Curr Psychiatry Rep (2016) 18: 63
DOI 10.1007/s11920-016-0699-9

Treatment Options for Individuals with PTSD and Concurrent TBI: A Literature Review and Case Presentation

Hans R. Watson[1] · **Musammar Ghani**[1] · **Terry Correll**[1]

Published online: 25 May 2016

Abstract Posttraumatic stress disorder (PTSD) is a well-studied mental health condition with existing guidelines and algorithms for treatment of PTSD. Those guidelines, while acknowledging an increased complexity, fail to provide clear PTSD treatment guidelines when an individual has a concurrent traumatic brain injury (TBI) diagnosis. Therefore, a literature review along with an accompanying case presentation is presented to demonstrate the minimum necessary considerations for approaching treatment of this complex population. Treatment approaches must be lead by providers that have the expertise and training necessary to consider all facets of the patient and their potential options. The provider must consider the pathophysiology of PTSD and TBI and be capable of leading a team to identify the patient's source(s) of dysfunction, current cognitive abilities, and potential indications for psychotropic medications and/or other types of therapeutic intervention.

Keywords Posttraumatic stress disorder · Traumatic brain injury · Cognitive behavioral therapy · Psychodynamic therapy · Psychopharmacology · Neuropsychological tests

This article is part of the Topical Collection on *Military Mental Health*

✉ Hans R. Watson
hansrwatson@yahoo.com

[1] Department of Psychiatry, Wright State University-Boonshoft School of Medicine, 627 S. Edwin C. Moses Blvd, Dayton, OH 45417-1461, USA

Introduction

The conflicts in Iraq and Afghanistan shine a light on the important problems of posttraumatic stress disorder (PTSD) and traumatic brain injury (TBI). While PTSD and TBI each have been studied individually and treatment guidelines have been established for both conditions separately, studies fail to establish clear PTSD treatment guidelines if the two conditions co-occur. Although some sources give valuable considerations to treating PTSD when combined with a TBI, it is difficult to find any source that gives specific steps that could be sequentially followed in the workup and treatment of individuals with concurrent TBI [1–4].

The focus of this paper is to propose a generalized treatment approach and present a case that demonstrates a simplified and comprehensive treatment plan for individuals with PTSD and a concurrent non-acute TBI. This paper intends to provide an overview that covers the minimum necessary steps and sequence that the treatment team should be discussing and considering in every workup. The details of how to accomplish each step will be identified by the treatment team and individualized with each patient. None of the suggested steps are new, but instead, we are attempting to give some organization to the approach. This project was inspired by a patient (Mr P) that the first author was assigned to see in the outpatient psychiatry residency clinic.

Neurobiology of PTSD

The brain of a healthy individual involves an interplay of the amygdala, frontal lobe, and hypothalamus [4, 5, 6••]. The amygdala is part of the limbic system and activates in response to fear and pleasure [4, 5, 6••]. When activated due to danger, the amygdala signals other parts of the brain to initiate the

fight or flight response [4, 5, 6••]. The amygdala also works with the hypothalamus to assign emotion to impactful experiences; thus, the hypothalamus converts the experience into a long-term memory for future use [4, 5, 6••]. The frontal cortex has the difficult job of analyzing the current situation and using previous memories to determine the true nature of the current situation and whether it is truly dangerous or not [4, 5, 6••]. The frontal lobe then directs the most appropriate action if the current situation is dangerous or suppresses the amygdala in non-dangerous situations [4, 5, 6••].

In individuals with PTSD, the frontal lobe is usually hypoactive and the amygdala is hyperactive. [7] Hypoactive frontal lobes have two disadvantages: first, the ability to distinguish between a true threat and a benign or false threat is diminished. Second, the calming or suppressing effect on the amygdala during a benign or false threat is reduced. These two disadvantages combine to establish a hyperactivated and hypervigilant state for the individual with PTSD. Treatment and resolution depend on reactivation or restoration of the frontal cortex function. The restoration of the frontal lobe functions will in turn suppress the amygdala in a benign or false threat situation [4, 5, 6••, 7, 8].

