CHAPTER 1

Understanding the Interface of Traumatic Stress and Mild Traumatic Brain Injury

Background and Conceptual Framework

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The recent wars in Afghanistan and Iraq highlight the need for paying increased attention to stress-related psychological disorders, traumatic brain injury (TBI), and the intersection between these conditions. Stress-related disorders, such as posttraumatic stress disorder (PTSD), and TBI are often considered "invisible injuries" because they are not as readily observable to others as a broken leg, scar, or amputation may be, yet they may impart significant disruption to life and impose functional disability. Within the broad categories of stress-related disorders and TBI, PTSD and mild TBI (mTBI) have emerged as particular concerns—both in their own right and as conditions that occur concomitantly in a large number of people. Although recent wars have focused attention on PTSD and mTBI in an unprecedented way, the issues surrounding the causes, effects, and management of these interacting conditions are relevant to both civilian and military contexts.

U.S. population-based surveys estimate the prevalence of PTSD in the general community to be at 7–8% (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), although rates of PTSD climb in higher risk groups such as people exposed to community violence (e.g., Silva et al., 2000; Zinzow et

al., 2009) or those serving in the military (e.g., Dohwenrend et al., 2006; Kulka et al., 1990). TBI likewise affects many people. Over 1.1 million people with TBI seek care annually in U.S. emergency rooms, with the majority of these TBIs classified as mild (Corrigan, Selassie, & Orman, 2010). As striking as these emergency room figures are, they may underestimate the occurrence of milder TBIs, given that many people will not seek emergency services if their loss of consciousness is brief and their symptoms are transient.

The prevalence rates of PTSD and mTBI in American service members returning from Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) have been reported to be as high as 13.8% and 19.5%, respectively (Tanielian & Jaycox, 2008), although rates of both PTSD and TBI among military personnel deployed from international forces were reported to be lower (Fear et al., 2010; Rona et al., 2011). As various chapters of this volume suggest, the prevalence of PTSD and mTBI in service members returning from OEF and OIF is difficult to capture (Carlson et al., 2011). Variance in estimates is likely attributable to limitations to real-time documentation of war-zone injuries and events, and differences in sampling strategies and time frames. The challenges in estimating prevalence also highlight the ambiguity surrounding the operational definitions of PTSD and mTBI, and how they are assessed. These issues are critical for all practitioners, regardless of the civilian or military context in which they work, and are addressed across many chapters of this volume.

In addition to the occurrence of each condition when considered alone, PTSD and mTBI frequently co-occur. For example, among those veterans in a RAND study reporting TBI, 33.8% also screened positive for PTSD. A study of 2,525 deployed U.S. Army infantry soldiers likewise showed high rates of PTSD–mTBI comorbidity (Hoge et al., 2008). In the U.S. Army study, 44% of soldiers reporting TBI with any alteration of consciousness also screened positive for PTSD, and 27% of soldiers reporting TBI with outright loss of consciousness screened positive for PTSD. In some treatment contexts serving combat veterans the comorbidity may occur even more frequently (Lew et al., 2009).

There are also a number of civilian contexts that may lead to both PTSD and mTBI. Motor vehicle accidents, domestic violence, and other types of assaults, for example, all confer risk of both PTSD and TBI. The commonality of these injury contexts is that the events that result in brain injury are often also psychologically traumatic. Further, in some contexts (e.g., combat, domestic violence), even if the specific event leading to a TBI does not precipitate PTSD, the TBI occurs in a context of persistent and repeated exposure to extreme psychological stress and/or life threat. This volume takes into consideration a range of contexts that might lead to the development of comorbid PTSD and mTBI.

Definitions

Although PTSD can accompany a TBI of any severity level, we focus in this volume on milder TBIs for two primary reasons. First, mTBI is among the most prevalent of TBIs and is seemingly more likely to be associated with PTSD than moderate and more severe TBI (Zatzick et al., 2010). Thus, many clinicians more commonly confront the PTSD–TBI comorbidity at the milder end of the TBI spectrum. Second, there is significant controversy regarding health care policy and best clinical practices for patients presenting with PTSD and mTBI (Hoge, Goldberg, & Castro, 2009). Because the milder forms of TBI may be the least well understood of all TBIs, it is mTBI that drives the sometimes polarizing policy and conceptual debates that currently permeate the field and leave clinicians searching for answers (Sayer et al., 2009).

