Rapid Treatment of PTSD: Why Psychological Exposure with Acupoint Tapping May Be Effective

David Feinstein, Ph.D.

Abstract

Combining brief psychological exposure with the manual stimulation of acupuncture points (acupoints) in the treatment of post-traumatic stress disorder (PTSD) and other emotional conditions is an intervention strategy that integrates established clinical principles with methods derived from healing traditions of Eastern cultures. Two randomized controlled trials and six outcome studies using standardized pre- and post-treatment measures with military veterans, disaster survivors, and other traumatized individuals corroborate anecdotal reports and systematic clinical observation in suggesting that (a) tapping on selected acupoints (b) during imaginal exposure (c) quickly and permanently reduces maladaptive fear responses to traumatic memories and related cues. The approach has been controversial. This is in part because the mechanisms by which stimulating acupoints can contribute to the treatment of serious or longstanding psychological disorders have not been established. Speculating on such mechanisms, the current paper suggests that adding acupoint stimulation to brief psychological exposure is unusually effective in its speed and power because deactivating signals are sent directly to the amygdala, resulting in the rapid attenuation of threat responses to innocuous stimuli. This formulation and the preliminary evidence supporting it could, if confirmed, lead to more powerful exposure protocols for treating PTSD.

Keywords: Acupuncture, Energy Psychology, Consolidation, Exposure, PTSD

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Rapid Treatment for PTSD: Why Psychological Exposure with Acupoint Tapping May Be Effective

David Feinstein

Extensive research attempting to understand PTSD still leaves substantial "challenges to the conceptualization of the disorder" (Zoellner, Eftekhari, & Bedard-Gilligan, 2008, p. 258) and its treatment. In a comprehensive assessment of the evidence on psychological and pharmaceutical treatment outcomes, the Institute of Medicine (IOM) of the National Academy of Sciences found that despite nearly three decades of research since the *DSM III* adoption of PTSD (American Psychiatric Association, 1980), the existing studies "do not form a cohesive body of evidence about what works and what does not" (Committee on Treatment of Posttraumatic Stress Disorder, 2008, p. 10). The single type of intervention (psychological or pharmaceutical) whose efficacy was judged as having been established according to the rigorous standards used in the IOM's review was psychological exposure.

Investigations of cognitive interventions were, surprisingly, inconclusive. For example, comparisons of exposure treatments alone and exposure combined with cognitive restructuring showed positive outcomes for each that persisted at one-year follow-up, but in most comparison studies, the addition of the cognitive restructuring did not enhance the treatment outcomes (e.g., Foa et al., 2005). The IOM's conclusions regarding the singular effectiveness of exposure in the psychological treatment of PTSD were corroborated in a follow-up review conducted for the American Psychiatric Association (Benedek, Friedman, Zatzick, & Ursano, 2009).

Psychological Exposure in the Treatment of PTSD

Psychological exposure is a component of various established PTSD treatments, such as Cognitive Behavior Therapy (CBT) and Eye Movement Desensitization and Reprocessing (EMDR). In exposure protocols, the activation of anxiety-producing memories or cues is part of the treatment strategy. Explanatory mechanisms such as habituation, extinction, reciprocal inhibition, counter-conditioning, information processing, and cognitive restructuring have been proposed by various investigators to explain the method's success (see Tryon, 2005). Exposure may be conducted by having the patient bring to mind feared events using images and narrative ("imaginal exposure"), by placing the person in actual (though safe) anxiety-provoking situations ("in vivo exposure"), or by using computer-simulated scenes ("virtual reality"). The process may be conducted in a progressive manner, with many exposures to increasingly distressful situations ("graduated exposure"), or the patient may be initially exposed to and kept in a highly stressful imagined situation ("implosion") or in a stressful in vivo situation ("flooding") until the anxiety and fear diminish. Repeated exposure to the stimulus with no aversive consequence often leads to a temporary termination of the anxious response, and as such experiences are consolidated into long-term memory, extinction becomes established (Santini, Muller, & Quirk, 2001).

In many protocols, repetition and/or length of exposure are relied upon to extinguish the anxiety response. In other approaches, physical interventions that are incompatible with an anxiety response are paired with the exposure based on the principle that incompatible physiological states cannot occur simultaneously. The earliest formulation of this strategy was

systematic desensitization (Wolpe, 1958). Wolpe paired deep muscle relaxation with graduated imaginal exposure to anxiety-producing scenes in order to induce "reciprocal inhibition." This in turn, after memory consolidation, produces permanent counter-conditioning. Subsequent variations have (a) utilized other physical interventions such as diaphragmatic breathing or bi-lateral stimulation, (b) trained patients prior to the exposure sessions to induce an unstressed state via relaxation techniques or biofeedback, or (c) used "interoceptive exposure," an approach akin to mindfulness meditation where the patient's focus is shifted from the anxiety-producing scene itself to the elevated heart rate, chest pains, dizziness, hot flashes, or other physical sensations that are components of the anxiety (Barlow, 2007).

