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On the Neural Basis of EMDR Therapy: Insights From qEEG Studies

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Eye movement desensitization and reprocessing (EMDR) therapy has been shown by empirical studies to be effective in relief from psychological traumas including posttraumatic stress disorder (PTSD). Several logical concepts regarding the origin of the EMDR effect have been presented, but no detailed neural explanation is available. This lack of a widely accepted scientific explanation for the EMDR effect has led to skepticism about the therapy by many therapists and potential clients. The authors present evidence based primarily on quantitative electroencephalogram studies that the neural basis for the EMDR effect is depotentiation of fear memory synapses in the amygdala during an evoked brain state similar to that of slow wave sleep. These studies suggest that brain stimulation during EMDR significantly increases the power of a naturally occurring low-frequency rhythm in memory areas of the brain, binding these areas together and causing receptors on the synapses of fear memory traces to be disabled. This mechanical change in the memory trace enables it to be incorporated into the normal memory system without the extreme emotions previously associated with it. EMDR is a medical procedure because it changes the physical structure of the brain to modify problematically stored memories.

Keywords: EMDR therapy; memory; PTSD; EEG; neuronal response frequency

Much study has been devoted to determine the neural basis of the positive effects of eye movement desensitization and reprocessing (EMDR) therapy. Bergmann (1998) proposed that EMDR therapy with bilateral brain stimulation results in the resetting of pacemaker cells in the septum. These cells are known to pace the firing of principal cells in the greater hippocampal system (the GHS, consisting especially of the amygdala and the hippocampus proper). Resetting of the pacemaker cells to delta rhythm (from the usual waking state theta rhythm) may increase the synchronization of the hemispheres and improve functional connectivity. Bergmann (2000) also discusses evidence that EMDR stimulates the cerebellar processing center, which results in activation of the dorsolateral and orbitofrontal cortices. He believes this leads to further integration of traumatic memory into general semantic networks, as well as other neocortical areas. Corrigan (2002) suggested that the EMDR effect is centered on the anterior cingulate cortex (ACC), and he hypothesized that EMDR promotes the disconnection between the affective and cognitive subdivisions of the ACC. This disconnection would lead to relief from the affective portion of the memory. MacCulloch and Feldman (1996), Martin, Hofmann, Wizelman, and Lempa (2008), and Söndergaard and Elofsson (2008) describe beneficial changes in psychophysiological activity, as well as decreases in subjective disturbance and stress reactivity during EMDR treatment. They believe these changes lead to or accompany the reduction in posttraumatic stress disorder (PTSD) symptoms during EMDR therapy. Similarly, Barrowcliff, Gray, Freeman, and MacCulloch (2004) and Maxfield, Melnyk, and

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A list of the EEG data files is available from the corresponding author. The authors have no conflicts of interest to declare.

Part of this study was presented in poster sessions at the EMDRI annual convention in Dallas (September 2007) and the American Psychological Association annual meeting in Boston (August 2008). This research was sanctioned by institutional review board letter IRB #07-019 granted by the University of the Rockies in Colorado Springs, Colorado.

Hayman (2008) have established that eye movements during EMDR therapy, as opposed to the eye stationary condition, reduce the vividness, emotional valence, and electrodermal arousal associated with negative autobiographical memories. Propper and Christman (2008) suggest that eye movements during EMDR increase the interaction between the two brain hemispheres and that this may explain the effects on memory of EMDR treatment for PTSD.

Stickgold (2002, 2008) has postulated that the lateral eye movements characteristic of rapid eye movement (REM) sleep are an indication that EMDR may invoke conditions of the REM sleep state and suggests inducing the state during therapy (Stickgold, 2007). Bergmann (2008) notes that PTSD is characterized by a decrease in thalamic activity; this decrease is reversed during EMDR therapy. He hypothesizes that the increase in thalamic activity promotes "the repair and integration of somatosensory, memorial, cognitive, and synchronized hemispheric functions" (p. 300). Solomon and Shapiro (2008) believe a memory held in attention during EMDR is transformed and then re-stored in a less toxic form through reconsolidation of the memory.

Despite these valuable and insightful studies, no scientifically proven account of the EMDR effect has yet been given. We present here a study designed to investigate the synaptic depotentiation concept presented by Rasolkhani-Kalhorn and Harper (2006).

Memory Recording and Editing

To understand how EMDR therapy can be so effective in suppressing symptoms arising from stress caused by traumatic events, it is necessary to review the mechanics of memory recording, editing, storage, and extinction. The cognitive aspects of memories are mediated in the hippocampus, whereas emotional aspects are mediated in the amygdala (Axmacher, Haupt, Fernández, Elger, & Fell, 2008; Bergmann, 2008; Sah, Faber, Lopez de Armentia, & Power, 2003). Together, the hippocampus and the amygdala are referred to in this article as the greater hippocampal system. They are located adjacent to each other beneath the temporal lobes on each side of the brain. Memory is recorded and extinguished by potentiation and depotentiation, respectively, of synapses on neurons recruited to form the memory chain. At the molecular level within the memory areas of the brain, potentiation and depotentiation are carried out through phosphorylation and dephosphorylation of alpha-amino-3-hydroxy-5-methyl-4-isoxazole (AMPA) receptors on synapses in the GHS (Lin, Yeh, Lu, & Gean, 2003). Changes in the balance of potentiation and depotentiation are, we believe, the basis of the effect that EMDR therapy has on the neural mechanics of memory.

Memory recording occurs during the waking state. It is edited and transferred to the neocortex during slow wave sleep (SWS) and is further strengthened during REM sleep (Born, Rasch, & Gais, 2006; Gais & Born, 2004; Ribeiro et al., 2004; Tononi & Cirelli, 2003). For example, an incident such as a conversation with one's child is recorded in "real time" during the day in the hippocampus (which records details such as what is said by whom); any negative emotions accompanying the conversation are simultaneously recorded in the amygdala. When one enters the first stage of sleep (SWS) during the night following the conversation, the combined memory trace from the hippocampus and the amygdala is replayed in the memory-editing matrix of the GHS and neocortex. Here individual synapses involved in the memory trace are strengthened by further movement of AMPA receptors onto the surface of the synapses (Ribeiro et al., 2004), or as hypothesized by Tononi and Cirelli (2003), synapses may in some cases be partially downregulated by synaptic depotentiation (but see Stickgold, 2007, for an alternative view). Downregulation would thus allow all memories of the same emotional and cognitive intensity to be recorded at approximately the same level of intensity. This operation would also preserve synaptic plasticity (the ability to change strength by adding or subtracting AMPA receptors on synapses involved in the memory). During the REM sleep stage following the SWS stage, the edited memory is fixed in the neocortex by genetic expression of molecules allowing more permanent memory traces to be formed. Thus, sleep is filled with significant events relating to memory.

