# TBI Symptoms Improve After PTSD Remediation With Emotional Freedom Techniques


## Abstract

A group of 59 veterans with clinical levels of posttraumatic stress disorder (PTSD) symptoms received Emotional Freedom Techniques (EFT) coaching in a randomized controlled trial. A significant percentage dropped below the clinical threshold after 6 sessions of EFT (86%, $p < .0001$) and remained subclinical at 3-month and 6-month follow-ups. Traumatic brain injury (TBI) and somatoform symptoms isolated from the data set for detailed analysis are presented in the current paper. Compared with pretest, significant reductions in TBI symptoms were found after 3 sessions, with a further reduction after 6 months (–41%, $p < .0021$). Participant gains were maintained on 3-month and 6-month follow-up ($p < .0006$). These results point to the poorly defined distinction between TBI and PTSD symptoms, the potential for partial TBI rehabilitation as a sequel to successful PTSD treatment, and the possibility of long-term maintenance of clinical gains.


**Keywords**: veterans, TBI, PTSD, trauma, Emotional Freedom Techniques, EFT

**Introduction**

Traumatic brain injury (TBI), in an oft-repeated phrase, has been called the "signature injury" of military personnel deployed to the war zones of Afghanistan and Iraq (e.g., Okie, 2005; Rona, 2012; Sammons & Batten, 2008). TBI along with posttraumatic stress disorder (PTSD) and major depression make up the "invisible wounds" from which service members often continue to suffer even well after combat has ended (Tanielian & Jaycox, 2008). With the most conservative estimates of TBI incidence ranging between 10% and 20% of all deployed troops (Ruff, Riechers, Wang, Piero, & Ruff, 2012; Schneiderman, Braver, & Kang, 2008; Theeler, Flynn, & Erickson, 2010), overall numbers of soldiers who have suffered a TBI are likely in the hundreds of thousands. A 2010 report based on official military sources estimated that 150,000 combatants had sustained some form of brain injury since the Iraq and Afghanistan conflicts began (Miller & Zwerdling, 2010). The context of combat (McCrea et al., 2008), military culture (Polusny et al., 2011), and soldiers' concerns about the repercussions of treatment on their careers (Miller & Zwerdling, 2010; Tanielian & Jaycox 2008) all make underreporting of TBI likely, particularly among those with milder injuries. A 2008 RAND survey of 1,965 veterans estimated a TBI prevalence rate of 19% (Tanielian & Jaycox, 2008). This suggests that, of the 2.4 million U.S. troops deployed since 2001, over 400,000 may have sustained a brain injury.

Research on TBI in military populations is confounded by factors that affect both its diagnosis and its treatment. Although consensus over the definition of a TBI does exist, with that of the American Congress of Rehabilitation Medicine (ACRM, 1993) appearing most frequently in the literature, identification of TBI is not always straightforward. Mild TBI (mTBI) is defined as an external injury to the brain, which includes confusion, disorientation, or a loss or altered state of consciousness for 30 min or less; posttraumatic memory disruption lasting less than 24 hr following the injury; and a Glasgow Coma Scale score of 13–15 (ACRM, 1993). Moderate or

severe TBI will involve a loss of consciousness and posttraumatic amnesia lasting longer, substantially so in the case of severe TBI (Bryant, 2011). Placing this definition in the context of military operations, the Defense and Veterans Brain Injury Center Working Group (2006) describes mTBI as "an injury to the brain resulting from an external force and/or acceleration/deceleration mechanism from an event such as a blast, fall, direct impact, or motor vehicle accident which causes an alteration in mental status." Yet confirming that such an injury has, in fact, occurred, can be challenging, particularly in a combat environment, where a soldier may sustain other, more critical bodily injuries concurrently or may continue to fight if the injury is mild or change in consciousness is brief, failing to recognize that a head injury has even occurred (McCrea et al., 2008). Neither is an assessment of symptoms a reliable tool for identification of TBI, given TBI's comorbidity with other, psychological, conditions, particularly PTSD and depression, and their overlapping sequelae. Comorbidity rates typically range from 30% to 40% (Hoge et al., 2008; Ruff, Riechers, & Ruff, 2010; Tanielian & Jaycox, 2008). Many of the manifestations of TBI—physical symptoms, such as headache, nausea, vomiting, dizziness, balance problems, fatigue, blurriness of vision, sensitivity to light or noise, and sleep disruptions; cognitive symptoms, such as concentration and memory problems; and emotional symptoms, including irritability, anxiety, and depression—are also known to characterize PTSD (Bogdanova & Verfaillie, 2012; Defense and Veterans Brain Injury Center Working Group, 2006; Kennedy et al., 2007). Identification of TBI and mTBI has thus mostly occurred through self-report or in-depth clinical interviews (Vanderploeg, Groer, & Belanger, 2012). And although the Veterans Health Administration (VHA) has mandated that a TBI Clinical Reminder screening instrument be completed for all soldiers returning from Iraq and Afghanistan, the instrument has not yet been validated (Vanderploeg et al., 2012).

With no validated methods of screening for TBI, the high comorbidity rates of TBI and PTSD, and the likelihood of underreporting and misdiagnosis, the evidence base for mTBI interventions remains limited (Bogdanova & Verfaillie, 2012). The Presidential Commission on Care of America's Returning Wounded Warriors recommended that integrated multidisciplinary care teams oversee treatment for those with TBI to address the "full spectrum of symptoms"—physical, cognitive, emotional—often associated with such an injury (Tanielian & Jaycox, 2008), and guidelines produced by the VA Consensus Conference (2010) indicated that treatment should be symptom-focused, effectively sidelining many of the issues of accurate diagnosis and comorbidities. As noted by Bogdanova and Verfaillie (2012, p. 9),

> standard clinical management of mTBI is typically focused on "prevention through education". This approach aims to facilitate expectations of complete recovery and to prevent secondary injuries; it also focuses on specialized medical treatment to reduce associated symptoms (such as headache, mood, and sleep problems) that may adversely impact on cognitive functioning.

At the time of the RAND study (Tanielian & Jaycox, 2008), no reliable evidence existed on the efficacy of these integrated, multidisciplinary approaches, and Bogdanova and Verfaillie (2012) reported mixed findings for the use of educational interventions in treatment of mTBI.

Cumulatively, in the field of research, this lack of consensus means that "the science of treating traumatic brain injury is very young" (Tanielian & Jaycox, 2008, p. 443). In practice, it means a sizeable population potentially afflicted by the unresolved effects of TBI. In a review of studies conducted in a civilian population, Carroll et al. (2004) concluded that most people with mTBI recover within 3 to 12 months. In a military population, however, Miller and Zwerdling (2010) estimated that between 5% and 15% would go on to experience long-term problems

following TBI; and Vasterling, Verfaillie, and Sullivan (2009, p. 675) reported that as many as 44%–50% of patients with mTBI were still experiencing three or more symptoms a year following their injury, including persistent psychological symptoms (e.g., irritability, anxiety, depression), cognitive impairments (e.g., in complex attention and working memory and executive function), and somatic complaints.