Combining Exposure Treatment with the Stimulation of Acupuncture Points

The protocol examined in this paper combines brief psychological exposure with the physical stimulation of specified areas of the skin. These areas generally correspond with acupuncture points (acupoints) which are believed to (a) send signals to the exposure-aroused limbic system that (b) reduce limbic hyperarousal (Dhond, Kettner, & Napadow, 2007; Fang et al., 2009; Hui et al, 2000, 2005), leading to (c) rapid reciprocal inhibition and (d) long-term counter-conditioning. Systematic desensitization and acupoint stimulation both attempt to produce reciprocal inhibition. They differ in how they attempt to produce it. Unique to acupoint stimulation, according to its proponents, is that the signals the procedure purportedly sends to the limbic system reduce hyperarousal with markedly greater speed (briefer exposure and fewer repetitions are sufficient) and power (stress responses of greater intensity can be eliminated) than relaxation-based interventions (Feinstein, 2009).

The choice of acupoints, the number of acupoints stimulated, the order in which they are stimulated, and the forms of stimulation (e.g., tapping, holding, massaging) vary with different practitioners, approaches, and clinical situations (Gallo, 2002). Common elements in treating PTSD typically include (a) using words or imagery to activate a traumatic memory or emotional trigger, (b) giving a 0 to 10 rating on the amount of subjective distress (after Wolpe, 1958) caused by the memory or trigger, and (c) having the client tap on between 4 and 14 predetermined acupoints for about 5 seconds each while keeping the memory or trigger mentally active. An auxiliary physical technique might include having the client alternate between humming and counting to stimulate the right and left brain hemispheres. Auxiliary psychological techniques might include an exploration of internal objections to overcoming the distress or identification of aspects of the problem that have not been addressed, any of which might shift the focus of what is being mentally activated during the acupoint stimulation. This part of the work often resembles the cognitive restructuring activities seen in CBT. Ratings of remaining subjective distress are asked for periodically and provide a quick gauge of progress as well as information that may lead to a shift in focus, which is done frequently and considered to be part of the flexibility the approach allows.

Preliminary Evidence on the Efficacy of Exposure/Acupoint Protocols

A small body of evidence offers preliminary support for claims that exposure/acupoint stimulation yields stronger outcomes than exposure strategies that incorporate conventional relaxation techniques. For instance, a randomized controlled trial (RCT) comparing a single

imaginal exposure/acupoint tapping session with a single imaginal exposure/diaphragmatic breathing session in the treatment of specific phobias of insects or small animals found the acupoint tapping to be statistically superior to the diaphragmatic breathing on four outcome measures (Wells, Polglase, Andrews, Carrington, & Baker, 2003). Two partial replications of that study supported its findings (Baker & Siegel, 2005; Salas, Brooks, & Rowe, 2010). Another RCT, with 32 high school students, compared self-applied exposure/acupoint stimulation with progressive muscle relaxation. Students with elevated scores on a standardized test-anxiety inventory showed a significant decrease in text-taking anxiety after using either approach, but the decrease for the acupoint group was significantly greater ($p < .05$) than the decrease for the progressive relaxation group (Sezgin & Özcan, 2009). A small controlled pilot study using two standardized pre-/post-treatment inventories, also investigating test-taking anxiety, showed exposure/acupoint tapping to attain in two sessions benefits that required five sessions of CBT (Benor, Ledger, Toussaint, Hett, & Zaccaro, D., 2009). Other studies or pilot investigations have shown efficacy for the approach with speaking anxiety, weight management, a variety of phobias, post-trauma anxiety, and PTSD (Feinstein, 2008a).