Sleep memory researchers have found evidence that not all of a memory recorded during the day is processed and transferred to neocortical areas during SWS. Most of it is simply ignored during SWS and is allowed to deteriorate over time as new memories are recorded over the discarded portion of the memory. Stickgold (2007) believes that only a "schematic, gistlike" memory is processed for integration into the existing memory framework. During

SWS, activity in the GHS and in the neocortex is time-locked and thus synchronized by high-amplitude slow waves (Clemens et al., 2007; Luo, Honda, & Inoué, 2001; Wolansky, Clement, Peters, Palczak, & Dickson, 2006). The edited memories are consolidated and transferred to neocortical areas, including the ventral medial prefrontal cortex (vmPFC) during SWS (Frankland & Bontempi, 2006; Takashima et al., 2006). The vmPFC is the portion of the prefrontal cortex situated above and behind the eyes. Sleep spindles of about 13 Hz (the spindles are made up of waves that recur 13 times per second), paced by the slow waves of SWS, accompany this communication of the GHS with the neocortex (Clemens et al., 2007; Gais, Mölle, Helms, & Born, 2002).

Memories recorded during extremely stressful conditions, which are often referred to as fear memories, have special, problematic characteristics (Murburg, 1997). The powerful signals resulting from traumatic events may cause maximal potentiation of amygdalar synapses that record the events. Overpotentiation may cause all of the approximately 84 AMPA receptor-binding sites on the postsynaptic density to be filled (Earnshaw & Bressloff, 2006). If synapses mediating the affective memory trace in the amygdala are maximally potentiated, as in PTSD, the emotional memory trace recorded in the amygdala cannot be effectively merged with the cognitive memory trace from the hippocampus (this failure of merging of the two information streams was hypothesized by Corrigan, 2002). Merging ordinarily takes place within another part of the limbic system of the brain, the anterior cingulate cortex (Devinsky, Morrell, & Vogt, 1995). If the memory cannot be further processed, it may remain trapped unchanged within the GHS. Without further processing, fear memories persist for extended periods, and often for life.

When these aversive memory circuits are triggered by whatever means, the terrifying emotions associated with them are recalled as if they were being reexperienced in the present moment (van der Kolk & Fisler, 1995). Stickgold (2007) refers to such memories as "near-veridical reenactments of the traumatic events" (p. 541). In this case, only the emotional memory from the amygdala may be recalled with all the fear and horror originally experienced (van der Kolk, 1994) without input from the cognitive portion of the memory recorded in the hippocampus. This silent suffering of recalled traumatic events is a symptom of PTSD. EMDR has been found to alleviate or eliminate PTSD symptoms. The neural basis of the effect of EMDR therapy in suppressing the emotional aspect of traumatic events is a natural question arising here. Rasolkhani-Kalhorn and Harper (2006) proposed that at least part of the answer lies in memory extinction or modulation by synaptic depotentiation.

Extinction of fear memories recorded in the basolateral tract of the amygdala has been much studied in animals (e.g., Hölscher, Anwyl & Rowan, 1997; Lin et al. 2003). Low-frequency tetanic (electrical) stimulation by electrodes penetrating (or sited near) synapses mediating the fear memory results in depotentiation of AMPA receptors on the synapses by a series of complex events (details in Bender, Bender, Brasier, & Feldman, 2006; Huang, Liang, & Hsu, 2001; Klee, Ren, & Wang, 1998; Lisman, 2001; Rubin, Gerkin, Bi, & Chow, 2005). Following depotentiation, the receptors can no longer open and are subsequently removed from the postsynaptic membrane (Earnshaw & Bressloff, 2006). The affected synapse is no longer able to convey the signals that mediate the memory; repetition of this process throughout the memory trace disrupts it and the memory is extinguished.

Rasolkhani-Kalhorn and Harper (2006) reviewed the parallels between conditions and events established for animal studies of synaptic depotentiation with conditions established during EMDR therapy. Lin et al. (2003) typically used one to five stimulation pulses per second (designated 1-5 Hz) during their animal experiments. Bilateral brain stimulation applied during EMDR therapy is in the lower part of this frequency range. The number of stimuli needed to depotentiate synapses mediating memory traces in animals is about 900 according to Kopp et al. (2006) and Earnshaw and Bressloff (2006). This is similar to the number of eye movements or other brain stimulation impulses applied during a typical EMDR therapy session. During the animal experiments, direct tetanic stimulation assures that the relevant synapses of the fear memory are targeted for depotentiation. During EMDR, holding a fear memory in attention targets the relevant synapses (Nader, Schafe, & LeDoux, 2000, noted that a memory trace is labile when held in attention; in this state it is most easily changed in strength). Finally, the desired outcome of both animal research and of EMDR therapy is achieved: depotentiation of the

mechanical substrate of a traumatic emotional memory. Rasolkhani-Kalhorn and Harper were, however, unable to show that EMDR therapy results in synaptic activation equivalent to that caused by direct tetanic stimulation. They recommended a research study to determine whether this final attribute of depotentiation of fear memory synapses by EMDR can be scientifically supported and, if possible, to determine the location within the brain where this activity occurs.

Verification of the connection between the relevant animal studies and EMDR therapy is important because, if a connection does exist, then the details of molecular processes known from these studies can be applied to processes invoked by EMDR therapy. In particular, if this parallel between the animal studies and EMDR can be supported, then it can be shown that fear memories leading to disorders such as PTSD are molecularly based. This adds to the evidence that PTSD is a medical disorder. This is critical for treatment of PTSD in countries where, for significant social reasons, mental disorders are highly stigmatized. It should also eliminate the dishonor felt by many of our servicemen and servicewomen when PTSD is diagnosed.

Introduction to EEG

The purpose of this quantitative electroencephalogram (qEEG)-based study is to determine whether EMDR therapy creates memory-modulating activities that are equivalent to those known from animal studies and in particular to determine whether EMDR therapy provides a mechanism that results in opening and closing of the receptors on synapses that mediate fear memories. If so, depotentiation of fear memories in humans is likely to occur by the same mechanisms revealed by the animal studies. A second aim was to determine the location of this synaptic activity.