The implications of persistent symptoms, even for mTBI, which comprises the vast majority of brain injuries sustained among service members, create considerable costs to both the individual and the broader society. In the presence of mTBI, work productivity, social functioning, and quality of life may all decline (Bogdanova & Verfaillie, 2012). RAND estimated that the costs of treating TBI in the year following diagnosis, comprising both acute hospital care and inpatient and outpatient rehabilitation, was in the range of $554–$854 million (in 2007 dollars), with moderate and severe TBI accounting for one third of the total number but 92% of total costs. This picture is further muddied by the same underreporting challenges cited above, as this estimate captures only those cases of TBI that were diagnosed and resulted in contact with the health care system (Tanielian & Jaycox, 2008), and by the need to extrapolate from values obtained from a civilian population because information on standard treatments used for deployment-related TBI was unavailable. The RAND report noted that while a single year of treatment is likely to suffice in estimating the per-case cost of treatment for those with mTBI, for moderate–severe TBI, costs are likely understated, factoring in neither treatment nor productivity costs that continue after the first year. Of those with probable TBI in the RAND assessment, the majority (57%) had not been evaluated by a physician. Other costs—for example, days of work missed by those with continuing health issues but no confirmed diagnosis of TBI—are not captured in these estimates. These estimation challenges notwithstanding, it seems reasonable to

conclude that the costs of persistent TBI symptoms, both treated and untreated, diagnosed and undiagnosed, are of a magnitude.

**The Current Study**

Research into TBI treatment is thus still basically in its infancy; the problem TBI presents both to those afflicted and to the society at large is considerable; and treatment recommendations, until more reliable assessment tools are developed and distinctions between disorders with overlapping symptoms can be more clearly defined (if they can), are focused on addressing symptoms. It was in this context, and aware of the frequent comorbidities of PTSD and TBI and the incidence rates of TBI in a veteran population, that we sought in the present study to assess whether the resolution of PTSD symptoms reported in Church, Hawk, et al. (2013), following the use of Emotional Freedom Techniques (EFT), would correlate with a reduction in TBI symptoms. We speculated that, based on comorbidity models that hypothesize a "biological interface" between TBI and PTSD (Kennedy et al., 2007), treatment that attenuates the symptoms of one may in turn lead to reductions in symptoms of the other. Alternatively, our reanalysis could simply point to the poorly defined distinctions between PTSD and TBI symptoms. Regardless of their source—whether TBI or PTSD—the point of our inquiry was to examine how effective EFT was in reducing these symptoms: the current goal of the VA's treatment guidelines (VA Consensus Conference, 2010).

Developed by Craig (Craig & Fowlie, 1995; Craig, 2010), EFT is a brief exposure therapy with somatic and cognitive components. In a typical EFT session, the subject pairs the memory of a traumatic event (i.e., exposure) with a statement of self-acceptance (i.e., cognitive acceptance, a goal in cognitive–behavioral therapy, or CBT) while simultaneously stimulating 12

different acupressure points, or "acupoints," with the fingertips. This "setup statement" takes the following form: "Even though I experienced [name the traumatic event], I deeply and completely accept myself."

In the case of a veteran who has sustained a head injury in heavy combat and experiences PTSD afterward, the client might use the following statement: "Even though I survived the detonation of the IED and other members of my unit did not, I deeply and completely accept myself." Clients repeat a "reminder phrase" to maintain exposure while they stimulate each acupoint using fingertip tapping. In this example, the reminder phrase might be "detonation of the IED." Before and after each cycle of tapping, participants rate their emotional distress on the Subjective Units of Distress (SUD) scale, a Likert-type scale ranging from 0 (*no distress*) to 10 (*highest distress possible*). The goal of EFT is to reduce a subject's SUD to near zero, or at least significantly reduce the distress, through successive rounds of tapping. In a paper reviewing the randomized controlled trials (RCTs) and outcome studies on EFT published to date, Feinstein (2010) theorized that "(a) tapping on selected acupoints (b) during imaginal exposure, (c) quickly and permanently reduces maladaptive fear responses to traumatic memories and cues."

It should be noted that there is disagreement in the literature over how important a component of EFT the tapping of meridian points actually is. Critics such as Waite and Holder (2003) suggest that EFT owes its efficacy to the other, better established techniques on which it draws (namely, exposure and CBT). Yet Waite and Holder's study failed to use three of the seven "essential" criteria defined by the American Psychological Association (APA) Division 12 Task Force on Empirically Validated Treatments (Chambless & Hollon, 1998; for a review of the criteria, see Church, Feinstein, Palmer-Hoffman, Stein, & Tranguch, 2013) and can be interpreted either as supporting or disconfirming the role of acupoint tapping (Feinstein, 2012).

Contradicting Waite and Holder's (2003) contention is research showing that when the acupressure component of EFT is isolated, effects are found that likely contribute to reports of EFT's overall treatment efficacy. For example, McFadden et al. (2011) explored the effects of an acupressure treatment in a sample of 38 subjects with mTBI. Participants were assigned either to the experimental group, which received Jin Shin, an intervention that, like EFT, targets acupoints on the body (in Jin Shin 26 points to EFT's 12), but without the associated exposure and CBT element (i.e., the setup statement with controlled recall); or to a control group, which received treatment using pressure on the body in places not considered acupoints. Members of the experimental group were shown to increase memory function on the Digit Span Test and to display significantly better working memory, as measured on the Stroop Task. Moreover, acupressure has been found to produce the same benefits as acunpuncture's needling (Cherkin et al., 2009), for which the World Health Organization (2003) reports there is good evidence in effectively treating some two dozen medical and psychological conditions. Activation, with needling, of the Large Intestine 4 acupoint on the hand, produced notable decreases in fMRI activity in the hippocampus, amygdala, and other areas of the brain associated with fear and pain (Hui et al., 2000).

*EFT and PTSD*

EFT has been shown to meet the APA Division 12 criteria for empirically supported treatments (Chambless & Hollon, 1998) as a "well-established treatment" for phobias, anxiety, depression, and PTSD (Church, Feinstein, et al., 2013), but for present purposes, we focus mainly on the research into PTSD. In an EFT intervention with 77 male Haitian counselors caring for orphans in the aftermath of the 2010 earthquake, Gurret, Caufour, Palmer-Hoffman, and Church (2012) found that the percentage of those meeting criteria for PTSD (as screened on

the PTSD Checklist, or PCL) was significantly reduced (from 62% to 0%, $p < .001$). In a randomized controlled pilot study, Church, Piña, Reategui, and Brooks (2012) tested EFT in a group of boys ($N = 16$) living in a residential treatment facility for children with a history of sexual, physical, or psychological abuse. PTSD was measured with the PTSD components of the Impact of Events Scale (IES). Again, PTSD scores declined so significantly for the treatment group ($p < .001$; as compared with a wait-list control) that no participant who had received EFT had PTSD scores in the clinical range 30 days after treatment.

Importantly, these findings have been replicated for samples of veterans suffering from PTSD. In one pilot EFT intervention, Church, Geronilla, and Dinter (2009) delivered six sessions of EFT focusing on combat and other traumatic memories to seven veterans (four of whom had served in the Iraq War, two in Vietnam, and one who experienced PTSD following sexual assault). At completion of these sessions, severity of symptoms had decreased by 46% ($p < .001$) and PTSD scores had decreased by 50% ($p < .016$). Gains were maintained when assessed 3 months later. Church (2010) included both veterans and their family members ($N = 11$, 9 of whom had been diagnosed with PTSD, 2 of whom exhibited symptoms of PTSD) in a 5-day EFT intervention delivering 10–15 hr of therapy in total. Participants' scores on the military version of the PCL (PCL–M) were significantly reduced ($p < .01$) following completion of EFT; these improvements held at 1-month, 3-month, and 1-year follow-ups.