Credible accounts of strong and rapid positive clinical outcomes with war veterans and disaster survivors after receiving exposure/acupoint protocols in the treatment of PTSD have been accumulating on websites such as http://www.stressproject.org/comments1.html and http://www.eft4vets.com/Andy.html. These anecdotal reports are being corroborated by systematic studies using standardized PTSD assessments. For instance:

- Church, Hawk, et al. (2010) conducted an RCT in which 59 military veterans with PTSD symptoms were randomly assigned to a treatment group or a wait-list control group. Fifty-four of the initial participants completed the study, including 29 in the treatment group and 25 in the control group. Pre- and post-treatment assessments included two standardized self-report inventories: (a) the military version of the Post-Traumatic Stress Checklist (PCL-M; Forbes, Creamer, & Biddle, 2001), a self-inventory that correlates well with clinician-rated assessments of PTSD (Monson et al., 2006), and (b) the Symptom Assessment 45 (SA-45; Davison et al., 1997). Six hour-long sessions using an exposure/tapping protocol were administered to each participant in the treatment group. The initial mean PCL-M score was 61.4 for the treatment group and 66.6 for the wait-list group. The PTSD cutoff is 50. The mean score after six treatment sessions had decreased to 34.6 ($p < .0001$), substantially below the PTSD cutoff, while it was essentially unchanged (65.3) for the wait-list group a month after the initial testing. The breadth of psychological distress as measured by the SA-45 had also diminished significantly at the end of treatment ($p < .0001$), as had the severity ($p < .0001$). Both measures remained stable for the control group.

- In another study of veterans and family members, pre- and post-treatment scores on the PCL-M were significantly reduced ($p < .01$) after 10 to 15 hours of exposure/acupoint therapy during an intensive five-day treatment period. Participants included 11 combat veterans or family members. Nine had been diagnosed with PTSD and the other two exhibited symptoms of PTSD. Improvements held on one-month, three-month, and one-year follow-ups (Church, 2010). A 10-minute video that includes brief excerpts from four of these treatments and of pre- and post-treatment interviews can be viewed at

http://www.vetcases.com and may be a useful reference for readers who are not conversant with exposure/acupoint protocols in the treatment of PTSD.

- Fifty adolescents who had been orphaned and traumatized twelve years earlier by the ethnic cleansing and warfare in Rwanda still exhibited symptoms of PTSD. Most were well above the cutoff for PTSD on two standardized measures, one a self-report inventory and the other an inventory completed by one of their caretakers at the orphanage. After a single imaginal exposure/acupoint session of 20 to 60 minutes combined with approximately six minutes learning two relaxation techniques, the average scores on both measures were substantially below the PTSD cutoff ($p < .0001$ on each). Interviews with the adolescents and their caretakers indicated dramatic reductions of symptoms such as flashbacks, nightmares, bedwetting, depression, withdrawal, isolation, difficulty concentrating, jumpiness, and aggression. On post-tests one year later, scores on both inventories held. Follow-up interviews also showed that the improvements persisted (Sakai, Connolly, & Oas, in press).

- Seven veterans (four who had been deployed in the Iraq war, two in Vietnam, and one who suffered from PTSD after sexual assaults) completed a well-validated pre-treatment inventory that detects the presence and severity of a range of psychological symptoms. Following six exposure/acupoint treatment sessions focusing on combat and other traumatic memories, the severity of symptoms decreased by 46% ($p < .001$) and the PTSD scores decreased by 50% ($p < .016$). Gains were maintained at three-month follow-up (Church, Geronilla, & Dinter, 2009).

- Twenty-nine low-income refugees and immigrants living in the United States were categorized as having the symptoms of PTSD based on exceeding a cut-off score on the civilian Postrauamtic Checklist-C (PCL-C). After one to three exposure/acupoint sessions, their PCL-C scores showed significantly less avoidance behaviors ($p < .05$), intrusive thoughts ($p < .05$), and hypervigilance ($p < .05$) than prior to treatment (Folkes, 2002).

Need for a Plausible Explanatory Model

These preliminary findings invite speculation on the mechanisms that underlie such apparently rapid and favorable clinical outcomes. Each of the treatments described above utilizes a common though unconventional procedure that is appearing in a variety of clinical formats, with "Thought Field Therapy" (TFT), the "Tapas Acupressure Technique" (TAT), and the "Emotional Freedom Techniques" (EFT) being among the most widely practiced. Although each has its own distinguishing protocols, they share as their presumed core active ingredients brief psychological exposure with simultaneous acupoint stimulation. These approaches are collectively referred to as "energy psychology" (Gallo, 2005) in a nod to the principles of acupuncture and other healing traditions that work with the body's "energy systems."

