use, alcohol & substance abuse, sleep activity, nightmares, frightened arousal Pittsburgh Sleep Quality Index (PSQI) – not included in the package, but available at http://www.sleep.pitt.edu/	**Sleep Study referral** Apnea ESS>12 (Epworth Sleepiness Scale) BMI >30 (Body Mass Index)	Zolpidem 5-10 mg qHS max duration 10 days Trazodone 25-50 mg qHS max dose 150 mg (sleep maintenance) Amitriptyline 25 mg qHS max dose 100mg (headache benefit) Quetiapine 25 mg qHS Max dose 100mg (PTSD, nightmare benefit)
Irritability	Anger Depression Mood swings Anxiety Tension Easily overwhelmed	**Administer:** PCL-M Screening Questionnaire, consider PHQ-9 or other depression inventory **Evaluate:** specific history & symptoms: physical fighting, alcohol intake, relationship problems, suicidal, homicidal	**Behavioral Health referral** Outward violence Excessive alcohol intake Suicidal ideation Homicidal ideation	Sertaline25-50 mg qD Titrate q7-10d max dose 150mg/d Citalopram 10 mg/day titrate to max dose 40 mg/day Allow 3-4 week therapeutic trial of each drug **Refer:** treatment failure of two meds
Cognition	Memory loss or lapse Forgetfulness Poor concentration Decreased attention Slowed thinking Executive dysfunction	**Administer:** MACE if injury within 24 hours. Other neurocognitive testing as available (eg ANAM or other neuropsychological testing) **Gather:** Collateral information from family, command and others		Normalize sleep & nutrition Pain control **Refer:** Speech/language pathology Occupational therapy Neuropsychology

making this determination. However, the above being said, many of these medications can cause side-effects that may need to be avoided if they have too sedating effects and/or have depressant effects. This includes tricyclic anti-depressants such as trazodone; many SSRIs such as mirtazapine, venlafaxine, duloxetine, quetiapine; almost all AEDs including gabapentin and pregabalin; beta-blockers, calcium-channel-blockers, and most muscle relaxers, such as cyclobenzaprine, and methocarbamol. Another way to look at this is to say the primary care/neurologist's headache and anti-depressant formulary has been mostly swept away.

In many cases of mTBI, patients are also taking medication for physical pain symptoms related to injury, mood, PTSD, headache, and sleep. It is not uncommon for these patients to be prescribed seven or eight different medications, which can increase the risk of drug-drug interaction as well as dependence. Providers should make it a rule to closely monitor target symptoms and whether certain medications are needed long-term and gradually wean medications over time since the majority of patients with mTBI will improve over time. This will also help in reducing further stigma that the service member has a serious medical condition as well as assist in a faster return to duty.

Summary and Conclusions

Due to the increased survivability compared to other conflicts and types of blast exposure as well as training, SOF military personnel are at markedly increased risk for mTBI. Therefore, it is of paramount importance for SOF medical providers to understand the process of evaluating and treating this "silent" disorder in order to maximize the functioning of our SOF warriors. Although much remains to be learned, we offer the following, preliminary, clinical guidelines for consideration in summary:

1. Patients in the field who produce failing scores on the MACE screening instrument, who experience injuries to the tympanic membranes following blast-exposure, who sustain serious head and neck injuries, and who manifest or complain of persistent cognitive and sensory deficits following explosions are likely to be at extremely high risk for suffering TBI.
2. Many experts believe that the majority of civilian blunt-force mTBI patients experience full recovery by

three to four months post-accident. Therefore, unless the occurrence of a brain injury has been established beyond doubt following blast exposure, providing supportive care and reassurance should facilitate the expectancy of full recovery in Special Forces combatants who have been exposed to blast-waves at close range.

3. When applied to mTBI, the word "mild" refers only to the acute change in functioning following blast-exposure or blunt-force trauma; it does *not* apply to the patient's long-term clinical outcome. Therefore, it is possible to experience one or more instances of mTBI and sustain serious cognitive, affective, and behavioral symptoms that persist and require medical treatment. Serious residual symptoms of mTBI following blast-exposure may exist even when the neurological exam, structural neuroimaging, and standard EEG are all normal.

4. While it is generally agreed upon that 10 to 15% of civilian patients with blunt-force mTBI will continue to experience moderate to severe symptoms that do not fully remit with the passage of time, we currently lack such data for military personnel with mTBI due to blast-exposure.

5. Because no two blast-exposures are exactly alike, we cannot predict which blast-exposed patients will fully recover and which ones will go on to develop problems which do not fully resolve, such as persistent symptoms of Post-Concussive Syndrome.

6. For a variety of reasons, SOF personnel may be reluctant to describe the full extent of cognitive and behavioral changes following blast-exposure. When this occurs, collateral informants such as family members, fellow unit members, or supervisors may provide useful information regarding possible changes in neurobehavioral functioning.

7. For blunt-force mTBI, functional neuroimaging (PET, SPECT, fMRI) is more likely to provide evidence of cerebral dysfunction than is structural neuroimaging (CT, MRI). In the future, this could well prove to be the case for mTBI patients with blast-exposure. Special MRI studies using new DTI technology also appear to have similar potential.

8. Some symptoms of mTBI due to blast-exposure overlap those of PTSD and secondary depression, which frequently occur co-morbidly with mTBI.

9. Double-blind, placebo-controlled studies supporting the efficacy of specific medications for treating symptoms associated with mTBI are almost completely lacking. Nevertheless, guidelines and rationales do exist for the use of various classes of psychotropic medications to treat persistent symptoms of mTBI.[36,37]

10. Given our limited knowledge base, it generally makes more sense to think in terms of using medications to target specific symptoms (e.g., stimulants for reduced concentration and persistent fatigue; mood-stabilizers with anticonvulsant properties for irritable or unprovoked aggression, etc.) than to think in terms of treating a well-defined clinical "syndrome" with a single medication.

11. When using medications to treat symptoms of mTBI, it is best to "go low and slow" in terms of titrating dosages because: (a) many SOF patients self-monitor for possible side-effects; and (b) there is often an anti-medication bias among military personnel (i.e., "real men don't take pills") that needs to be overcome if pharmacological treatment is to be accepted.

12. When mTBI and PTSD co-occur in the same patient it seems prudent to target the symptoms of one condition at a time for treatment with medication. There are currently no accepted guidelines as to whether to treat mTBI or PTSD first.

13. Behavioral treatments and cognitive rehabilitation can be useful adjuncts to medical treatment once the mTBI patient reaches the home-front. Family education is also critical, so that significant others can better understand the SOF patient's problems and needs.

14. In the unfortunate event that a patient must be separated from military service, the servicemember should be encouraged to "grieve" for the loss of his military career and encouraged to take full advantage of VA services and benefits as soon as possible when discharged. SOF medical providers need to be aware of the USSOCOM Care Coalition (http://www.socom.mil/care_coalition /) and refer SOF patients into this program according to long term needs and definitely if they are going through medical evaluation board proceedings.

15. Over the next few years, there is likely to be a flood of new research on the effects and treatment of blast-exposure. SOF and military care-providers will need to update themselves on at least a yearly basis with regard to new developments in the clinical assessment and treatment of mTBI due to blast-exposure.

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