Church and Brooks (2012) expanded this work in a sample of 109 male veterans and their spouses (Total $N = 218$). Prior to the intervention, 83% of the veterans and 29% of spouses met clinical criteria for PTSD on the PCL. Following a weeklong retreat that included both group sessions and individual therapy, only 28% of veterans and 4% of spouses still met criteria for PTSD. These gains were maintained for the veterans at a 4- to 6-week follow-up and dropped

even further for spouses ($p < .003$).

These promising results with veterans with PTSD were corroborated in an RCT conducted by Church, Hawk, et al. (2013), from which the data for the present study were extracted. An EFT treatment group ($n = 29$) was compared with a wait-list control ($n = 30$). PTSD symptoms were measured using the PCL–M. The breadth and severity of psychological symptoms such as anxiety and depression were measured on the Symptom Assessment 45, and all participants were found to be in the clinical range. After six hour-long EFT sessions, the treatment group showed significant reductions on the mean PCL–M score ($p < .0001$) while the control group remained nearly unchanged. The breadth and severity of psychological distress was also significantly reduced in the treatment group ($ps < .0001$) while remaining unchanged in the control group. The Church, Hawk, et al. (2013), study produced data on various facets of EFT treatment, which is being analyzed over time, including in the present paper.

*EFT and TBI*

In contrast to the accumulating evidence for EFT as a well-established treatment for PTSD, little is known of the intervention's effects on TBI or on comorbid TBI and PTSD. In a single-subject case study, Craig, Bach, Groesbeck, and Benor (2009) examined a subject who had sustained a TBI in a severe auto accident 10 years prior. The subject's medical records indicated a left frontal subdural hematoma on admission to hospital after the accident. The subject also suffered multiple fractures and was observed to have high anxiety, requiring restraint in order to effect treatment. The subject was eventually discharged, ambulatory with the aid of a rolling walker, but continued to experience problems with attention, as well as mild to moderate deficits of verbal and written expression, reading comprehension, attention, memory, reasoning, and problem

solving.

When the subject presented for EFT treatment approximately 10 years after the accident, she walked with the aid of a cane and reported suffering from panic attacks, vertigo, cognitive impairment, and balance problems. Prior to treatment, the subject was assessed with EEG and demonstrated a marked amplitude of high-frequency beta waves (24 to 38 Hz), consistent with anxiety. Following treatment, the amplitude of high-frequency beta was reduced, while an increase in the alpha and theta bands was observed (4 to 13 Hz). The subject reported marked improvements in cognitive and physiological functioning subsequent to treatment, including being able to walk without a cane, and reported that these were sustained at 1-year follow-up.

This case study highlights a feature of EFT that mitigates the necessity to distinguish PTSD from mTBI: a focus on symptoms rather than diagnoses. Clinical EFT is client-centered. It asks participants to list their symptoms and rate their severity before and after treatment. If self-assessed symptoms improve, treatment is considered successful. EFT dispenses with diagnosis in favor of client-assessed ratings of symptom severity. Thus, regardless of whether the diagnosis is mTBI or PTSD, or to what degree symptoms of the two overlap, treatment success is predicated on whether the client reports improvement of symptoms. This client-centered approach avoids the pitfalls that often hamper diagnosis. Given the VA Consensus Conference's (2010) recommendation that treatments for mTBI be symptom-based, this seems all the more appropriate an approach to intervention.

## Method

### *Participants*

Participant recruitment, assessment, and treatment are fully described in Church, Hawk, et al. (2013). The sample ($N = 59$) was recruited through referrals by VA clinicians and through social networking, both online and through veterans groups. To be included in the study, participants had to show PTSD symptoms in the clinical range, as evidenced by the PCL–M (score of >49; National Center for PTSD, 2008). The only exclusion criterion was a score of 4 or higher on two questions of the Symptom Assessment 45, on the basis of risk of violence. In addition, all participants had to be under the care of a clinician from a VA or other licensed health care facility. The EFT intervention was delivered as a complementary and supporting supplement to the standard of care. We did not track type and frequency of standard of care, because standardization across a variety of treatments and facilities would have been difficult and because we felt that this reporting requirement would present a burden on a subject population with already low treatment completion rates (e.g., Seal et al., 2010, found that only 30% of VA patients completed a recommended treatment program within a year of PTSD diagnosis).

A total of 149 veterans were initially contacted for participation in this study. Following initial contact and eligibility screening, 74 declined to participate and 16 did not meet inclusion criteria. Using permuted block randomization, we assigned the remaining 59 participants to either an EFT group ($n = 30$) or to a standard of care wait-list control (SOC/EL; $n = 29$). Subjects provided informed consent, and the study was approved for human subject protection by the Copernicus institutional review board and was posted on ClinicalTrials.gov (NCT00743041).

Participants were an average of 52 years old (range = 24–86), and the vast majority were male ($n = 53$). They had been deployed in a range of operations. One subject in the EFT group

dropped out after 3 sessions, and four subjects in the control group dropped out before the second session. Following the complete six coaching sessions, 29 in the EFT group and 20 in the SOC/WL groups completed assessments. At the 3-month follow-up, data were obtained for 25 in the EFT group and 17 in the SOC/WL group; at 6 months, the numbers were 26 and 13, respectively. Those subjects lost to attrition cited an uncomfortable level of emotion during memory recall, the burden of filling out forms, such as the PCL–M, and a lack of time as their reasons for not completing the study.

*Measures*

As described in Church, Hawk, et al. (2013), participants completed assessments at baseline; after three sessions of the intervention; after six sessions of EFT coaching, at treatment completion; and at 3- and 6-month follow-ups. The SOC/WL group completed the assessments following the 30-day waiting period. To maximize participant compliance, this study was designed to capture multiple symptom domains on a one-page form. These included demographic characteristics; somatoform disorders; pain; lifestyle choices; alcohol, cigarette, recreational drug, and prescription drug use; and TBI symptoms. In Church, Hawk, et al., we were focused on PTSD symptoms. These were assessed with the Global Severity Index and the Positive Symptom Total on the Symptom Assessment–45 (SA-45; Davison et al., 1997; Maruish, 1999), measuring symptom severity and breadth, respectively. Participants also completed the PCL–M self-assessment (Weathers et al., 1993), which assesses 17 items that correspond with the PTSD diagnostic criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994). The SA-45 was used as a screening tool for the study, and it and the PCL–M were used at assessment points throughout the study specifically to

measure PTSD symptoms. Findings have already been summarized, and complete results are available in Church, Hawk, et al. (2013).