The field of acupuncture is based on the premise that stimulating specific points on the skin produces a favorable impact on the body's "vital energies" and, consequently, on physical and emotional health and well-being (Stux, Berman, & Pomeranz, 2003). The World Health

Organization (2003) has identified approximately two dozen medical or psychological conditions where evidence strongly supports the efficacy of acupuncture and several dozen more where the evidence is promising but still inconclusive. Although some investigators are not persuaded that the active ingredient of acupuncture is more than placebo (e.g., Ernst, 2006), the American Academy of Medical Acupuncture (http://www.medicalacupuncture.org) has more than 1600 physicians in its membership and publishes one of several peer-reviewed acupuncture journals in the United States. Although the procedures used in energy psychology do focus on acupuncture points, the approach has been more closely associated with acupressure since energy psychology relies on manual stimulation rather than on the use of needles. Acupressure protocols for a range of emotional conditions have been developed (e.g., Gach & Henning, 2004), and several RCTs have shown the stimulation of traditional acupuncture points to be superior to otherwise identical procedures that instead used "sham" points for reducing anxiety and pain (e.g., Barker et al, 2006; Kober et al., 2002; Lang et al. 2006; Wang et al., 2007). A survey of evidence that included a literature review of 45 peer-reviewed studies published since 2000 found at least preliminary support for the efficacy of acupressure with a majority of the conditions for which the World Health Organization found acupuncture to be effective (Natural Standard and Harvard Medical School, 2008).

Energy psychology has, however, been highly controversial within the mental health field, in part because its explanatory models often do refer to "energies" (e.g., Gallo, 2005) or "thought fields" (e.g., Callahan & Callahan, 1996) that cannot be detected by standardized scientific instrumentation. After a review of theoretical and methodological problems in research on psychotherapies utilizing the stimulation of acupoints, Baker, Carrington, and Putilin (2009) concluded that a critical area for future research is to "delineate the mechanism of action that produces [the] observed efficacy" (p. 45). This paper attempts that by (a) examining the probable mechanisms in the success of conventional exposure therapies, (b) speculating on additional mechanisms that may be introduced by acupoint stimulation, and (c) comparing PTSD outcome data on conventional exposure protocols with outcome data on exposure/acupoint protocols.

Mechanisms in Conventional Exposure Treatments for PTSD

In Pavlovian fear conditioning, when a neutral stimulus (e.g., a tone) is paired with an aversive stimulus (e.g., an electric shock), the initially neutral stimulus itself will, after a number of pairings, cause a fear response even after the aversive stimulus is discontinued. Extinction occurs after the now conditioned stimulus is repeatedly presented without the aversive stimulus (the tone without the shock), based on the principle that a conditioned behavior that is not reinforced will not persist. Brain studies are consistent with these clinical observations, showing, for instance, that neuronal firing in the lateral nucleus of the amygdala that had been evoked by the conditioned stimulus diminishes over time when the conditioned stimulus is presented without an aversive stimulus (Repa et al., 2001). Although early formulations assumed that this was based on a weakening of associations, extinction is now generally thought to involve new learning rather than unlearning. Abundant evidence shows that "fear reduction does not involve the weakening of associations per se, but rather involves the formation of new associations [that] override the influence of pathological ones" (Foa & McNally, 1996, p. 339). This conclusion is based on findings that even after extinction, fear memories "could be reinstated under certain contextual cues . . . such as dysphoric moods and environmental distress" (p. 339), suggesting

that the fear associations are not erased but are rather supplanted by new associations. Thus a tone that had earlier been associated with an electric shock becomes, after repeated exposure to the tone with no electric shock, associated with the updated information that no aversive consequences follow the tone. This new learning supersedes the earlier conditioning, resulting in its extinction.

Alternative Theories

A competing or at least complementary theory focuses on the process of "memory reconsolidation" rather than extinction to explore how cues that had evoked intense fear can most effectively be neutralized. This is based on findings suggesting that when a memory has been brought back into awareness, it must again be consolidated (re-consolidated) into long-term memory (Garakani, Mathew, & Charney, 2006; Nader, Schafe, & LeDoux, 2000). Although consolidation—the process by which newly learned information is stored in memory—was previously believed to occur only at the time of the experience, a research program at New York University led by Joseph LeDoux has demonstrated that "consolidated memories, when reactivated through retrieval, become labile (susceptible to disruption) again and undergo reconsolidation" (Debiec, Doyere, Nader, & LeDoux, 2006, p. 3428). Rats conditioned with a tone-shock pairing received a single presentation of the tone along with a protein synthesis inhibitor. The tone lost its power to evoke fear behavior, suggesting that the protein synthesis inhibitor prevented the tone-shock association from being reconsolidated into memory (Nader et al., 2000). Speculation on clinical implications of such findings has focused on interventions (pharmaceutical and psychotherapeutic) that could be introduced during the period that the memory is retrieved and labile (e.g., Debiec & LeDoux, 2006; Kindt, Soeter, & Vervliet, 2009; Monfils, Cowansage, Klann, & LeDoux, 2009; Schiller et al, 2010). Although subsequent research revealed limitations in the model (for instance, Milekic & Alberini, 2002, found that the more time that elapses between initial conditioning and memory reactivation, the less the impact of the chemical intervention), several lines of investigation are integrating the exposure and the memory reconsolidation models (e.g., Mamiya et al., 2009; Monfils et al., 2009; Schiller et al., 2010). Monfils et al. were able to prevent the spontaneous return of the fear response in rats by integrating the two approaches, and Schiller et al. produced similar results with humans.