The cycling (opening and closing) of receptors on fear memory synapses is caused by direct electrical stimulation of the synapses during the animal experiments referred to above. This is not feasible or desirable for activation of synapses in studies of humans. We wished to determine whether repetitive bilateral brain stimulation (BBS) such as that used in EMDR therapy induces similar receptor cycling. Although EEG records taken from participants undergoing EMDR therapy show electrical activity throughout the brain, we were interested only in the activity of principal neurons in the memory relevant areas. These include the amygdala (for fear memories), the hippocampus (for cognitive memories), and portions of the prefrontal cortex (which mediates combined input from these GHS components as long-term memory).

Depolarization (firing) of neurons results from the accumulation in the neuronal body (soma) of small electrical currents initiated by their synapses. The larger electrical current caused by neuronal depolarization can be detected on the EEG, which magnifies the recorded electrical signals by factors ranging from 20,000 to 200,000 or more. The neurons tend to depolarize synchronously in carrying out the functions for which each brain area is responsible. This results in waves of activity on the EEG. In this study, we concentrated on recording and analysis of delta waves that recur one to four times per second (1-4 Hz). We were able to analyze the waves by using various quantitative methods to extract information such as frequency and power. The sample of an EEG record shown in Figure 1 demonstrates the appearance of electrical activity in the brain.

Figure 1 shows that the principal neurons in the frontal cortex fired 4 times during the 3 seconds illustrated; the indicated firing frequency is 4/3 = 1.33 Hz. The power/frequency graph on the right side of the panel shows that the power generated is about 40 μV^2 at this frequency. We found that synaptic depotentiation during EMDR requires much greater power than that shown here. The qEEG analyses are elaborated further in the following sections.

Location of the activity relevant to this study was obtained by using simple topographic mapping of the EEG data from each electrode. These maps indicate that most of the activity generated by BBS during EMDR is in the prefrontal cortex (Figure 2, left panel). A slightly more refined location was found by using a technique known as LORETA (low-resolution electromagnetic tomography). This study showed that the activity was principally confined to the basal portion of the prefrontal cortex in the vmPFC (Figure 2, right panel) extending posteriorly (toward the back of the head) to the amygdala. This brain area is significant to this study because it is known that both the vmPFC and the amygdala are involved in memory recording and down-regulation or extinction (Akirav & Maroun, 2006; Kalisch et al., 2006; Williams et al., 2006).

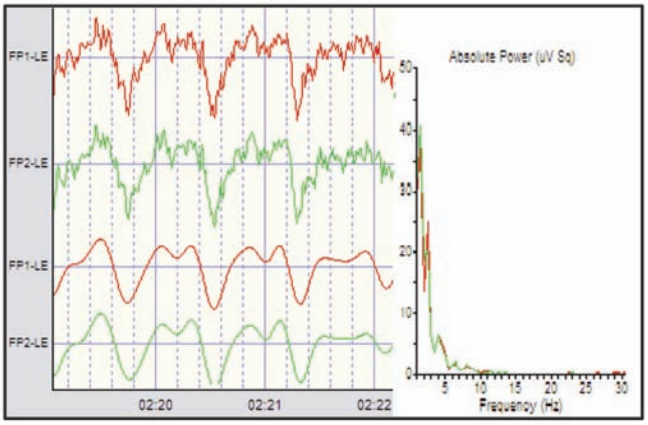

Figure 1. EEG record showing electrical activity recorded from the forehead of Participant 2 (Fp1 and Fp2, frontopolar electrodes; locations shown in Figure 2, left panel).
NOTE: The upper two curves show raw data and the bottom two show delta range data (all frequencies higher than 4 Hz filtered out). Power/frequency graph on right.

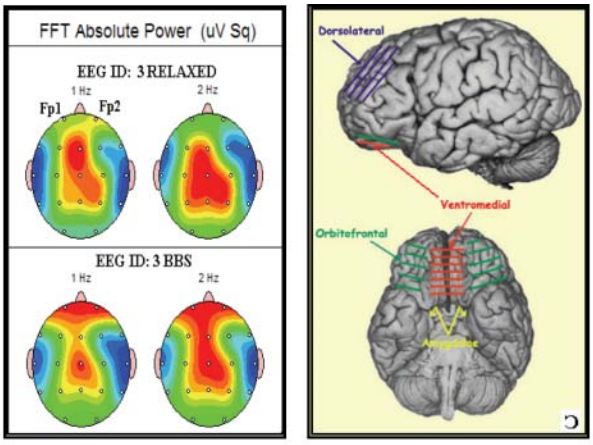

Figure 2. Left: Topographic maps showing areas of maximum intensity of electrical activity, Participant 3. Upper two maps show activity before BBS (bilateral brain stimulation) during the relaxed state; lower two show activity during BBS. Right: Location of ventromedial prefrontal cortex and the closely associated amygdala.
SOURCE: Brain image on right is reprinted with permission from "The Brain from Top to Bottom" by Bruno Dubuc, 2003. Retrieved September 20, 2008, from http://thebrain.mcgill.ca/flash/a/a_08/a_08_cr/a_08_cr_anx/a_08_cr_anx_1b.jpg

We will proceed first to describe the methods and equipment used to gather and analyze the EEG data used in the experiments, followed by presentation of results of the experiments and a discussion of the implications of these results.

Materials and Methods

Equipment

A Lexicor Neurosearch 24 EEG unit was used to acquire digitized electroencephalographic data for the first three people who participated in the experiment. A 19-channel full-head electrocap was used; electrodes were placed according to a standard location technique. The third participant disclosed that the procedure we were using caused distress that interfered in her case with the therapy session. She reported "getting panicky" by the time the actual therapy session began; application of the 19 electrodes had taken about 40 minutes. Also, we had found that montages of records from frontopolar electrodes Fp1 and Fp2 (Figure 2, left) were sufficient for our purposes. Following the third experiment, we used a two-channel BrainMaster Atlantis II EEG acquisition unit for the remaining participants. The application of only two electrodes reduced participant preparation time to about 10 minutes, and we had no complaints from the participants of subsequent experiments. We also used the two-channel acquisition system for laboratory (as opposed to clinical) experiments during the study. Although the resolving power of the 19-channel system is higher, and the power scale is different for the two systems, overall shape and spectral power curves are similar in the two systems.