In the present study, we were specifically interested in the effect of EFT on TBI and physical symptoms. At the time the study began, however, there were no generally accepted brief TBI screens (GAO, 2008; Legome, 2006). Given these constraints on data collection, and the absence of reliable and valid assessments, we therefore selected 9 items from the Patient Health Questionnaire somatoform module of the Primary Care Evaluation of Mental Disorders (PRIME-MD; Spitzer et al., 1999; see Appendix A), a self-report inventory used to screen for somatoform disorders and which has been found to have good reliability and validity. We also compiled a list of 17 symptoms commonly associated with TBI in the literature (Lawler & Terrigno, 1996; Nolin, Villemure, & Heroux, 2006; Schwarzbold et al., 2008; VHA, 2007; see Appendix B). Participants rated how often they were bothered in the 4 weeks preceding the survey by these 9 somatoform symptoms. They also rated whether they had ever had the 17 specific symptoms that were suggestive of TBI. Respondents rated each item on a 3-point scale (*never, occasionally,* or *frequently*). Data from the Somatization subscale of the SA-45 were used to capture the incidence and severity of nonspecific somatic complaints.

*Procedure*

As described in Church (2012), EFT was introduced to participants as peer-to-peer coaching rather than as therapy. The purpose of this was to minimize the power differential between practitioner and client and to support the therapeutic alliance with the participant's primary caregiver. Participants remained under the care of their primary care provider during the study. EFT coaching was intended as a supplement to treatment as usual.

The EFT intervention was delivered with fidelity to the EFT manual (Craig, 2010) in a structured format first tested in a pilot study (Church et al., 2009). The first author, a certified EFT practitioner, led the investigation; all 15 coaches were also certified in EFT and coached between 1 and 12 veterans ($M$ = 4). Five of the coaches were licensed mental health practitioners, three had professional counseling license, and two were working toward completing their license at the time the study began. No significant differences between licensed and unlicensed practitioners were found on any of the background characteristics.

Practitioner compliance was monitored by investigators through detailed session reports. Six hour-long sessions of EFT were held between coaches and participants, either in the coach's office or by telephone. During each session, coaches and participants created lists of traumatic events and then self-rated their level of emotional distress on the SUDs (ranging from 0 = *no distress* to 10 = *highest distress possible*). Participants self-applied EFT until SUDs approached zero. Participants used this method to work through each of the traumatic memories they had listed at the outset. Participants were encouraged to use the EFT method in between sessions as well.

## Results

### *Comparison of SOC/WL vs. EFT Group Pre- and Posttreatment*

*Analyses*. Linear mixed-effects models were conducted on the Somatization total score, the TBI total score, and total symptom score with patient-specific intercepts modeled over time. Independent variables were group, time (SOC/WL: pretreatment, 30-day wait assessment; EFT: pretreatment, after six sessions), and Group × Time (see Table 1). Given the significant difference between groups for insomnia, cigarette smoking, and number of prescription medications, the correlations between these variables and the somatization, TBI, and total score

variables were examined. Significant correlations ($p < .041$) were found between cigarette smoking and somatization ($r = -.299$), TBI total ($r = -.279$), and the total score ($r = -.334$). Therefore, we controlled for cigarette smoking in subsequent analyses.

We also found a significant difference between groups on the number of days between the two assessment points, $t(33.3) = -5.93$, $p < .001$ (SOC/EL: $M \pm SD = 28.8 \pm 7.4$; EFT: $M \pm SD = 58.6 \pm 25.8$). Thus, all analyses controlled for time between initial assessment and follow-up. To adjust for three planned pairwise comparisons (SOC/WL vs. pretreatment vs. posttreatment; EFT pretreatment vs. posttreatment; SOC/WL posttreatment vs. EFT posttreatment) in models with significant Group × Time interactions, we used an adjusted alpha level of $p < .017$.

*Findings*. The Group × Time interaction was significant ($p < .006$) in all of the models (see Table 2). Both the SOC/WL versus EFT posttest comparisons and EFT pretreatment to posttreatment comparisons were significant in all models. At posttest, the EFT group had fewer symptoms than the SOC/WL group. In addition, symptoms in the EFT group decreased significantly from pre- to posttest. The SOC/WL group remained unchanged.

*EFT Treatment Combined SOC/WL and EFT Groups: Change Over Time*

*Analyses.* Linear mixed-effects models were conducted on the Somatization total score, the TBI total score, and total symptom score with patient-specific intercepts modeled over time periods (pretreatment, after three sessions, after six sessions, at 3-month follow-up, and at 6-month follow-up). Time between sequential assessments was controlled for in the model to adjust for the possible effect of time that may have resulted from the intervention delay in the SOC/WL group. Group and the interaction between group and time period were also included in

the model; the purpose of this was to identify any changes in outcome that may have resulted from the delayed intervention in the SOC/WL group. To adjust for ten planned pairwise comparisons between time points in models with time effects, we used an adjusted alpha level of $p < .005$.

*Findings*. The results of the EFT change analyses are presented in Table 3. There was a significant main effect for time ($p < .0006$) in all of the models. Group and the Group × Time interaction were nonsignificant in all models. Symptoms significantly decreased between the pretest and Session 6 assessments in all three models ($p < .0001$). There was also a significant decrease in symptoms between the Session 3 and Session 6 assessments for all models ($p < .0027$). For the TBI total model, no other comparisons were significant, indicating that improvement from pretest occurred during the EFT treatment period but remained stable in the follow-up periods. For the Somatization and Symptom total models, a significant decrease in symptoms occurred between the pretest and both of the follow-ups (3 and 6 months), indicating continuing improvement on symptoms ($p < .002$). In addition, the 3-month follow-up was also significantly lower for the Somatization model ($p < .005$). No adverse events were reported.

**Discussion**

The present study offers initial, tentative evidence for the potential remediation of TBI symptoms in a population co-presenting with clinical PTSD. In the preceding analysis of the same sample (Church, Hawk, et al., 2013), the EFT group showed significant reductions on measures of PTSD (PCL–M scores) as well as in the breadth and severity of psychological distress (all $p$s $< .0001$); their PTSD scores remained subclinical at the 3-month and 6-month follow-ups. Conversely, the wait-list control group remained unchanged. In the current study, TBI and somatoform symptoms were isolated out of the data set for analysis. We found that symptoms were

significantly reduced after three EFT sessions, and further reductions were shown after six sessions ($p < .0021$). Moreover, gains in symptom totals were maintained at both 3-month and 6-month follow-ups ($p < .0006$). Again, the wait-list control remained unchanged.

There are a number of clinical implications to the present findings. The association between TBI and a broad spectrum of long-term psychiatric, neurologic, and psychosocial morbidities makes the rapid and sustained improvements in TBI symptoms in the current study noteworthy. These morbidities include an increased likelihood of depression and postconcussion symptoms; peripheral visual and tandem gait impairment; and increased likelihood of marital problems, disability, underemployment, and low income (Vanderploeg, Curtiss, Luis, & Salazar, 2007). In a population-based follow-up study in persons with mTBI, Stålnacke (2007) found that, 3 years after the sustained head injury, a majority were still experiencing postconcussion symptoms (such as headache, fatigue, and memory loss) and exhibiting signs of posttraumatic stress (as measured on the IES). As a counterpoint to this, in our sample, both total somatoform symptoms and TBI symptoms were significantly reduced following six 1-hr sessions of EFT, and gains were maintained at both 3-month and 6-month follow-up, indicating durable improvement.