Ruden (2005; in press) has identified electrochemical mechanisms (involving receptor pathways and electrical potentials in the amygdala) by which certain interventions, such as those used by the Monfils and Schiller teams as well as those used in energy psychology, appear to permanently eradicate links between the conditioned stimulus and the fear response. This formulation challenges conventional thinking. If verified, the earlier commonsense explanation, that extinction involves a weakening or elimination of old associations, may need to be revived. That account, as described earlier, was abandoned because of the spontaneous return of symptoms following extinction. Instead, extinction came to be seen as a case of new associations *overriding* maladaptive associations (Foa & McNally, 1996). These old associations stay intact, even after the exposure treatment suppresses them, and can be reactivated.

With reports of treatments that prevent spontaneous return of the fear response (e.g. Monfils et al., 2009; Schiller et al., 2010), it may be that exposure treatment can lead to (a) a complete depotentiation (eliminating long-standing signal transmissions between neurons) of

conditioned fear pathways (e.g., Kim et al., 2007; Ruden, in press) *or* (b) to overriding established fear pathways, which themselves stay intact. The exposure methods being used until recently simply may not have been capable of reliably depotentiating stimulus-response links and thus had to depend upon new learnings overriding old ones, leaving extinction vulnerable to reversal. If subsequent investigation verifies that certain non-drug protocols prevent spontaneous recovery of the fear response, this ability to more thoroughly eradicate maladaptive fear will hold important clinical implications.

The Central Role of the Amygdala in Fear Conditioning

Citing limitations to existing explanatory models for the efficacy of exposure therapies, Tryon (2005) suggested that exposure therapies work based on complex "memory mechanisms that learn" (p. 78). The central role of the amygdala as a "memory mechanism that learns" in fear conditioning is well established. As summarized by McNally (2007), "information about the CS [conditional stimulus] and the US [unconditioned stimulus] converge within the lateral nucleus of the amygdala, and output from this structure prompts expression of the behavioral indicants of fear" (p. 756).

Much is yet to be established regarding the neural mechanisms of the amygdala's threat response (LeDoux, 2007), but basic sequences can be outlined. Sight, sound, touch, pain, movement, and taste impulses are sent to the thalamus (smell, the most primitive of the "at-a-distance" senses, is regulated by the olfactory bulb). The thalamus translates them into a form the cerebral cortex will be able to recognize and interpret and then selectively relays this information to various areas of the cortex (Jones, 2007). Such information may or may not, depending upon a variety of factors, be registered in the person's conscious awareness, yet it is amenable to cognitive processing (Smith & Bulman-Fleming, 2004).

In situations that are initially assessed as involving possible danger, however, the thalamus (or olfactory bulb in the case of smell) sends the related sensory information not only to the cerebral cortex but also directly to the amygdala (LeDoux, 2007). Most sensory information bypasses the amygdala, but in a process analogous to virus detection software, certain sensory patterns—such as the sudden entry of an object into one's visual field or a face expressing rage—are sent directly to the amygdala's lateral nucleus. Stress also puts the amygdala on alert via the release of norepinephrine by an area of the primitive brainstem called the locus ceruleus, one of the brain's primary producers of norepinephrine (Scaer, 2007). Sensory information can come to the amygdala directly from the thalamus (the *short path*, designed for a rapid response that bypasses conscious analysis or volition) or from the thalamus via the cerebral cortex, which first processes the information before selecting which sensory input to send to the amygdala (the *long path*, allowing for a cognitive and often conscious evaluation of possible danger).

When the thalamus or locus ceruleus sends a signal directly to the amygdala (short path), the amygdala, "standing at the center of threat assessment, recruits other key structures in determining the threat response" (Kent, Sullivan, & Rauch , 2002, p. 133), initiating a series of coordinated neurological events reminiscent of the operation of a military command center. Reciprocal connections allow the amygdala to send signals back to the thalamus in response to the possible danger, creating a feedback loop that sharpens sensory focus to aid in threat