Case Report

Mr. P is a 44-year-old right-handed, married white male that recently retired from a 20-year career with the US Marine Corps (USMC). Mr. P was deployed multiple times to both Iraq and Afghanistan. During his combat duties, he suffered from the results of being in seven separate incidents of roadside bombings. He experienced loss of consciousness with posttraumatic amnesia (PTA) in at least three roadside bombing incidents; the PTA lasted for at least a few hours but less than a day in each case. In spite of the PTA, Mr. P continued to perform his duties overseas and upon return from deployment. Two years after his return from the last deployment, he received a post-deployment mental health screening that revealed short-term memory problems and symptoms consistent with PTSD.

Three years after his last deployment, Mr. P retired from the USMC and moved to our local area to be near family. Due to a referral from his last duty station, Mr. P presented to the outpatient mental health clinic in our area with his wife. Mr. P's chief complaint was "I need a refill of my meds." Only when asked during review of systems did he and his wife reveal that since his last deployment, he had significant trouble with short-term memory and difficulty tolerating public places. He reported that in his final deployment, he experienced two roadside bombings and was present when multiple comrades were killed in action. After his last deployment, Mr. P started experiencing daily headaches that affected his vision and cognition. The wife reported that there was a clear change in the patient's personality since returning from the last deployment. They revealed that eventually, the memory impairments impeded the patient's ability to perform his duties, and his commander ordered a medical evaluation that resulted in a referral to the TBI treatment facility near his duty station. A review of the previous medical records from the TBI treatment facility revealed historical diagnoses of PTSD, an unspecified TBI, and unspecified cognitive disorder.

Mr. P revealed that pre-deployment, he had been a "very chill guy" that was slow to become angry. He reported that he did very well in his duties as a senior non-commissioned officer in the field artillery. He reports that prior to his combat experiences, he was able to learn and remember easily the complex calculations required to direct aiming, elevation, propellent type/amount, and round type for the artillery guns. He reported that his quick learning ability was part of why he was promoted so rapidly. He shared that he was comfortable taking extra duties and manage multiple important tasks or supervising multiple groups at the same time. He reported that even participation in combat did not make him very nervous for the first few deployments. During the initial appointment, Mr. P and his wife shared that since returning from his final deployment, he was a "changed person." They reported that he was "always forgetting what he was doing or important appointments," did not leave the house for days or weeks because he was "on edge" in public places and always "scanning for danger". They reported poor sleep, frequent nightmares, and difficulty relaxing. He endorsed additionally a lack of interest in pleasurable activities to include sex and feeling excessive guilt, low energy, diminished concentration, feeling very impatient and irritated with family and friends, increased anxiety over insignificant problems, hypervigilance, intrusive combat-related daytime memories, difficulty going to public places, continued combat-related nightmares, feeling emotionally detached, actively isolating from friends, struggling to find purpose in life, difficulty concentrating on tasks, and forgetfulness.

Mr P was prescribed dextroamphetamine/amphetamine (Adderall XR) 20 mg daily by the previous providers at the TBI center to treat the unspecified cognitive disorder. Mr. P denied any improvement in memory while he was taking the dextroamphetamine/amphetamine. He complained of the following medication side effects: diminished appetite, insomnia, worsening headaches, and tremor. Mr. P reported that he had been out of the medication for over a week prior to his initial appointment at our clinic and had noticed improved sleep, elimination of the tremor, and improved appetite. He denied any effect on his memory upon discontinuation of dextroamphetamine/amphetamine. He denied use of any other medications or supplements to help with memory or cognition. This was confirmed with a review of the medical record and by his wife.

In spite of the change in personality, headaches, and memory problems, Mr. P had not received any head imaging before his initial appointment at our outpatient clinic. He did receive botulinum injections for headaches, limited neuropsychological testing at the military TBI center, and a diagnosis of unspecified cognitive disorder. However, the neuropsychological testing did not address memory impairment causes or location of brain dysfunction. An MRI and neurologic consult were ordered after the patient's initial appointment in our clinic. The neurologist found no evidence of gross damage to the brain structures on head imaging. After normal MRI results, neuropsychological testing was performed and showed intact memory encoding and recall with some slowed processing. A treatment team meeting between psychiatry and neurology determined that the patient's short-term memory impairment was likely due to PTSD and less likely to be a result of the TBI.

The following labs were drawn: complete metabolic panel (CMP), complete blood count (CBC), thyroid stimulating hormone (TSH), B12/folate, electrolytes, renal panel, hepatic panel, and rapid plasma reagin (RPR). No abnormalities were identified.