Girard (2007) found that the use of telemedicine can be critical for the identification and treatment of TBI in the military and the VA. Particularly after veterans have returned home from deployment and in some cases settled in rural regions where availability and accessibility of medical facilities limits their treatment options, telemedicine offers the potential to redress gaps in services. With its potential for increased privacy, telemedicine may also be able to reach the segment of the military population that perceives a stigma associated with PTSD and TBI and is therefore reluctant to seek treatment (see, e.g., Miller & Zwerdling, 2010). Hartung and Stein (2012) compared the efficacy of in-person office EFT with telephone-delivered EFT in this same

sample and found that 67% of those treated by phone no longer met clinical criteria for PTSD following the intervention. Although this was of lower efficacy than the office intervention, improvements were still significant ($p < .05$), and for situations in which access to in-person treatment is constrained, EFT delivered via telemedicine is a viable alternative.

There are also questions of cost—personal, societal, and economic—that make EFT a viable treatment option. As summarized in Church and Brooks (2012), the long-term effects of PTSD include an association with increased levels of domestic violence, overrepresentation in the prison population, increased risk of suicide, and a deleterious effect on parenting and marital skills. Vanderploeg et al. (2007) found similar associations of mTBI with marital problems, disability, underemployment, and low income. Kanter (2007) estimated the lifetime cost of treating a single veteran diagnosed with PTSD as over $1,400,000; in the same year, RAND estimated the total costs of treating TBI in the veteran population in only the first year after diagnosis to between $591 and $910 million. Contrast these figures with the relative cost of six 1-hr treatments in which a patient uses EFT with minimal equipment or space requirements: EFT requires no residential facility, nor do patients generally undergo lengthy in-house stays. For that same $1,400,000 estimated by Kanter for treating a single veteran with PTSD, 2,000 veterans could receive six EFT sessions.

There are limitations to the methodology of our study that both impair generalizability and raise a number of questions that should be explored in future research. The lack of a validated screening instrument for TBI limits the generalizability of both this study and many others. When we began the study, no generally accepted brief TBI screens existed (GAO, 2008; Legome, 2006). The VA Consensus Conference (2010) recommended that treatments for mTBI be focused on symptoms, and indeed we chose measures that would screen for both somatoform

and TBI symptoms, based on the PHQ Somatoform Module of the Prime MD-PHQ and the literature on TBI. We did not screen for confirmed diagnoses of TBI, and the gains we report are for reductions in symptom totals rather than for specific symptoms that may be particularly problematic in the aftermath of TBI (e.g., memory loss). Nor did we collect data on the length of time that had passed since the TBI might have been sustained or on how long respondents had been experiencing their reported symptoms. Rather, respondents reported the frequency of somatoform symptoms only for the 4 weeks prior to the survey and their more general experience of symptoms often associated with TBI within an open time frame.

Several of the limitations that are discussed in Church, Hawk, et al. (2013), also apply here: Although research is limited on the efficacy of TBI treatments, thus making it difficult to design an RCT in which the EFT group is compared with an active intervention group rather than a wait-list control, there are treatments known to be efficacious for the symptoms under focus. If remaining symptom focused, future research could design comparisons between these treatments and EFT. Without tracking the standard of care participants were receiving in their primary VA treatment program, we are unable to definitively claim that the improvement observed in the EFT versus SOC/WL groups is solely attributable to EFT. Sympathetic attention in the therapeutic relationship could also account for a portion of the observed positive effect of EFT.

It would be worth designing future studies that take these variables into account—for example, testing the efficacy of EFT in a sample in which a TBI is known to have been sustained, comparing the intervention's effects in groups with mild versus moderate or severe TBI, and studying its impact in veterans with symptoms that persist years after injury. These caveats aside, there are numerous factors that complicate diagnosis of TBI, as described earlier. Particularly for those veterans whose discharge from the military preceded the VA's institution

of the TBI Clinical Reminder screening, our screening measures had the potential to capture a broader swathe of those possibly affected by TBI regardless of their self-identification or confirmed diagnosis of the injury.

One theoretical limitation of EFT has been due to the inability to isolate its acupoint tapping element from its cognitive and exposure element. Waite and Holder (2003) argued that it may be due to these elements from established therapies that EFT is effective. A recent dismantling study provided evidence that acupoint stimulation is an active ingredient rather than a placebo (Fox, 2013). It compared EFT to an active control condition (breathing and mindfulness). Other than tapping, the two conditions were as similar as possible. The study found that the EFT group showed significant emotional improvement over the control group. EFT shares other theoretical limitations with acupuncture; though several studies have found physiological correlates of acupuncture meridians, no theory about their mechanisms of action has yet achieved a consensus view in the scientific community.

Finally, our research also points to the challenges that exist in disentangling TBI symptoms from those associated with PTSD. As explored in the introduction, there is thought to be frequent comorbidity between PTSD and TBI, but prevalence rates become obscured by the considerable overlap in their symptomatology and disagreement over the attribution of symptoms to each condition. To qualify for the study, participants had to meet the clinical criterion for PTSD symptoms as measured by the PCL-M; they did not have to have a confirmed TBI. Some of the symptoms captured on our measures, such as “feeling your heart pound or race,” shortness of breath, and high blood pressure, could be evidence of the anxiety those with PTSD observe while reexperiencing a past trauma or exhibiting persistent arousal, including an exaggerated startle response. Others, such as problems reading or writing, could be indicative of

the concentration difficulties associated with PTSD-induced arousal. We cannot say conclusively that the symptoms the EFT intervention targeted, and effectively reduced, were the by-products strictly of PTSD, of TBI, of both, or of an entirely separate condition (e.g., depression has many physical manifestations as well).

Although we cannot be sure which elements of each disorder were resolved by EFT, the data indicate that EFT effectively attenuates this matrix of symptoms. Given the complex, and complexity, of symptoms associated with these conditions, the symptom-oriented approach now advocated by the VA might hold higher clinical utility than attempts to further parse diagnoses. However, Miller and Zwerdling's (2010) description of the separate centers being built for treatment of TBI and PTSD at Fort Bliss, the third largest base in the U.S. military, suggests that a symptom-oriented approach is far from universally accepted.

As a counterpoint, Jonas et al. (2011) speculated that "the effects of brain injury are approached better by assessing the full spectrum of trauma-related morbidities—rather than dividing them into subcomponents—and then treating the whole person with an approach that enhances the patient's inherent healing mechanisms and capacities" (p. 250). TBI and PTSD often occur in tandem; to treat them separately as isolated conditions may slow progress toward alleviating symptoms—a primary clinical goal. The ability of EFT to offer at least partial rehabilitation of TBI while effectively treating PTSD symptoms makes it worthy of consideration as an adjunctive intervention for veterans—and quite possibly as a primary one.

**References**

American Congress of Rehabilitation Medicine. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8,* 86–87.

Australian Centre for Posttraumatic Mental Health. (2007). *Australian guidelines for the treatment of adults with acute stress disorder and posttraumatic stress disorder*. Melbourne, Australia: Author.

Bogdanova, Y., & Verfaellie, M. (2012). Cognitive sequelae of blast-induced traumatic brain injury: Recovery and rehabilitation. *Neuropsychology Review, 22,* 4–20.

Brewin, C. R. (2005). Encoding and retrieval of traumatic memories. In C. R. Brewin (Ed.), *Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives* (pp. 131–150). New York, NY: Guilford Press.

Brewin, C. R. (2007). What is it that a neurobiological model of PTSD must explain? *Progress in Brain Research, 167,* 217–226.

Bryant, R. (2011). Post-traumatic stress disorder vs. traumatic brain injury. *Dialogues in Clinical Neuroscience, 13,* 251–262.