evaluation. The amygdala activates the hippocampus—which plays a central role in the formation of episodic and narrative memory—to access outcome information from analogous past experiences, providing a fact-based context for evaluating the potential threat. The prefrontal cortex and other cortical areas, once they have processed the sensory data relayed by the thalamus (long path), send the amygdala information based on preconscious cognitive analysis as well as the person's conscious response to the sensory input. The amygdala's central nucleus stands ready to initiate, in an instant, a full-fledged fight-or-flight reaction. But a group of cells between the amygdala's assessment areas and its call-to-action area (the central nucleus) are activated to create a barrier that inhibits the threat response while the danger is still being assessed (Likhtik, Popa, Apergis-Schoute, Fidacaro, & Paré, 2008). Meanwhile, the paralimbic network is enlisted to aid in prioritizing among the multiple streams of information that flood the amygdala when the senses signal a potential threat (Kent et al., 2002). In monitoring these events, the prefrontal cortex is prepared to prompt the amygdala to instantly terminate the entire sequence if an absence of danger is established (Shin et al., 2004).

Mechanisms in PTSD that Complicate Treatment

In PTSD, this grand achievement of evolutionary psychology goes awry. Memories or cues that do not constitute immediate threat nonetheless trigger full-fledged threat responses. Because PTSD-related traumatic memories tend to be more disorganized than other memories, specific emotional and perceptual elements of the memory may become exaggerated and serve as triggers for a threat response (van der Kolk, 1996/2007, p. 282). From this strong link between memories of the experience and physiological responses similar to those that occurred during the trauma—where in effect the traumatic experience is continually reactivated rather than integrated—a widening range of symptoms ensues (Schillaci et al., 2009).

These symptoms are characterized by the "repetitive replaying of the trauma in images, behaviors, feelings, physiological states, and interpersonal relationships" (van der Kolk & McFarlane, 1996/2007, p. 7). Responses to these intrusions may include (a) behaviors intended to avoid triggers for trauma-related emotions, (b) attempts to control overwhelming emotions with medication, alcohol, or other drugs, and (c) a spontaneous generalized emotional numbing, as if to counter the prevailing vulnerability to sudden unanticipated hyperarousal. In addition to difficulties modulating physiological responses is a tendency to "respond preferentially to trauma-related triggers at the expense of being able to attend to other perceptions" (van der Kolk & McFarlane, 1996/2007, p. 10). Guilt and self-blame about having or not being able to overcome the condition often ensue, along with the "demoralization of chronic hyperarousal" and a "progressive disruption of the individual's underlying neurobiology" (McFarlane & Yehuda, 1996/2007, p. 158). These debilitating symptoms are frequently accompanied by comorbid conditions such as sleep disorders, panic disorders, major depressive disorders, or phobias, which "become increasingly autonomous in their pattern of recurrence" (McFarlane & Yehuda, 1996/2007, p. 169). With such a wide range of serious symptoms presenting with PTSD, finding the most effective focus for treatment can be a formidable clinical challenge.

Exposure Treatments Target the Nucleus of PTSD

At the causal core of these cascading symptoms was a traumatic experience that could not be integrated and that in turn became a disjointed memory that catalyzes further traumatization. The memory and associated cues continue to evoke a physiological response analogous to that caused by the original trauma. This failure to integrate a terrible experience empowers it to take on a life of its own. Self-help efforts, supportive counseling, talk therapy, cognitive restructuring, and other interventions that rely on higher cortical structures are often ineffective in resolving the trauma due to the simple physiological fact that the amygdala's ability to control the higher brain centers is much stronger than the ability of the higher brain centers to control the amygdala. Projections from the amygdala into areas of the brain that involve cognition are, for instance, far more numerous than projections from these areas into the amygdala (LeDoux & Phelps, 2008), so the source of psychic disruption in the primitive brain is not readily corrected by interventions that do not directly address deep brain structures. Abram Kardiner (1941), a pioneer in the understanding and treatment of PTSD, spoke of PTSD's physiological "nucleus" (cited in van der Kolk, 1996/2007, p. 217). Current understanding would suggest that the physiological nucleus of PTSD is in the neurological pathways that result in the amygdala initiating an acute fear state in response to memories or cues associated with the trauma (van der Kolk & McFarlane, 1996/2007). And this is exactly what exposure treatments target.

Psychological exposure, again the most effective element to have been identified in the successful treatment of PTSD, results in internal and external stimuli no longer producing maladaptive (i.e., evoked though threat is no longer present) fear. In exposure treatment, a stimulus that produces maladaptive fear is repetitively paired with information that danger is not present until the stimulus no longer evokes a fear response. A measure of successful treatment is, in fact, the ability to recall the memory without reliving the fear that was part of the original trauma. When the fear response has been extinguished, "many stimuli that were associated with fear through generalization no longer elicit fear" (Rothbaum & Foa, 1996/2007, p. 492). Decreases in anxiety and fear-based behaviors follow. Although the cascade of PTSD-related difficulties described above may still need attention, the effective treatment of physiological reactivity is, in fact, likely to "have widespread beneficial effects on the overall system and can secondarily decrease intrusions, concentration problems, numbing, and the ways victims experience themselves and their surroundings" (van der Kolk & McFarlane, 1996/2007, p. 17).