A Tac/AudioScan™ unit by Neuro*Tek*™ was used to provide tactile stimulation (BBS) at a controlled frequency during many of the lab experiments and most of the clinical experiments. Intensity of stimulation was set at maximum. A digital metronome was used during some of the lab experiments to pace brain stimulation input frequency.

Data Processing

The usual data processing techniques included visual inspection of the EEG raw data recordings for overall wave properties and their changes during each stage recorded. The most relevant and representative portions of each recording were further examined by separating the frontopolar traces, Fp1 and Fp2, from the remainder of the EEG traces recorded (as in Figure 1).

We used programs developed by NeuroGuide™ to examine the raw data in these traces and to obtain the several data sets reported in this study. These data sets included measurements of wave frequency and

power. Absolute power of representative sequences of the EEG records was calculated and imaged by the program. Because the frequencies most relevant to memory modification are in the delta range, we used filters that pass only these frequencies to produce images for visual inspection and frequency analysis. We sequestered data into 6- and 10-second panels for visual displays and then manually counted the number of waves of each type in each panel to obtain frequency of neuronal firing.

EEG Protocol

We obtained 3- to 5-minute EEG recordings of six brain states of each participant, providing 90 to 150 two-second "epochs" for each state for each participant. The initial state of eyes open, relaxed attention to a dot on the wall before the participant was followed by several phases of eyes closed conditions: relaxed attention as if listening to a lecture, holding in attention a previously chosen fear memory, three phases of applications of BBS with the fear memory in attention, and, finally, a repetition of Condition 2, relaxed attention. Fifteen to 20 minutes of EMDR with BBS were recorded during each clinical experiment.

Participants in the Experiment

The EEGs of 6 participants, all exhibiting symptoms of PTSD, were recorded during the experimental procedure. Participant ages ranged from 26 to 74 years; three were female. Prior to the experiments, each was given a clinical interview to determine trauma history and general appropriateness for the procedure. A personality assessment inventory was administered (except for the fourth participant, who could not read English), along with a posttraumatic stress inventory based on the Foa PDS, and a test for degree of likelihood of dissociation (the dissociative experiences scale, DES). A brief psychological profile was prepared for each participant. The participants had in common a traumatic memory they could easily visualize during the procedure. Four prospective volunteers were deemed unsuitable for the experiment because their history (which often included childhood sexual abuse) suggested possible disorders of extreme stress (DESNOS) or chronic PTSD. These volunteers were given names of relevant practitioners and were advised to seek treatment.

EMDR Therapy Procedures

Our principal aim in the EEG studies of EMDR was to determine the reaction of the brain to the part of the EMDR protocol that includes BBS (Phase 4). A standard EMDR procedure for Phase 4 was used during the EMDR sessions; other phases of the standard protocol were carried out before or after the EEG recording stage and these are not further elaborated in this article. The participant was instructed to hold in attention a fear memory previously discussed with the EMDR therapists (TRK and MH) while BBS was applied. A body scan was conducted prior to beginning the BBS, and somatic effects were monitored during and after the EMDR session with BBS.

The usual BBS source was vibrating pads held in the palms of the hands with a vibration frequency of 1 to 2 Hz (one to two vibrations per second). The vibrations were bilaterally applied; that is, a vibration in the right palm was followed by a vibration in the left palm, and so on. Lateral eye movements were used only briefly (Participants 1 and 4) in the EMDR sessions recorded during this study. The principal difference with an ordinary EMDR session was that the participants' eyes were closed during the EEG recording (except for the first state of eyes open, relaxed attention). The recordings were interrupted at 1- to 2-minute intervals for the usual questions and comments by the therapist or the participant. Length of the sessions with BBS was 15 to 45 minutes; session length was determined by changes in the Subjective Units of Distress (SUDs) scale. We discontinued the EMDR session with BBS shortly after the participant reported a SUDs score of 1 or 0, or if the subjective feeling caused by memory recall was not changing for a significant period of time (the SUDs scale was devised by Wolpe, 1958, to monitor the progress of clients undergoing an earlier form of synaptic depotentiation therapy).

Lab Studies of Bilateral Brain Stimulation

In addition to the EEG recordings made during the clinical phase, we performed extensive laboratory studies of EEG frequency and power of brain waves generated by various forms of brain stimulation. These were designed to further investigate in a lab environment (as opposed to the clinical environment)

unexpected responses of the brain to the procedures we were employing in the clinical studies. This phase of the project included investigation of possible reasons for the exceptionally strong responses of various forms of brain stimulation and the apparent differences in input frequency as compared to output frequency (discussed in the Results section below).

Tactile stimulation was found to be practical for these studies, but aural modes introduced noise in nearby electrodes. The most noise-free stimulation mode was the Tac/AudioScan™ vibrating pads held in the palms of the hands. Reactions to stimulation caused by tapping of the shoulders, palms, neck, and face were recorded. We also studied the electrical characteristics of lateral eye movements as originally used by Francine Shapiro (1989a), which is still perhaps the most common stimulation method during EMDR. This form of stimulation was always successful in creating high-magnitude brain waves in the frontal lobes, but it was not used in calculation of power because it creates a complex added electrical component in the EEG records. The eyes form electrical dipoles whose movement generates an electrical signal (Malmivuo & Plonsey, 1995, chap. 28). This added component complicates the analysis of spectral power of the waves generated by the brain itself. Brain stimulation studies continue and will be further assessed for their implications to EMDR therapy.

Results

EMDR Therapy

Symptoms of PTSD of all participants of the EMDR EEG experiments were significantly reduced. All had SUDs scores of 2 or less at the end of the EMDR sessions, and somatic effects were reduced or eliminated. Each was asked after the BBS session to reassess the somatic effects reported at the beginning. Each reported no continuing pain or lessened pain. The participant with a SUDs score of 2, when reexamined 1 week later, reported that the fear memory was no longer experienced as an emotionally negative event, thus indicating a SUDs score of 0; also, the participant no longer had somatic symptoms associated with the memory. We contacted all participants over a 1- to 10-month period following their therapy session. They reported continued positive effects in their lives and sustained relief from the fear memory and its concomitant somatic pain.

qEEG Recordings and Analysis

We stress in this article the results of the qEEG data, following Schiff's (2005) warnings about the possibly erroneous assumption of synchrony of event arrivals interpreted from EEG records. That is, calculations based on timing of arrivals of EEG signals at given electrodes may be in error if it is assumed that these signals come from specific coupled systems in the brain; they may come from uncoupled systems. Only limited information obtained from standard processing is reported here because of this possibility of error introduction.