Carlson, K., Kehle, S., Meis, L., Greer, N., MacDonald, R., Rutks, I., & Wilt, T. J. (2009, August). *The assessment and treatment of individuals with history of traumatic brain injury and post-traumatic stress disorder: A systematic review of the evidence*. Washington, DC: Department of Veterans Affairs.

Carlson, K. F., Kehle, S., Meis, L. A., Greer, N., MacDonald, R., Rutks, I., . . . Wilt, T. J. (2011). Prevalence, assessment, and treatment of mild traumatic brain injury and posttraumatic stress disorder: A systematic review of the evidence. *Journal of Head Trauma Rehabilitation, 26,* 103–115.

Carroll, L. J., Cassidy, J. D., Holm, L., Kraus, J., & Coronado, V. G. (2004). Methodological

issues and research recommendations for mild traumatic brain injury: The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitative Medicine, 43*(Suppl.), 113–125.

Chambless, D., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology, 66,* 7–18.

Cherkin, D. C., Sherman, K. J., Avins, A. L., Erro, J. H., Ichikawa, L., Barlow, W. E., … Deyo, R. A. (2009). A randomized trial comparing acupuncture, simulated acupuncture, and usual care for chronic low back pain. *Archives of Internal Medicine, 169,* 858–866.

Church, D. (2010). The treatment of combat trauma in veterans using EFT (Emotional Freedom Techniques): A pilot protocol. *Traumatology, 16,* 55–65.

Church, D. (2012, June). *Reductions in pain, depression, and anxiety symptoms after PTSD remediation in veterans*. Presented at the Association for Comprehensive Energy Psychology conference, San Diego.

Church, D., & Brooks, A. (2012, October). *CAM and energy psychology techniques remediate PTSD symptoms in veterans and spouses*. Data presented at the Omega Institute conference "Veterans, Treatment, & Trauma" October.

Church, D., Geronilla, L., & Dinter, I. (2009). Psychological symptom change in veterans after six sessions of EFT (Emotional Freedom Techniques): An observational study. *International Journal of Healing and Caring, 9*(1), 1–12.

Church, D., Hawk, C., Brooks, A., Toukolehto, O., Wren, M., Dinter, I., & Stein, P. (2013). Psychological trauma symptom improvement in veterans using EFT (Emotional Freedom Techniques): A randomized controlled trial. *Journal of Nervous and Mental Disease, 201,* 153–160.

Church, D., Feinstein, D., Palmer-Hoffman, J., Stein, P. K., & Tranguch, A. (2013). *Empirically supported psychological treatments: The challenge of evaluating clinical innovations.* Manuscript under review.

Church, D., Piña, O., Reategui, C., & Brooks, A. (2012). Single-session reduction of the intensity of traumatic memories in abused adolescents after EFT: A randomized controlled pilot study. *Traumatology, 18,* 73–79.

Craig, D. (2010). *The EFT manual* (2nd ed.). Santa Rosa, CA: Energy Psychology Press.

Craig, G., Bach, D., Groesbeck, G., & Benor, D. J. (2009). Emotional Freedom Techniques (EFT) for traumatic brain injury. *International Journal of Healing and Caring, 9*(2), 1–12.

Craig, G., & Fowlie, A. (1995). *The EFT manual.* Sea Ranch, CA: Author.

Defense and Veterans Brain Injury Center Working Group on the Acute Management of Mild Traumatic Brain Injury in Military Operational Settings. (2006, December 26). *Clinical practice guidelines and recommendations*. Author.

Diepold, J. H., & Goldstein, D. (2009). Thought field therapy and QEEG changes in the treatment of trauma: A case study. *Traumatology, 15,* 85–93.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behavior Research and Therapy, 38,* 319–345.

Elder, G. A., Dorr, N. P., De Gasperi, R., Gama Sosa, M. A., Shaughness, M. C., Maudlin-Jeronimo, E., . . . Ahlers, S. T. (2012). Blast exposure induces post-traumatic stress disorder-related traits in a rat model of mild traumatic brain injury. *Journal of Neurotrauma, 29,* 2564–2575.

Fang, J., Jin, Z., Wang, Y., Li, K., Kong , J., Nixon , E. E., . . . Hui K. K.-S. (2009). The salient characteristics of the central effects of acupuncture needling: Limbic-paralimbic-neocortical network modulation. *Human Brain Mapping, 30,* 1196–1206.

Feinstein, D. (2010). Rapid treatment of PTSD: Why psychological exposure with acupoint tapping may be effective. *Psychotherapy: Theory, Research, Practice, Training, 47,* 385–402.

Feinstein, D. (2012). Acupoint stimulation in treating psychological disorders: Evidence of efficacy. *Review of General Psychology, 16*, 364–380. doi:10.1037/a0028602

Foa, E. B., Keane, T. M., & Friedman, M. J. (Eds.). (2000). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress studies*. New York, NY: Guilford Press.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (Eds.). (2009). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed.). New York, NY: Guilford Press.

Foa, E. B., Gillihan, S. J., & Bryant, R. B. (in press). Challenges and successes in dissemination of evidence-based treatments for posttraumatic stress: Lessons learned from prolonged exposure therapy for PTSD. *Psychological Science in the Public Interest.*

Fox, L. (2013). Is acupoint tapping an active ingredient or an inert placebo in Emotional Freedom Techniques (EFT)? A randomized controlled dismantling study. *Energy Psychology: Theory, Research, and Treatment 5*(2), 15-28.

Girard, P. (2007). Military and VA telemedicine systems for patients with traumatic brain injury. *Journal of Rehabilitation Research & Development, 44,* 1017–1026.

Gironda, R. J., Clark, M. E., Ruff, R. L., Chait, S., Craine, M., Walker, R., & Scholten, J. (2009). Traumatic brain injury, polytrauma, and pain: Challenges and treatment strategies for the Polytrauma Rehabilitation Network. *Rehabilitation Psychology, 54,* 247–258.

Gurret, J.-M., Caufour, C., Palmer-Hoffman, J., & Church, D. (2012). Post-earthquake rehabilitation of clinical PTSD in Haitian seminarians. *Energy Psychology, 4*(2).

Hartung, J., & Stein, P. K. (2012). Telephone delivery of EFT (Emotional Freedom Techniques) remediates PTSD symptoms in veterans. *Energy Psychology, 4*(1).

Harvey, A. G., Kopelman, M. D., & Brewin, C. R. (2005). PTSD and traumatic brain injury. In J. J. Vasterling & C. R. Brewin (Eds.), *Neuropsychology of PTSD: Biological, cognitive and clinical perspectives* (pp. 230–246). New York, NY: Guilford Press.

Hoge, C. W., McGurk, D., Thomas, J. L., Cox, A. L., Engel, C. C., & Castro, C. A. (2008). Mild traumatic brain injury in U.S. soldiers returning from Iraq. *New England Journal of Medicine, 358,* 453–463.

Hui, K. K.-S., Liu, J., Makris, N., Gollub, R. W., Chen, A. J. W., Moore, C. I., . . . Kwong, K. K. (2000). Acupuncture modulates the limbic system and subcortical gray structures of the human brain: Evidence from fMRI studies in normal subjects. *Human Brain Mapping, 9,* 13–25.

Institute of Medicine. (2007). *Treatment of posttraumatic stress disorder: An assessment of the evidence*. Washington, DC: National Academies Press.