As several chapters in this volume highlight, ambiguous definitions surrounding mTBI and related constructs add to the confusion. "Mild TBI" represents a relatively broad range of injury attributes and outcomes. Most definitions of TBI, including those espoused by multi-agency consensus conferences (Menon, Schwab, Wright, & Maas, 2010), the World Health Organization (Ruff et al., 2009), the Centers for Disease Control and Prevention (National Center for Injury Prevention and Control, 2003), and the American Congress of Rehabilitation Medicine (1993) require two basic components: (1) at least a transient disruption in brain function and/or other evidence of brain injury and (2) the precipitant of an external force to the head. Operational definitions set upper severity limits for mTBI, the most common of which are (1) loss of consciousness (LOC) not to exceed 30 minutes and (2) posttraumatic amnesia (PTA) (i.e., the duration of time in which the formation of new memories is impaired following the injury) not to exceed 24 hours. The term "concussion" is often used interchangeably with mTBI and has been recommended as a less stigmatizing term as compared to "TBI" (Hoge et al., 2009), but as Bigler and Maxwell (Chapter 2, this volume) point out, not all experts agree that the two terms are synonymous.

Further contributing to potential misunderstanding, the brain injury may be confused with its functional sequelae, which are often labeled as postconcussive symptoms or postconcussive syndrome (PCS). Some commentators argue that TBI should be considered a chronic disorder that extends beyond the initial injury (Masel & DeWitt, 2010) and that the original injury is rarely a discrete, time-limited event. However, as described by Bigler and Maxwell (Chapter 2, this volume), evidence is just now emerging regarding the potential chronicity of the actual pathophysiology of milder injuries. The implications of this evidence also suggest that TBI severity is continuous as a construct, rather than reflecting discrete categories.

Thus, the term "mild TBI," especially when referring to an enduring disorder, may imply different meanings to different researchers and clinicians. Clear definitions of mTBI are needed to improve clinical care, standardize research, and facilitate better demarcations between mTBI and its comorbid disorders.

Definitions of PTSD are somewhat more straightforward but are likely to show some continued evolution as formal taxonomic systems are revised. Although revision of PTSD criteria are currently under consideration for the next edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (Friedman, Resick, Bryant, & Brewin, 2011), authors throughout this volume adopt the current criteria for PTSD delineated in the text revision of the fourth edition of the DSM (DSM-IV-TR; American Psychiatric Association, 2000). Like TBI, in thinking about PTSD, it is important to distinguish between the initial traumatic event, one that might or might not lead to PTSD, and the symptoms and functional impairment that can result from psychological trauma exposure. Finally, it is relevant that PTSD symptoms occur on a continuum and do not necessarily remain static in time in terms of their presence, severity, or functional impact.

PTSD and mTBI: A Conceptual Framework

As displayed in Figure 1.1, we view the potential causal pathways between psychological trauma/PTSD and mTBI/postconcussive symptoms as complex, leaving open the possibilities of bidirectional influences. Not mutually exclusive, PTSD and mTBI (and associated postconcussive symptoms) may also occur independently as a result of a common event or context that increases the risk of both psychological trauma exposure and brain injury. Figure 1 provides a combat example, but the same relationships would hold true of moving vehicle, industrial, or occupational accidents; interpersonal assaults; and other circumstances that are both psychologically traumatic and confer risk of brain injury. Although not depicted in the figure, the clinical presentation of the comorbidity is also complicated by other factors such as orthopedic injury, pain conditions, substance abuse and dependence, and other mental health disorders, such as depression.

In keeping with this framework, we constructed this volume to address the complexities of caring for patients with comorbid PTSD and mTBI, rather than limiting discussion to an oversimplified dichotomy surrounding the question of whether postconcussive symptoms are primarily "neurological" or "psychiatric" in nature. The clinical presentation of the comorbidity is indeed complex, with PTSD and mTBI characterized by overlapping symptom presentations, neural substrates, and functional consequences. It is important to remember that sophisticated research into the mechanisms

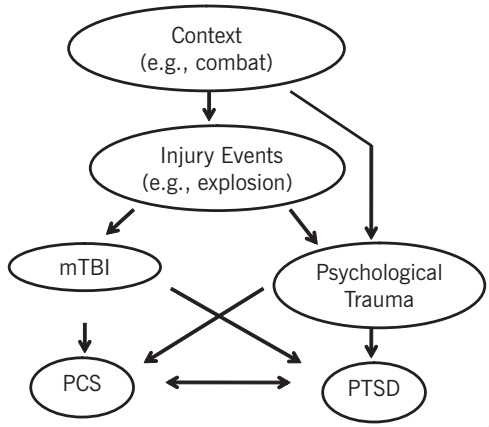

FIGURE 1.1. Hypothesized relationships among context, injury events, mild traumatic brain injury (mTBI), psychological trauma, postconcussive symptoms (PCS), and posttraumatic stress disorder (PTSD).

of mTBI is in its infancy, and there is much we simply do not yet understand about the effects of mild brain injuries. We also recognize that each of these conditions is also commonly associated with other psychiatric disorders including depression and substance abuse, and other physical comorbidities such as chronic pain. Our emphasis centers on how best to recognize this comorbidity and its associated clinical problems, how best to structure interventions for various aspects of the presentation, and which factors might be worthwhile to consider from the perspective of health care delivery systems. More specifically, we attempt in this volume to address the following four questions:

1. To what extent and via what mechanisms do PTSD and mTBI potentially complicate each other?
2. Which other factors complicate recovery from PTSD and mTBI?
3. What do we know about treatment of patients with comorbid PTSD/mTBI?
4. How should care of patients with PTSD and mTBI be optimally structured?