Figure 3 shows typical waveforms recorded prior to the beginning of EMDR therapy, while the participant was alert and not thinking of the fear memory. These provide a baseline of EEG power and frequency for comparison with the power and frequency of waves recorded during the succeeding stages of the EEG and EMDR protocols. These baseline EEG recordings show typical waking state frequency and power distribution: intermittent low-amplitude delta waves, occasionally more powerful theta and alpha waves, and variable-power beta waves. These groupings of frequencies are best illustrated in the fast Fourier transform (FFT) power/frequency graph shown on the right side of each panel in Figure 3. This combination of frequencies is typical of the brain state during which memory is recorded by synaptic potentiation in memory areas of both the limbic and neocortical systems, as noted above.

An increase in power, and thus of wave amplitude (indicated by the more well-defined delta waves in Figure 4), occurs when the participant considers the fear memory in attention; that is, merely recalling the memory increases the power of the brain waves. Rhythmical delta activity in the frontal lobes is unusual in the waking state, and according to **Niedermeyer (2003), it is** possibly related to thought processes under unusual conditions. Recalling a traumatic experience would be such a condition. This power increase of frontal delta waves when attending to a fear memory is significant, we believe, to depotentiation therapies in general. This aspect of the results is discussed below.

Figure 5 demonstrates that a two- to threefold increase in EEG power occurs when BBS is applied during EMDR (Phase 4 of the standard EMDR protocol). The delta waves become even more sharply defined (shown on left side of each panel), and their power is increased (graph on right side of each

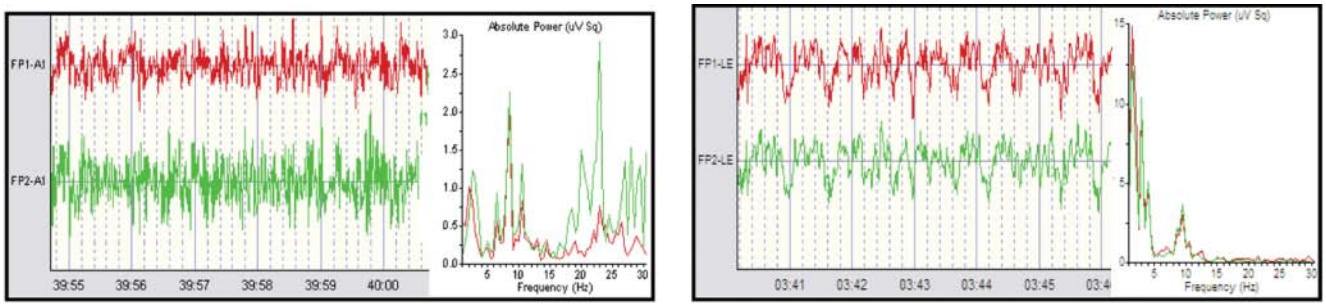

Figure 3. EEG of relaxed state, Participants 1 (left) and 2 (right).
NOTE: Absolute power scale for the two time sections is different because of change of equipment (left section, Atlantis II; right, Neurosearch 24). Both show low-power delta activity of 1.5 to 2 Hz in the absolute power graph on the right of each panel.

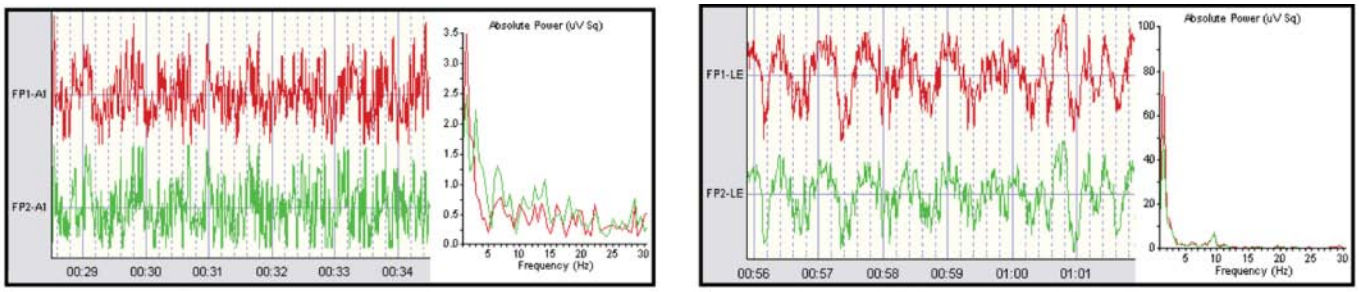

Figure 4. EEGs of Participants 4 (left) and 2 (right) with fear memory in attention but without brain stimulation.
These records show an increase in power and greater definition of delta waves over the relaxed state shown in Figure 3.
NOTE: Acquisition units as in Figure 3.

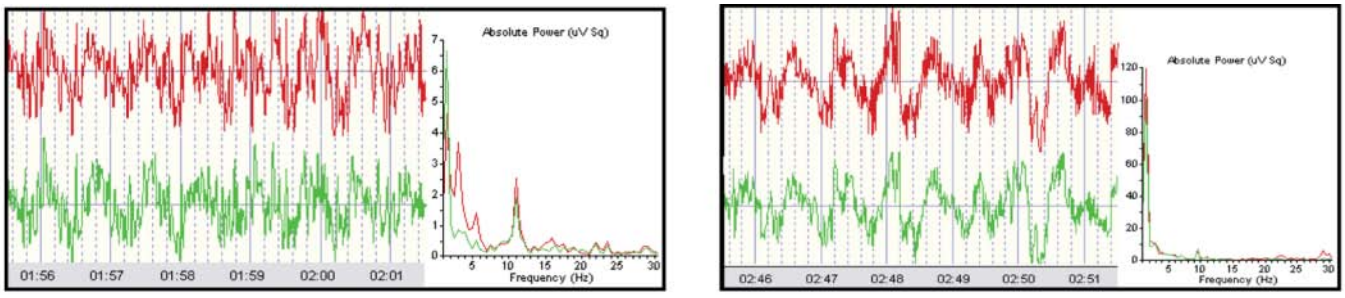

Figure 5. Examples of EEGs of Participants 5 (left) and 2 (right) during EMDR therapy. BBS applied (vibrating pads in palms of hands), eyes closed, and fear memory in attention.
NOTE: Acquisition units as in Figure 3.

panel). Implications of this great increase in power to the modulation or extinction of a fear memory trace are discussed below.