Jonas, W. B., Walter, J. A. G., Fritts, M., & Niemtzow, R. C. (2011). Acupuncture for the trauma spectrum response: Scientific foundations, challenges to implementation. *Medical Acupuncture, 23,* 249–262.

Kanter, E. (2007). *Shock and awe hits home.* Washington, DC: Physicians for Social Responsibility.

Kennedy, J. E., Jaffee, M. S., Leskin, G. A., Stokes, J. W., Leal, F. O., & Fitzpatrick, P. J. (2007). Posttraumatic stress disorder and posttraumatic stress disorder-like symptoms and mild traumatic brain injury. *Journal of Rehabilitation Research and Development, 44,* 895–920.

Legome, E., & Wu, T. (2006, September 6). Postconcussive syndrome. *eMedicine: Emergency Medicine, Trauma and Orthopedics*.

Lew, H. L., Otis, J. D., Tun, C., Kerns, R. D., Clark, M. E., & Cifu, D. X. (2009). Prevalence of chronic pain, posttraumatic stress disorder, and persistent postconcussive symptoms in OIF/OEF veterans: Polytrauma clinical triad. *Journal of Rehabilitation Research and Development, 46,* 697–702.

McCrea, M., Pliskin, N., Barth, J., Cox, D., Fink, J., French, L., . . . Yoash-Gantz, R. (2008). Official position of the Military Task Force on the Role of Neuropsychology and Rehabilitation Psychology in the Evaluation, Management, and Research of Military Veterans with Traumatic Brain Injury. *The Clinical Neuropsychologist, 22,* 10–26.

McFadden, K. L., Healy, K. M., Dettmann, M. L., Kaye, J. T., Ito, T. A., & Hernández, T. D. (2011). Acupressure as a non-pharmacological intervention for traumatic brain injury (TBI). *Journal of Neurotrauma, 28,* 21. doi: 10.1089/neu.2010.1515

Miller, T. C., & Zwerdling, D. (2010, June 8). At Fort Bliss, brain injury treatments can be as elusive as diagnosis. *ProPublica and NPR*. Retrieved from http://www.propublica.org/features/at-fort-bliss-brain-injury-treatments-can-be-as-elusive-as-diagnosis

National Center for PTSD. (2008). *PTSD Checklist*. Retrieved from www.ncptsd.va.gov/ncmain/ncdocs/assmnts/ptsd_checklist_pcl.html

National Collaborating Centre for Mental Health. (2005). *Post-traumatic stress disorder (PTSD): The management of PTSD in adults and children in primary and secondary care*. London, England: National Institute for Clinical Excellence.

Nolin, P., Villemure, H., & Heroux, L. (2006). Determining long-term symptoms following mild traumatic brain injury: Method of interview affects self-report. *Brain Injury, 20,* 1147–1154.

Okie, S. (2005). Traumatic brain injury in the war zone. *New England Journal of Medicine, 352,* 2043–2047.

Polusny, M. A., Kehle, S. M., Nelson, N. W., Erbes, C. R., Arbisi, P. A., Thuras, P. (2011). Longitudinal effects of mild traumatic brain injury and posttraumatic stress disorder comorbidity on postdeployment outcomes in National Guard soldiers deployed to Iraq. *Archives of General Psychiatry, 68,* 79–89.

Rao, V., & Lyketsos, C. G. (2002). Psychiatric aspects of traumatic brain injury. *Psychiatric Clinics of North America, 25,* 43–69.

Report of (VA) Consensus Conference. (2010). *Practice recommendations for treatment of veterans with comorbid TBI, pain, and PTSD.* Retrieved from http://www.ptsd.va.gov/professional/pages/handouts-pdf/TBI_PTSD_Pain_Practice_Recommend.pdf

Rona, R. J. (2012). Long-term consequences of mild traumatic brain injury. *British Journal of Psychiatry, 201,* 172–174.

Ruff, R. L., Riechers, R. G., & Ruff, S. S. (2010). Relationships between mild traumatic brain injury sustained in combat and post-traumatic stress disorder. *F1000 Medicine Reports, 2,* 64. doi: 10.3410/M2-64

Ruff, R. L., Riechers, R. G., II, Wang, X.-F., Piero, T, & Ruff, S. S. (2012). A case-control study examining whether neurological deficits and PTSD in combat veterans are related to episodes of mild TBI. *BMJ Open, 2,* e000312. doi: 10.1136/bmjopen-2011-000312

Sammons, M. T., & Batten, S. V. (2008). Psychological services for returning veterans and their families: Evolving conceptualizations of the sequelae of war-zone experiences. *Journal of Clinical Psychology, 64,* 921–927.

Schneiderman, A., Braver, E. R., & Kang, H. K. (2008). Understanding sequelae of injury mechanisms and mTBI incurred during the conflicts in Iraq and Afghanistan: Persistent postconcussive symptoms and PTSD. *American Journal of Epidemiology, 167,* 1446–1452.

Schwarzbold, M., Diaz, A., Martins, E. T., Rufino, A., Amante, L. N., Thais, M. E., . . . Walz, R. (2008). Psychiatric disorders and traumatic brain injury. *Neuropsychiatric Disease and Treatment, 4,* 797–816.

Seal, K. H., Maguen, S., Cohen, B., Gima, K. S., Metzler, T. J., Ren, L., … Marmar, C. R. (2010). VA mental health services utilization in Iraq and Afghanistan veterans in the first year of receiving new mental health diagnoses. *Journal of Traumatic Stress, 23,* 5–16.

Spitzer, R. L., Kroenke, K., & Williams, J. B. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary study. Primary Care Evaluation of Mental

Disorders. Patient Health Questionnaire. *Journal of the American Medical Association, 282,* 1737–1744.

Stålnacke, B.-M. (2007). Community integration, social support and life satisfaction in relation to symptoms 3 years after mild traumatic brain injury. *Brain Injury, 21,* 933–942.

Swingle, P. (2010). Emotion Freedom Techniques (EFT) as an effective adjunctive treatment in the neurotherapeutic treatment of seizure disorders. *Energy Psychology, 2*(1), 27–37.

Swingle, P., Pulos, L., & Swingle, M. K. (2004). Neurophysiological indicators of successful EFT treatment of post traumatic stress. *Subtle Energies & Energy Medicine, 15,* 75–86.

Tanielian, T., & Jaycox, L. H. (Eds.). (2008). *Invisible wounds of war: Psychological and cognitive injuries, their consequences, and services to recovery.* Santa Monica, CA: RAND.

Terrio, H., Brenner, L. A., Ivins, B. J., Cho, J. M., Helmick, K., Schwab, K., . . . Warden, D. (2009). Mild traumatic brain injury screening: Preliminary findings in a US Army Brigade Combat Team. *Journal of Head Trauma Rehabilitation, 24,* 14–23.

Theeler, B. J., Flynn, F. G., & Erickson, J. C. (2010). Headaches after concussion in US soldiers returning from Iraq or Afghanistan. *Headache, 50,* 1262–1272.

U.S. Government Accountability Office. (2008, February 8). *VA health care: Mild traumatic brain injury screening and evaluation implemented for OEF/OIF veterans, but challenges remain.* Washington, DC: Author.