Principles Derived from Conventional Exposure Treatments with PTSD

Although the review by the IOM (Committee on Treatment of Posttraumatic Stress Disorder, 2008) concluded that exposure treatment is the only intervention for PTSD whose efficacy meets rigorous scientific standards, a number of isolated studies have found cognitive behavioral interventions to be as effective as psychological exposure (e.g., Paunovic & Öst, 2001). In practice, strategies combining cognitive behavioral and exposure techniques are often applied. Despite encouraging outcomes compared with supportive counseling, psychodynamic talk therapy, and pharmacological interventions, the limitations of exposure treatments alone or exposure treatments in combination with cognitive behavioral techniques must also be recognized. For instance, in a study that the IOM considered to be among the most robust of those it identified as demonstrating an effective treatment of PTSD, Monson et al. (2006) found that 15 of 30 combat veterans initially diagnosed with PTSD showed a reliable decrease in PTSD symptoms following a cognitive processing protocol that included exposure treatment. Fifty

percent, however, did not, and 60% still met the criteria for PTSD after treatment. In addition, while symptoms of reexperiencing and emotional numbing improved significantly with the cognitive/exposure protocol in comparison with a wait-list condition, other symptoms such as behavioral avoidance and hyperarousal "did not differentially improve" (Monson et al., 2006, p. 904). The IOM report, nonetheless, agreed with the authors that: "This trial provides some of the most encouraging results of PTSD treatment for veterans with chronic PTSD" (Monson et al., 2006, p. 898).

Exposure treatments that successfully produce extinction of targeted fears are also, as noted earlier, vulnerable to subsequent recurrence. Attempting to maximize positive outcomes and their resilience, clinical experience and research have been combined in deriving a number of principles and guidelines that are widely applied. A sampling of these include:

1. Brief exposure, as is used in systematic desensitization (10 to 15 seconds in each round of the protocol), may be effective for low levels of arousal, but not for highly distressing stimuli. In addition, a large number of sessions over an extended period of time is required for brief exposure to be effective even with low levels of arousal (Rothbaum & Foa, 1996/2007).

2. Prolonged exposure is in fact generally needed in the treatment of anxiety disorders, with 20 minutes often being required before the anxiety associated with a simple phobia begins to diminish and up to 60 minutes with agoraphobia (Foa, Steketee, & Rothbaum, 1989). For trauma scenes, up to 100 minutes of flooding (where anxiety-provoking triggers are presented in an intense, sustained form) were required before decreases in anxiety were reported (Keane, 1995).

3. Clients are required to "focus their attention on the traumatic material and . . . not distract themselves with other thoughts or activities" (Brewin, 2005, p. 272). In fact, allowing the client to shift away "from the most traumatic cues" is believed to be "no more effective in attaining extinction to the anxiety than past episodes of intrusive recall have been" (Lyons & Keane, 1989, p. 147).

4. Exposure works for fear and anxiety but does not seem effective in the treatment of guilt or other complex emotions that require higher order cognitive constructs (Foa & McNally, 1996).

Comparisons between these guidelines and principles and those based on experiences and outcome data with exposure/acupoint treatments result in some provocative contrasts.

Mechanisms Involved in Exposure/Acupoint Protocols

Reports from clinicians using exposure/acupoint protocols in the treatment of PTSD and other anxiety disorders (e.g., Ayers, 2008; Church, 2010; Church, Geronilla, et al., 2009; Church, Hawk, et al., 2010; Feinstein, 2008b; Sakai et al., in press) do not correspond with the principles and guidelines developed by practitioners using conventional exposure methods. Specifically (keyed to the above list):

1. Brief exposure combined with acupoint stimulation has been found to be effective with conditions that involve high as well as low levels of arousal, and a few rounds of brief exposure during a single therapy session are often able to uncouple the association between a stimulus and a maladaptive fear response.

2. Prolonged or repeated exposure was not required to obtain desired clinical outcomes.

3. The focus during the exposure sessions was not fixed but rather allowed to shift among traumatic memories and thoughts, beliefs, physical sensations, emotions, and expectations.

4. Guilt and other emotions that require higher-order cognitive constructs such as shame and grief have responded to the approach.