As noted in Figures 3, 4, and 5, EEG records taken during the clinical experiments show a progressive increase in EEG wave power through the stages from baseline relaxed attention through attention to a fear memory and then to fear memory with BBS (Phase 4 of the EMDR protocol). Representative 20-second sequences were sampled from the raw data during the experiments and subjected to power analysis. These analyses are summarized in Figure 6

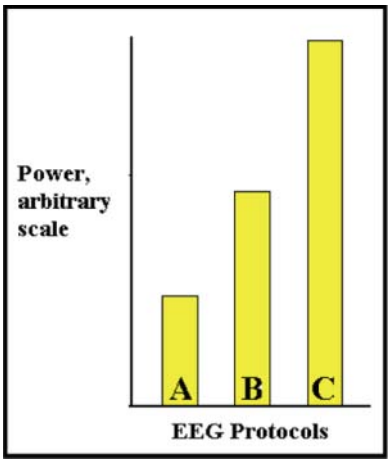

Figure 6. Relative EEG spectral power observed in Participant 2 in the following attentional states (EEG protocols): (A) relaxed, (B) with fear memory, and (C) EMDR session with fear memory and BBS.

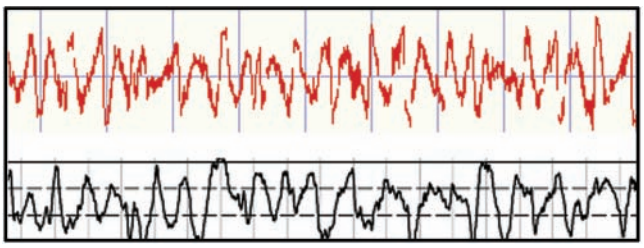

Figure 7. Illustration of similarity of EEG data recorded during BBS (upper) and slow wave sleep (lower).
NOTE: Data for upper waveform is from Participant 1; bottom waveform is from Rétey et al. (2005). Copyright 2005 National Academy of Sciences, USA, used with permission. Width of section: 20 seconds.

for data from Participant 2 to demonstrate the power changes in the EEG records during various stages of the recordings. Stimulation source in the bar labeled C (EMDR therapy) was vibrating pads held in the palms of the hands.

The power, frequency, and overall appearance of the EEG recorded during BBS (lateral eye movements) are compared in Figure 7 to an EEG recording of SWS. Data for the upper waveform shown is from frontopolar electrode Fp2 (recording electrode on the right side of the forehead over the prefrontal cortex of Participant 1). The lower curve is taken from Figure 2 of Rétey et al. (2005), showing EEG waves recorded during SWS. Wave characteristics recorded during the two brain states (EMDR and SWS) are similar, and implications of this similarity are discussed below.

Surprisingly, BBS input frequency did not dictate EEG output frequency (Figure 8). Input frequency (number of taps, vibrations or eye movements per second) during the laboratory and clinical experiments ranged from 0.5 to 2.5 Hz, whereas the evoked response (EEG) frequency ranged from about 1.25 to 1.75 Hz. The trend toward 1.5 Hz output was constant across participants, equipment, and brain stimulation technique used. This frequency also appears on EEG records during the pre-EMDR phases (Figures 1, 3, and 4). Brain stimulation applied during EMDR greatly amplifies this natural rhythm (Figures 5 and 6). A preferred neuronal response frequency in the memory networks (Dang-Vu et al., 2008; Lakatos et al., 2005) may be activated by BBS. Alternatively, or possibly in addition, a naturally occurring low-frequency thalamocortical driving mechanism (Mölle, Marshall, Gais, & Born, 2004; Rigas & Castro-Alamancos, 2007; Wolansky, Clement, Peters, Palczak, & Dickson, 2006) may be activated during BBS (Bergmann, 2008). Interestingly, Lakatos et al. (2005) found a spontaneous oscillation of around 1.86 Hz (standard deviation 0.25 Hz) in the auditory cortex of rhesus monkeys. That is, the natural firing (depolarization) frequency of these cortical neurons was about 1.86 Hz. However, these researchers found that this innate frequency changed to match the applied frequency. For example, when the applied frequency is 1 Hz, the evoked frequency is also 1 Hz. As stated above, we found that the frequency of firing of neurons in the memory areas of the brain that are activated by BBS remained almost constant at about 1.5 Hz, regardless of the input (BBS) frequencies used.

Discussion

EMDR Therapy and Synaptic Depotentiation

EMDR therapy is basically simple: The therapist instructs the client to hold a traumatic memory in attention while receiving mild brain stimulation, such as by vibrating pads held in the palms of the hands. The attended memory must be explicit and should include elements such as a specific iconic representation of the most disturbing image or images from the memory, as well as the specific emotional responses caused by the traumatic incident. The client is also instructed to attend to somatic effects resulting from attending to the

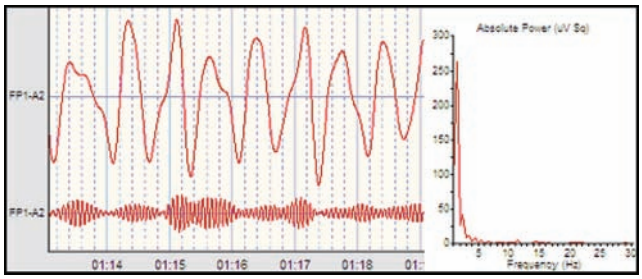

Figure 8. Comparison of BBS input with EEG output. NOTE: Input was BBS using 1 Hz lateral eye movements, Participant 1. The evoked response shown here consists of 1.5 Hz delta waves and 13.5 Hz beta spindles paced by the delta waves. Delta and low beta bandpass filters applied; width of section is 6 seconds.

memory and to monitor any changes that occur in the body image during the procedure. As the therapy session proceeds, the client may be encouraged to separate from the emotional aspects of the memory by thinking of it in progressively more cognitive, rather than affective, terms. This is also the natural neural course of an EMDR session, as synapses that mediate the emotional part of the memory go offline because of depotentiation.