Vanderploeg, R. F., Curtiss, G., Luis, C. A., & Salazar, A. M. (2007). Long-term morbidities following self-reported mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 29,* 585–598.

Vanderploeg, R. D., Groer, S., & Belanger H. G. (2012). Initial developmental process of a VA semistructured clinical interview for TBI identification. *Journal of Rehabilitation Research and Development, 49,* 545–556.

Vasterling, J. J., Verfaillie, M., & Sullivan, K. D. (2009). Mild traumatic brain injury and posttraumatic stress disorder in returning veterans: Perspectives from cognitive neuroscience. *Clinical Psychology Review, 29,* 674–684.

Veterans Health Administration. (2007). *Traumatic brain injury screening* (VHA directive 2007-013). Washington, DC: Author.

Waite, W. L., & Holder, M. D. (2003). Assessment of the Emotional Freedom Technique: An alternative treatment for fear. *Scientific Review of Mental Health Practice, 2,* 20–26.

World Health Organization. (2003). Acupuncture: Review and analysis of reports on controlled clinical trials. Geneva, Switzerland: Author.

## Appendix A

Symptoms From the PHQ Somatoform Module of the Prime MD-PHQ

- Stomach pain
- Back pain
- Pain in arms, legs, joints
- Headaches
- Chest pain
- Feeling your heart pound or race
- Shortness of breath
- Constipation, loose bowels, or diarrhea
- Nausea, gas, or indigestion

## Appendix B

Specific Symptoms Often Associated With TBI

- Recurrent headaches
- High blood pressure
- Seizures
- Concussion injuries
- Head injuries
- Bleeding from nose, mouth or ears
- Dizziness
- Memory loss
- Loss of consciousness for under 30 minutes
- Loss of consciousness for over 30 minutes
- Full or partial loss of vision
- Problems reading or writing
- Decrease or loss of hearing
- Ringing in the ears (tinnitus)
- Inability to tolerate light
- Diminished sense of taste or smell
- Fainting spells

**Table 1.** Participant characteristics by group prior to intervention.

| Variable | SOC/WL ($n$ = 29) | EFT ($n$ = 30) | Total ($N$ = 59) | Statistic | $p$ |
|---|---|---|---|---|---|
| Age, *M* (*SD*) | 54.1 (11.1) | 49.4 (16.2) | 51.7 (14.0) | $t(51.6) = 1.31$ | .20 |
| Male, *n* (%) | 25 (86.2) | 28 (93.3) | 53 (89.8) | $\chi^2(1) = 0.82$ | .37 |
| Deployment | | | | | |
| Gulf War era, *n* (%) | 9 (32.1) | 15 (50.0) | 24 (41.4) | $\chi^2(1) = 1.90$ | .17 |
| Other deployments, *n* (%) | 19 (67.9) | 15 (50.0) | | | |
| Tours, *M* (*SD*) | 1.1 (0.3) | 1.2 (0.5) | 1.2 (0.4) | $t(44.3) = -1.55$ | .13 |
| PCL–M, *M* (*SD*) | 65.1 (9.3) | 62.3 (8.8) | 63.7 (9.1) | $t(57) = 1.19$ | .24 |
| Any exercise, *n* (%) | 20 (71.4) | 23 (82.1) | 43 (76.8) | $\chi^2(1) = 0.90$ | .34 |
| Any smoking, *n* (%) | 13 (46.4) | 5 (17.9) | 18 (32.1) | $\chi^2(1) = 5.24$ | .02 |
| Any alcohol, *n* (%) | 16 (57.1) | 10 (35.7) | 26 (46.4) | $\chi^2(1) = 2.59$ | .11 |
| Any drug use, *n* (%) | 3 (10.7) | 5 (17.9) | 8 (14.3) | $\chi^2(1) = 0.58$ | .45 |
| Insomnia | | | | | |
| Severe, *n* (%) | 15 (51.7) | 10 (33.3) | 25 (42.4) | $\chi^2(3) = 10.08$ | .02 |
| Moderate severe, *n* (%) | 13 (44.8) | 9 (30.0) | 22 (37.3) | | |
| Subthreshold, *n* (%) | 1 (3.4) | 10 (33.3) | 11 (18.6) | | |
| None, *n* (%) | 0 (0) | 1 (3.3) | 1 (1.7) | | |
| Treatment medications, *M* (*SD*) | 4.7 (3.9) | 1.4 (2.0) | 3.0 (3.4) | $t(36.6) = 3.79$ | .01 |

*Note*. SOC/WL = standard of care wait-list; EFT = Emotional Freedom Techniques; PCL–M = PTSD Checklist–Military.

**Table 2.** Somatization (SA-45 Subscale), TBI symptoms (Appendix B), and total symptom score (Appendix A) means and standard errors for EFT completers ($n$ = 28) at pretest and after 6 sessions and baseline and 30-day assessment for SOL/WL completers ($n$ = 24).

| | SOC/WL | | EFT | | | |
|---|---|---|---|---|---|---|
| | Pretest | 30-day[a] | Pretest[b] | 6 sessions[a,b] | | |
| Variable | *M* (*SE*) | *M* (*SE*) | *M* (*SE*) | *M* (*SE*) | *F*(1, 44) | *p* |
| Somatization total | 10.13 (0.79) | 9.43 (0.73) | 8.23 (0.81) | 4.83 (0.95) | 8.36 | .0059 |
| TBI total | 11.11 (0.97) | 10.41 (0.91) | 10.27 (1.0) | 6.09 (1.15) | 10.66 | .0021 |
| Symptom total | 21.25 (1.4) | 19.73 (1.36) | 18.67 (1.47) | 10.80 (1.66) | 19.30 | <.0001 |

[a]EFT posttest < SOC/WL posttest, $p < .004$.

[b]EFT posttest < EFT pretest, $p < .008$.

**Table 3.** Time main effects for both EFT and posttest SOC/WL combined.

| Variable | Pretest *M* (*SE*) | 3 sessions *M* (*SE*) | 6 sessions *M* (*SE*) | 3-month *M* (*SE*) | 6-month *M* (*SE*) | *F* (*df*) | *P* |
|---|---|---|---|---|---|---|---|
| Somatization total[a] | 8.76 (0.58) | 7.86 (0.54) | 5.86 (0.54) | 5.83 (0.69) | 6.214 (0.65) | 11.05 (4, 152) | <.0001 |
| TBI total[b] | 10.03 (0.70) | 9.20 (0.66) | 7.62 (0.66) | 7.88 (0.83) | 8.09 (0.80) | 5.15 (4, 151) | .0006 |
| Symptom total[c] | 18.80 (1.10) | 17.06 (1.04) | 13.48 (1.03) | 13.71 (1.29) | 14.31 (1.24) | 10.85 (4, 151) | <.0001 |

[a] Pretest > 6-session assessment, $p < .0001$; pretest > 3-month assessment, $p = .0006$; pretest > 6-month assessment, $p = .0011$; 3-session assessment > 6-session assessment, $p < .0001$; 3-session assessment > 3-month assessment, $p = .005$.

[b] Pretest > 6-session assessment, $p < .0001$; 3-session assessment > 6-session assessment, $p = .0027$.

[c] Pretest > 6-session assessment, $p < .0001$; pretest > 3-month assessment, $p = .0011$; pretest > 6-month assessment, $p = .002$; 3-session assessment > 6-session assessment, $p < .0001$.