At the initial appointment at our clinic, Mr. P notified his psychiatric provider (first author) that because of significant side effects and no clear improvement with regard to cognition, he would not continue taking the dextroamphetamine/amphetamine. He was given options for treatment that included psychotherapy, medications, and exposure therapy. The patient chose to initiate an selective serotonin reuptake inhibitor (SSRI) (sertraline) which was started at 25 mg with instructions to increase the dose to 50 mg daily after 4 days. He was asked to follow up in 5 weeks. He declined any talk therapy or exposure therapy.

At the follow-up appointment, the patient reported that he had followed the titration as scheduled without any side effects. However, about 5 days prior to our appointment, he reported that he "suddenly started feeling like a zombie." He reported that this was the first incidence of the "zombie feeling" and that he had not missed a dose before that day. He reported that the "zombie feeling" lasted 2 days until he discontinued the medication; within 6–7 h of stopping the medication, he reports being fully recovered and returned to baseline. The psychiatric provider discussed other medication options; however, the patient declined the use of any other medications at that time. He did accept an invitation to engage in weekly individual psychotherapy with this psychiatric provider (first author).

The patient and the first author decided to start brief psychodynamic therapy. The first author felt that while the patient was unable to identify many of the causes for his symptoms, the patient had significant ego strengths to undergo brief psychodynamic psychotherapy. The patient started weekly 50-min psychotherapy sessions with the goals of decreasing or eliminating combat-related nightmares, eliminating intrusive daytime memories, decreasing the avoidance symptoms in public places, and improving his ability to engage in interpersonal relationships.

The initial phase of psychotherapy focused on identifying benign stimuli that were causing significant hypervigilance symptoms. Once the patient was aware and able to process the thoughts that were causing feelings of danger with benign stimuli, the intensity of the hypervigilance and associated irritability with close family members reduced significantly. The middle phase of therapy involved exploring the patient's upbringing, patterns of attachment, positive and negative familial identifications, and how it could have contributed to the patient's developing PTSD symptoms after repeated combat exposure and loss of comrades. By the end of the middle phase of treatment, the patient reported that the nightmares and intrusive thoughts throughout the day had decreased from nightly/daily occurrence to once a month or less. The final phase of psychotherapy focused on setting future goals in the arena of personal, professional, and social settings. By the end of brief psychodynamic therapy which lasted 14 sessions, Mr. P's symptoms had abated sufficiently that he no longer met criteria for PTSD or depression. Repeat neuropsychological testing 5 months after the termination of therapy showed no change to his IQ, but improved short-term memory.

Mr. P followed up with the second author in the outpatient clinic after termination of psychotherapy and decided to start hydroxyzine 25 mg twice a day as needed for anxiety. Mr. P continues to function near pre-deployment levels and is currently taking online business college courses in preparations to start a small agricultural business. He is sleeping well and has not reported any nightmares. His wife reports that his irritability has decreased significantly, and his familial interactions/relationships have greatly improved. His wife reported that his personality was "back to the guy I married" and that his memory was "almost as good as before."

Compared to pre-TBI, he has some slightly slowed processing speed but he has not had a recurrence of the PTSD symptoms, memory impairments, or other notable cognitive deficits.

Treatment of PTSD when TBI Is Present

The treatment of PTSD in the presence of a non-acute TBI should be accomplished in two phases. First, we recommend thorough evaluation of the TBI severity and symptoms. Second, the treatment team should utilize the results from the TBI workup to identify the best approach for the PTSD treatment. The information in this paper applies to a non-acute TBI (often called chronic TBI) patient with PTSD.

Section I: Neuropsychiatric Workup

Neurological Exam and Imaging

The initial workup of a patient with a TBI should involve a comprehensive neurological exam with head imaging. Anytime a patient is exposed to trauma with shearing forces and has loss of consciousness or impairment of cognition, imaging of the head should be obtained [1–3, 7–10]. The ability to obtain head imaging is not always possible in combat zones. Personal experience of the first author has been that in combat, post concussive symptoms are often ignored and are likely masked by the sympathetic hormonal response from the combat experience. Therefore, in the outpatient setting, it is increasingly important to obtain imaging to rule out anatomic or organic defects as the cause of continued neuropsychiatric symptoms like poor memory, decreased attention, impaired cognition, personality changes, or headaches.

If any organic pathology is detected on imaging, psychiatry is likely to assume the supporting role and neurology is most likely to assume the lead for further workup.