Organization of the Volume

The first section of the volume describes the mechanisms underlying PTSD and mTBI, the clinical presentation and potential courses of each condition,

and mechanisms by which the two conditions may complicate each other. Bigler and Maxwell (Chapter 2) define mTBI and describe the scale of the problem with epidemiological data. The thrust of the chapter, however, is on understanding the mechanisms of neural injury, the potential course of physiological recovery, and how neuroimaging may be used to learn more about the smaller subset of mTBI survivors who do not recover quickly from the injury and continue to express symptoms and display marked functional impairment. Iverson (Chapter 3) presents a biopsychosocial model of mTBI, emphasizing the role that psychosocial and other contextual factors may contribute in addition to underlying pathophysiology in impeding recovery from brain injury. Hayes and Gilbertson (Chapter 4) describe the clinical presentation of PTSD and the psychosocial, environmental, and neural factors that alter risk of its development and maintenance. In their chapter, Hayes and Gilbertson additionally discuss the neurobiological and neuropsychological correlates of PTSD, including areas of overlap with TBI. Finally, Verfaellie, Amick, and Vasterling (Chapter 5) draw more broadly from the literature on non-TBI sources of neurocognitive variation to consider TBI-related neurocognitive mechanisms that may heighten risk of PTSD or affect the course and expression of PTSD.

The second section of the volume recognizes that PTSD and TBI do not occur in a vacuum and that associated conditions may interfere with recovery from them. Although there are many factors that may complicate recovery from both mTBI and PTSD, we focus on two of the most common and potentially debilitating of such associated conditions: chronic pain and substance abuse. Otis, Fortier, and Keane (Chapter 6) highlight the frequency in which chronic pain occurs with PTSD and mTBI and describe the complexities of the clinical presentation in patients with PTSD, mTBI, and pain disorders. They further discuss treatment options, drawing from an existing integrated intervention for PTSD and chronic pain. Similarly, Najavits, Highley, Dolan, and Fee (Chapter 7) discuss patterns of substance use predating and following PTSD and TBI and how the trimorbidity of substance use disorders, PTSD, and TBI, may complicate treatments. Najavits and her colleagues draw from established interventions designed to address PTSD and substance use to make recommendations for treatment of the trimorbidity.

The third section of the book addresses the clinical management of comorbid PTSD and mTBI. Because of the complexities of assessing PTSD and mTBI, especially when they are comorbid, we devote two chapters to assessment. In the first, Ulloa, Marx, Vanderploeg, and Vasterling (Chapter 8) focus on assessment of PTSD and mTBI when considered alone and in combination. In the second, Elhai, Sweet, Guidotti Breting, and Kaloupek (Chapter 9) consider the special circumstances of evaluations that are performed in litigation, compensation/disability evaluations, and other

contexts that pose threats to the validity of the assessment process. The next two chapters discuss psychosocial treatment interventions for PTSD and mTBI. Ponsford (Chapter 10) focuses on cognitive rehabilitation therapy for mTBI. Bryant and Litz (Chapter 11) discuss cognitive-behavioral interventions for PTSD, including how they might be best structured when mTBI factors into the clinical presentation. The final two chapters of the volume center on the context in which care is delivered to patients with PTSD and mTBI. Bryant, Castro, and Iverson (Chapter 12) consider military mTBI in particular as a special context and describe clinical considerations relevant to the military environment and combat-related TBIs. Finally, Hendricks, Krengel, Iverson, Kimerling, Tun, Amara, and Lew (Chapter 13) adopt a health economics approach to consider factors that could potentially guide allocation of clinical resources in contexts in which such resources are limited. Although Hendricks et al. use Veterans Health Administration data to illustrate the costs associated with treatment, many of the factors discussed can be more broadly generalized to other health care contexts.

We conclude with a synthesis of the information presented throughout, returning to the questions posed above. Through this volume, we hope to spark continued discussion of the complexities of providing care to patients with PTSD and mTBI using some of the differing perspectives on the topic to move toward rational, well-considered approaches to service delivery. In doing so, we anticipate that the work contained within this volume will have broad appeal to the diverse set of professionals from mental health, rehabilitation, and neuroscience fields that care for, and through empirical research, try to better understand these patients with the complex comorbidity of PTSD and mTBI.

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