Quite often, and in every case for participants of this experiment, a single EMDR session with BBS (Phase 4 of the EMDR protocol) is sufficient to erase or modulate any extreme emotions that may accompany the traumatic memory (Shapiro, 1989a, 1989b). Persons undergoing therapy are usually surprised at the rapid change in their perception of the traumatic event. One of the participants stated after the EMDR session, "I try to feel afraid when I think of [the attacker] but I cannot feel fear or even remember the fear. What has happened in the last hour of the years since the incident occurred?" Most people who have undergone EMDR therapy feel they have experienced a profound change in the way they react emotionally to the targeted memory. Understanding the neurological basis of the EMDR effect is important for both therapists and their clients.

As outlined above, the fear memories mediated in the basolateral complex of the amygdala are extinguished in animals when low-frequency tetanic (electrical) stimulation is applied to a fear memory synapse. This stimulation causes cycling of the receptors on the postsynaptic membrane at the stimulation frequency (usually from 1 to 5 Hz) and results in depotentiation of the AMPA receptors as the calcium concentration in the synapses decreases during the interstimulus interval (Lin et al., 2003). About 900 tetanic impulses are required to extinguish the fear memory. For example, Lin et al. found that depotentiation of a fear memory synapse in a laboratory animal is complete after 15 minutes of tetanic stimulation at 1 Hz, that is, 1 pulse per second ($15 \times 60 \times 1 = 900$ pulses). Higher stimulation frequencies of up to about 5 Hz speed the process of synaptic depotentiation in the experiments by Lin et al. These researchers found no depotentiation when the frequency of stimulation was 0.1 Hz (1 stimulation pulse each 10 seconds).

The qEEG data reported above from EMDR sessions show a similar number of depolarizations of neurons supporting fear memory synapses in human participants during a typical 50-minute EMDR therapy session (which includes many interruptions of BBS to consult with the client). In our study, we found that BBS caused immediate slowing of the depolarization rate of neurons in the frontal lobes from the dominant waking state frequency of around 7 Hz (Figure 3) to about 1.5 Hz (Figure 5). As noted, this evoked response may result from the intrinsic properties of the principal cells in the memory networks of the neocortex and the limbic system or from a thalamocortical rhythm usually associated with SWS. BBS also generates a several-fold increase in wave power (Figure 6). The high power of the induced waves insures that all synapses mediating the memory held in attention become synchronously active. The change to high-power, low-frequency waves of neuronal depolarizations is a change from conditions favorable for synaptic potentiation to one of depotentiation.

The AMPA receptors on fear memory synapses are entirely depotentiated in the animal experiments, resulting in extinction of the memory. We believe that during EMDR these receptors are either entirely removed as in the animal experiments or reduced to a number that allows some emotional charge to remain. In either case, the affective and cognitive aspects of the memory mediated in the GHS can now be combined in the ACC, and the memory trace is no longer confined by its hyperpotentiation to the limbic memory areas. This removal or normalization of AMPA receptors in the amygdalar synapses is like removal of scar tissue that has led to the problematical functioning of the memory trace.

Subsequently, the complete memory can be sent to higher brain centers, such as Broca's area, where a narrative to express the memory is added. It can also be fully processed for the first time since its origin in the SWS editing matrix, where it is further normalized for integration into the memory matrices of the neocortex.

This completes a chain of logic showing the equivalence of brain stimulation during an EMDR therapy session to tetanic stimulation leading to depotentiation of fear memory synapses of animals. We conclude that the EMDR effect in humans results from similar depotentiation. We believe that the desensitization indicated by the *D* in EMDR results from depotentiation of fear memory synapses.

We have in this study emphasized the fear memory type that is associated with PTSD and mediated, we believe, by maximal potentiation of synapses in the basolateral complex of the amygdala. As noted, many researchers believe such memories cannot be processed in higher centers of the brain and remain unchanged in the limbic system. More normal fear memories that *can* be processed in higher centers can also cause both psychological and physiological discomfort that can be alleviated, like PTSD, by EMDR therapy. These might include memories of merely worrisome events that occur in our lives, but which, unlike the PTSD fear memories, do not normally limit our ability to cope with life. We believe the same mechanisms that result in depotentiation of the neural traces that mediate PTSD memories also operate to reduce the emotional impact of memory traces mediating more ordinary life events.

Similarities and Differences Between EMDR and Slow Wave Sleep

Frequency and power of waveforms induced by BBS during EMDR therapy are similar to those of the slow oscillations and delta waves of SWS (Figure 7). Also, low beta waveforms developed during BBS (Figure 8) are similar to sleep spindles invoked during SWS.

The slow waves induced by BBS are most evident at the frontopolar electrodes Fp1 and Fp2 (Figure 2, left), and other data demonstrate that this frontal lobe activity is principally centered in the vmPFC (which includes the semantic cortex) extending posterioriorly to the amygdala (Figure 2, right). It is significant that EEG recordings by Takashima et al. (2006) show that the vmPFC is also a center of powerful delta activity during SWS. This similarity of activity on EEG records during BBS and SWS suggests that EMDR activates the naturally occurring memory processing system of SWS. A difference between the waking and SWS states is that during EMDR therapy (the awake state) we consciously direct our attention to a specific memory, whereas during SWS nonconscious processes in the brain select a memory automatically. Another significant difference is that the acetylcholine (ACh) neuromodulatory system is at a minimum of activity during SWS, whereas during the waking state and REM sleep it is high.

Acetylcholine concentration has a profound and complex influence on memory processes in the GHS (Gais & Born, 2004; Hasselmo, 1999; Power, 2004; Rasch, Born, & Gais, 2006). These researchers found that normal levels of ACh during the waking state and REM sleep result in isolation of the GHS from upward communication with the neocortex during memory formation. This restriction of upward communication is thought to reduce the possibility of incorporating irrelevant data that might interfere with concurrent memory recording in the GHS. It has also been found that high ACh concentration favors memory acquisition and processing during the waking state and REM sleep, whereas low ACh concentration during SWS favors memory consolidation (Gais & Born, 2004). These two researchers also noted that low ACh concentration causes the emergence of the slow waves that characterize SWS (Figure 7).