Mr. P was an example of someone who never received head imaging even though there were repeated traumas with change in personality and cognition.

Neuropsychological Testing

Anytime memory or cognitive impairments are present with TBI, neuropsychological testing is recommended. While it is reasonable to wait for results of the neurological exam and imaging before starting neuropsychological testing, this step remains very important. Neuropsychological testing helps to identify whether memory problems are due to problems of encoding and recall or due to another interfering process such as PTSD [11]. It can further identify parts of the brain that have dysfunction that is not visible on imaging. Quality testing often reveals the patient's current abilities and modalities of learning [8, 9, 11]. Understanding the patient's cognitive strengths and weaknesses is important when deciding how to approach PTSD treatment.

Mr. P's neuropsychological testing showed slower processing speed with no impairment in memory encoding or recall.

Laboratory Examination

Labs should be obtained concurrently with imaging to rule out metabolic causes of altered mental status. We recommend the following labs when evaluating and planning treatments for patients with a history of TBI: complete metabolic panel (CMP), complete blood count (CBC), thyroid stimulating hormone (TSH), B12/folate, electrolytes, renal panel, hepatic panel, blood levels of prescribed medications, and rapid plasma reagin (RPR) [8–10].

Labs for Mr. P were all within normal limits.

Section II: Psychiatric Treatment Options for PTSD

The information from the neuropsychiatric workup should be considered when identifying individual options for treatment of the PTSD.

Medications

Medications can be helpful in the treatment of PTSD and TBI, but they can also be harmful. Review studies demonstrate that there is insufficient data to confirm whether medications alone or combined with psychotherapy are superior [12]. This necessitates the treatment team to identify the etiology of the patient's symptoms and how the TBI might affect the brain function.

Medication decisions will be affected by the outcomes of the initial neurological, physical, and psychiatric evaluations [9, 10]. In 2002, David Mintz explained how medications can be either helpful or harmful (nocebo effect) depending on the patient's perspective and opinions about pharmacotherapy [13]. Therefore, a provider must make medication choices only after eliciting the patient's feelings about being on medications and what meaning the patient places on taking psychiatric medications. This can help to reduce the nocebo effect from medications [13].

While medications can prove to be helpful with PTSD and TBI, recent PTSD studies have shown that medications alone provide only a small to moderate improvement [14••]. When treating concurrent PTSD and TBI, understanding the limits of pharmacotherapy and the benefits of other treatment approaches is essential for formulating a holistic treatment plan. In our experience, although individuals can benefit by the addition of medications, they can also develop the nocebo effect, in which the patient experiences idiosyncratic/paradoxical responses to medications.

Antidepressant medications such as selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants (TCAs), or serotonin-norepinephrine reuptake inhibitors (SNRIs) are identified in both TBI and PTSD studies as the best medication classes for long-term improvement [14••, 15••]. These results are not surprising when applying a neurobiological perspective; both SSRIs and SNRIs show increased neurogenesis in areas like the hippocampus in other mental health conditions such as depression [16]. Therefore, we recommend following the PTSD recommendations to use SSRIs and SNRIs as the first-line treatment in patients with PTSD and concurrent TBI.

Although benzodiazepines use in PTSD treatment is still common, multiple recent studies have shown that benzodiazepines are harmful and should not be used in PTSD or TBI [17•, 18••]. We recommend avoiding benzodiazepine medications or other anticholinergic medications where possible.

Mr. P showed a nocebo effect to medications. We explored this in psychotherapy and he came to understand and shared with the first author that only prescribing medications was perceived as the provider not caring enough to spend the time needed for recovery. Therefore, with Mr. P, therapy was the best option for treatment.

Psychotherapy

Choosing a psychotherapy type should consist of considerations about which type would best benefit the patient and less about what is the latest trend. Studies are showing that many different psychotherapy types are equally effective in long-term outcomes, provided they have a trauma focus to the therapy [19].

According to a multiple sources, psychotherapy is a vital non-pharmacological intervention for PTSD [1, 2, 4, 6••, 8]. Studies show that psychotherapy improves the frontal lobe and hypothalamus' functioning resulting in the calming of the amygdala [6••]. It does this by fear extinction, a process where the brain stops identifying benign stimuli as dangerous [6••]. While some studies link certain psychotherapeutic modalities to a more rapid improvement of acute symptoms, long-term studies show similar outcomes between different psychotherapeutic modalities that have an exposure element [20•].

Psychotherapy considerations for patients with TBI damage to the orbitofrontal and/or medial dorsolateral frontal cortex must include options that utilize other parts of the frontal cortex. This is often accomplished in exposure therapy that physically places the patient in an environment reminiscent of the original trauma-inducing stimulus [9, 10]. Being physically present can do this because it stimulates different parts of the frontal cortex thus increasing the chance of utilizing a healthier part of the brain in the learning process [9, 10].

Providers who treat patients with PTSD and concurrent TBI should learn and understand the strengths and weaknesses of the different psychotherapeutic modalities. These include trauma-focused cognitive behavioral therapy (CBT), eye movement desensitization and reprocessing (EMDR) therapy, brief psychodynamic psychotherapy and more [19]. This empowers the provider to develop, with the patient, an individualized psychotherapeutic plan that synergizes the strengths of the different therapeutic modalities with the strengths and abilities of the patient.

Conclusion

The signature injuries of the wars in Iraq and Afghanistan are PTSD and TBI [21]. Indeed, many veterans of the wars in Iraq and Afghanistan will have symptoms of both TBI and concurrent PTSD [22]. Due to the variations in TBI presentation, detailed PTSD treatment guidelines in the setting of comorbid TBI would be difficult to establish and are not presently available. Meanwhile, the guidelines that are established for treatment of PTSD and TBI are far too often ignored with treatment deviating from established guidelines for PTSD or TBI [17•, 18••]. This paper provides a literature-based overview of the bare minimum steps involved in the evaluation and treatment of patients with PTSD and comorbid TBI. This overview consists of a complete neurological exam, head imaging, neuropsychological testing, and laboratory evaluation. Following the neurologic workup, the evaluating team should consider the results of that workup, as well as the patient's preferences, strengths, and weaknesses to design a personalized treatment plan for the patient suffering from the debilitating symptoms of PTSD and concurrent TBI.

Compliance with Ethical Standards

Disclaimer The views expressed in this paper are those of the authors and do not reflect the views of the US Department of Defense. None of the authors have a conflict of interest to report.

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1. Defence Centers of Excellence For Psychological Health & Traumatic Brain Injury [Internet]. Maryland: Defence Centers of Excellence For Psychological Health & Traumatic Brain Injury; 2016 [cited 2016 Feb 15]. Available from: http://www.dcoe.mil/psychologicalhealth/provider_resources.aspx.
2. VA/DoD Clinical practice guideline for management of post-traumatic stress [Internet]. Washington DC: U.S. Department of Veterans Affairs National Center for PTSD; Date of first publication unknown 2016 Jan 5; [cited 2016 Feb 15]. Available from: http://www.ptsd.va.gov/professional/index.asp.