McCoy and McMahon (2007) found that activation of certain ACh receptors induces depotentiation in layer 2/3 of the rat visual cortex. Amygdalar synapses in humans may similarly be influenced by ACh-based activity, and this may promote depotentiation. If so, the normal ACh level during EMDR as contrasted to ACh absence during SWS may explain why slow waves during slow wave sleep only modulate synaptic strength while such waves created during EMDR tend to depotentiate fear memory synapses.

Effect During Sleep of Sensory Input During the Waking State

Kattler, Dijk, and Borbély (1994) discovered that vibratory stimulation of the palm of the right hand during wakefulness increases delta power in the EEG recorded over the left somatosensory cortex during the next succeeding episode of SWS (for review, see Peigneux et al., 2004; and for an animal study of the phenomenon, see Vyazovskiy, Borbély, & Tobler, 2000). That is, if during the waking state a stimulation of the hand is applied, an increase of delta wave power occurs when the memory is replayed during the next SWS episode. The implication of this insight to EMDR therapy for PTSD is that during SWS

following the day of the therapy, the memory of the therapy session is likely to be replayed with the higher power elicited by BBS during the therapy. This may lead to further reprocessing of any remaining portions of the affective, emotional portion of the memory. This might also be repeated during subsequent nights for 1 to 3 months or longer, as is the case for most memories (Frankland & Bontempi, 2006; Takashima et al., 2006; Takehara-Nishiuchi & McNaughton, 2008). We have found the phenomenon of further improvement in mental states associated with a traumatic memory over the days or weeks following EMDR therapy in our clinical practice and during the EEG-EMDR experiments reported here. Anecdotal evidence suggests that other therapists have found similar improvements.

Other Depotentiation Therapies

Therapies relying on the exposure of the client to traumatic memories, including Behavioral Therapy (Wolpe, 1958 and 1990), Flooding Therapy (Keane, Fairbank, Caddell, & Zimering, 1989), and Exposure Therapy itself (Foa, et al., 1999) fall into the category of depotentiation therapies when considered from the likely mechanism of action (although Lee, 2008, believes that exposure therapy has a mechanism of action different to that of EMDR). Unlike EMDR, these therapies do not include brain stimulation such as BBS. Instead, they rely on repeated exposure to the fear memory over many sessions (up to 100 or more in the case of Wolpe's, (1958) technique). The basis for the therapeutic effects of these therapies is likely to be depotentiation of fear memory synapses triggered by the increase in power of delta waves (Figure 6) during simple recall of the memory. Several sessions are required because the delta wave amplitude induced by merely thinking of the traumatic event apparently does not reach the critical level for widespread and rapid depotentiation of synapses in the amygdala as it does during BBS.

Why No Depotentiation of Hippocampal Memories?

Why does EMDR decrease affective aspects of traumatic memories in the amygdala while leaving intact, or even enhancing (Propper & Christman, 2008), the associated cognitive aspects of the memories recorded in the hippocampus proper? We believe this phenomenon results from the overpotentiation of synapses in the amygdala caused by maximal intensity of emotions during traumatic events. Recording of the cognitive aspect of memory in the hippocampus is likely to result in more normal potentiation of hippocampal synapses.

Also relevant to this question is a study by Hendler et al. (2003) that shows that the amygdala might be especially susceptible to pathological processing of traumatic emotional memories because of its lack of efficient sorting mechanisms for content recording. For example, the amygdala might be less efficient than the hippocampus in filtering out or modulating signals from the environment that are too strong to be accommodated by the amygdalar memory recording mechanism itself. The high intensity of these signals can result in pathological processing of the memory. The amygdala seems not to have developed adequate safeguards for protection against overdriving of its mechanisms.

Implications for Teaching Depotentiation Therapies in Other Countries

Because the theory of depotentiation of AMPA receptors on fear memory synapses in the amygdala during EMDR is now supported by the qEEG studies reported here, depotentiation therapy is likely to be accepted in countries that insist on scientifically based therapies. A protocol designed for psychological first aid could be developed and taught to medical personnel in local health posts around the world. This is particularly important in countries such as Iran, Nepal, and Afghanistan, all of which have a large number of people who suffer from PTSD and a small number of available trauma therapists (there are fewer than 10 psychologists in Nepal according to the director of the western regional hospital in central Nepal). The nature and results of the treatment show that stress disorders such as PTSD are the result of problematic memory trace storage, and for this reason are medical conditions underlying what have been considered mental illnesses. This is particularly important in countries where significant social stigmas exist against treatment for mental illnesses. Depotentiation therapies such as EMDR are particularly welcome in such countries.

Conclusions

Dr. Francine Shapiro (2001), the discoverer of EMDR, has long believed that EMDR is successful because it activates a self-healing mechanism of the brain. However, the neural basis of this healing has not been

known with certainty. EEG records of EMDR sessions reported here show that low-frequency brain stimulation invokes a large response from the memory areas of the brain; this response is at a frequency of about 1.5 Hz regardless of the input frequency over the range of about 0.5 to 2.5 Hz. Animal studies show that low-frequency tetanic (electrical) stimulation results in depotentiation of AMPA receptors on synapses mediating fear memories in the basolateral complex of the amygdala, thereby extinguishing them. Evidence is given here, based largely on qEEG data, that depotentiation therapy such as EMDR establishes the same conditions as those existing in the animal studies, but with low-frequency stimulation of the brain substituting for tetanic stimulation. Depotentiation of fear memory synapses in the amygdala disrupts the fear memory circuits; this mechanical change results in a beneficial perceptual change. It is also significant that the brain state established during EMDR is found to be similar to that of the memory editing system of SWS.

EMDR therapy is the simplest and most successful psychological therapy for healing or mitigating affective disorders such as PTSD caused by an increasingly traumatic world. It requires no equipment or drugs. It can be taught, along with necessary safety procedures, to medical personnel down to the level of village nurses in some Third World countries. Shapiro and Maxfield (2003) support this more egalitarian concept of the application of EMDR therapy. EMDR group therapy such as that reported by Konuk et al. (2006) is likely to be the only practical and efficacious protocol for use in cases of mass trauma resulting from natural or manmade disasters such as earthquakes and wars. This aspect of EMDR therapy is currently being studied by one of us (TRK).

All psychologists should learn the EMDR technique; the world is in desperate need of more trauma therapists. Those who treat our servicemen and servicewomen returning from Iraq and other wars should be aware of the efficacy of EMDR treatment for psychological traumas sustained in war, as well as the fact that PTSD is as much a battle injury as a bullet wound.

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