3. Kaufman DM, Milstein MJ. Kaufman's clinical neurology for psychiatrists. 7th ed. Traumatic Brain Injury. New York: Saunders Elsevier; 2015. p. 527–536.
4. Sadock BJ, Sadock VA, Ruiz P. Kaplan & Sadock's synopsis of psychiatry, Trauma and stressor related disorders. 11th ed. New York: Wolters Kluwer; 2015. p. p. 437–450.
5. Sadock BJ, Sadock VA, Ruiz P. Kaplan & Sadock's synopsis of psychiatry, Neural sciences. 11th ed. New York: Wolters Kluwer; 2015. p. 1–92.
6.•• Welton R, Kay J. The neurobiology of psychotherapy. Psychiatric Times [Internet]. 2015 October [cited 2016 Feb 15]. Available from Psychiatric Times: http://www.psychiatrictimes.com/neurobiology-psychotherapy **This study used neuroimaging to demonstrate that therapy restores frontal lobe functioning and brings the amygdala back into regulation. Thus demonstrating the mechanism of action found with the resolution of the neuroanatomic dysfunction found in PTSD**.
7. Hoffman SW, Harrison C. The interaction between psychological health and traumatic brain injury: a neuroscience perspective. The clinical neuropsychologist [Internet]. 2009 October [cited 2016 Feb 15]. Available from Taylor & Francis Online: http://www.tandfonline.com/doi/abs/10.1080/13854040903369433.
8. Tasman A, Kay J, Lieberman JA, First MB, Maj M. Psychiatry. 3rd ed. In: Meaney MJ, LeDoux JE, Liebowitz ML. Neurobiology of anxiety disorders. West Sussex, England: Wiley-Blackwell; 2008 p. 317–338.
9. Silver JM, McAllister TW, Yudofsky SC. Textbook of traumatic brain injury. 2nd ed. Arlington: American Psychiatric Publishing Inc; 2011.
10. Arciniegas DB, Zasler ND, Vanderploeg RD, Jaffee MS, Garcia TA. Management of adults with traumatic brain injury. 1st ed. Arlington: American Psychiatric Publishing Inc; 2013.
11. Sadock BJ, Sadock VA, Ruiz P. Kaplan & Sadock's synopsis of psychiatry. 11th ed. Examination and diagnosis of the psychiatric patient. New York: Wolters Kluwer; 2015. p. 192–289.
12. Hetrick SE, Purcell R, Garner B, Parslow R. Combined pharmacotherapy and psychological therapies for post traumatic stress disorder (PTSD). The Cochrane Collaboration. 2010: issue 7.
13. Mintz D. Meaning and medication in the care of treatment-resistant patients. Am J Psychother. 2002;56(3):322–37.
14.•• Hoskins M, Pearce J, Bethell A, Dankova L, Barbui C, Tol WA, et al. Pharmacotherapy for post-traumatic stress disorder: systematic review and meta-analysis. The British Journal of Psychiatry. 2015;206:93–100. **This study demonstrates that certain SSRI medications have a small benefit to PTSD treatment**.
15.•• Puetz TW, Youngstedt SD, Herring MP. Effects of pharmacotherapy on combat-related PTSD, anxiety, and depression: a systematic review and meta-regression analysis. PLoS One [Internet]. 2015 May [cited 2016 Feb 15]. 10(5) Available from Public Library of Science: http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0126529 **This study demonstrates the lack of data to support efficacy of a unilateral pharmacologic approach for PTSD Treatment**.
16. Anacker C, Zunszain PA, Cattaneo A,Carvalho LA, Garabedian MJ,Thuret S, et al. Antidepressants increase human hippocampal neurogenesis by activating the glucocorticoid receptor. Pub Med Central [Internet]. 2011 Jul [cited 2016 Feb 15]. Available from PMC: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3121947/.
17.• Abrams TE, Lund BC, Bernardy NC, Friedman MJ. Aligning clinical practice to PTSD treatment guidelines: medication prescribing by provider type. Psychiatric Services [Internet]. 2013 Feb [cited 2016 Feb 15]; 64 (2):142–148. Available from Psychiatric Services: http://ps.psychiatryonline.org/doi/full/10.1176/appi.ps.201200217 **This study demonstrates that in spite of literature supported contraindications to certain traditional treatment for PTSD, such as benzodiazapines, providers continue with traditional treatment modalities in spite of the contraindications**.
18.•• Guina J, Rossetter SJ, DeRhodes BJ, Nahhas RW, Welton RS. Benzodiazepines for PTSD: a systematic review and meta-analysis. Journal of Psychiatric Practice. 2014 Jul. 21(4) 281–303. **This study demonstrates that, though traditionally used, the long term harm associated with the use of benzodiazepines merits a contraindication**.
19. Bisson JI, Roberts NP, Andrew M, Cooper R, Lewis C. Psychological therapies for chronic post-traumatic stress disorder (PTSD) in adults (Review). The Cochrane Collaboration. 2013: issue 12.
20.• Levi O, Bar-Haim Y, Kreiss Y, Fruchter E. Cognitive-behavioural therapy and psychodynamic psychotherapy in the treatment of combat-related post-traumatic stress disorder: a comparative effectiveness study. Clinical Psychology and Psychotherapy [Internet]. 2015 Available from wiley online library: http://onlinelibrary.wiley.com/doi/10.1002/cpp.1969/abstract **This study demonstrates that long term outcomes PTSD treatment are not dependent on therapy styles as multiple styles have similar long term outcomes**.
21. Hoge CW, McGurk D, Thomas JL, Cox AL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med. 2008;358(5):453–63.
22. Carlson KF, Kehle SM, Meis LA, Greer N, MacDonald R, Rutks I, et al. Prevalence, assessment, and treatment of mild traumatic brain injury and posttraumatic stress disorder: a systematic review of the evidence. J Head Trauma Rehabil. 2011;26(2):103–15.