Practitioners of exposure/acupoint protocols are not alone in noting discrepancies between conventional exposure guidelines and clinical experience. As efficacy studies established EMDR as an effective treatment for PTSD (Spates, Koch, Cusak, Pagoto, & Waller, 2000), outcome data began to accumulate that were not consistent with the observations and guidelines derived from experiences with other exposure treatments (Rogers & Silver, 2002). Rogers and Silver note, for instance, that "previous research suggests that repeated brief exposures only result in fear decrement when stimulus intensity and arousal are both low. Yet EMDR uses very brief (20–30-s) exposures [even though] stimulus intensities are high, since clients are asked to start by focusing on the most distressing scene" (p. 49). Although the structure of EMDR is consistent with the basic definition of an exposure therapy in that it involves "systematic and repeated confrontation with phobic stimuli" (Craske, 1999, p. 107), the differences between clinical experience with EMDR and conventional formulations of exposure therapy were so substantial that EMDR's originator classified EMDR as an information processing therapy (the basic tenet of such therapies with PTSD is that symptoms arise when traumatic events are emotionally unresolved but can be eliminated by fully processing the memory) rather than as primarily an exposure therapy (Shapiro, 1995).

Although EMDR protocols are not concerned with acupoints, both EMDR and the acupoint protocols utilize such brief psychological exposure that their reported effectiveness with severe trauma would not be predicted by conventional formulations for exposure therapies. The mechanisms in EMDR are still not clear (e.g., Lee & Drummond, 2008), but it is among the most strongly supported modalities utilizing psychological exposure, with more than 100 peer-reviewed studies establishing its efficacy (listed on http://www.emdr.com/studies.htm), including the classic study by van der Kolk et al. (2007) showing that 76% of adult-onset PTSD patients were entirely asymptomatic six months after EMDR treatments.

The Contribution of Acupoint Stimulation to Conventional Exposure Strategies

Certain acupoints, when stimulated, are believed to send deactivating signals to the amygdala. Several preliminary studies support this premise. Acupoints appear to have distinctive electrical properties, as contrasted with other areas of the skin, including lower impedance,

leading to speculation that they are conduits for electrical signals into the body (Ahn et al., 2008). Needling a toe acupuncture point used in Traditional Chinese Medicine to treat eye disorders activated, as shown by functional Magnetic Resonance Imaging (fMRI), the occipital lobes of the brain (Cho et al., 1998). When the investigators needled non-acupoints that were 2 to 5 centimeters away from the vision-related points, activation in the occipital lobes was not observed. This demonstration of a correlation between acupoint stimulation and the activation of specific areas of the brain as anticipated by ancient acupuncture literature, published in the *Proceedings of the National Academy of Sciences,* gained considerable notice since there are no known neural pathways between the two areas that could account for the speed of activation shown by the fMRI. Langevin and Yandow (2002), however, subsequently demonstrated a strong correspondence between the pathways on which acupuncture points are situated, described as "meridians" in acupuncture theory, and interstitial connective tissue. They also found an 80% correspondence between the sites of acupuncture points and the location of intermuscular or intramuscular connective tissue planes, suggesting that "channels connecting the surface of the body to internal organs" (p. 257) are in the body's connective tissue. The implications of these findings are, as described by Oschman (2006), that the semiconductive properties of the body's connective tissue allow acupoint stimulation to rapidly send electromagnetic signals to specific areas of the body independent of the nervous system.

In a study of direct relevance to the action of acupoint stimulation in treating PTSD, conducted at Harvard Medical School, the needling of a particular acupoint on the hand (Large Intestine 4) produced prominent decreases of fMRI signals in the amygdala, hippocampus, and other brain areas associated with fear and pain (Hui et al., 2000). Subsequent studies by the same team led to the conclusion that "functional MRI and PET studies on acupuncture at commonly used acupuncture points have demonstrated significant modulatory effects on the limbic system, paralimbic, and subcortical gray structures" (Hui et al., 2005, p. 496). Further investigation provided "additional evidence in support of previous reports" that acupuncture is able to produce "extensive deactivation of the limbic-paralimbic-neocortical system" (Fang et al., 2009). Meanwhile, a series of reports using electroencephalogram (EEG) analysis to explore neurological effects of exposure/acupoint tapping (as contrasted with the traditional use of needles) showed normalized brain wave patterns upon activation of a traumatic memory that disrupted such patterns prior to treatment (Diepold & Goldstein, 2009), normalization of theta waves after claustrophobia treatments (Lambrou, Pratt, & Chevalier, 2003), and decreased right frontal cortex arousal in treating trauma following motor vehicle accidents (Swingle, Pulos, & Swingle, 2004), corroborated by pre-/post-treatment test scores. Together, these laboratory findings suggest that the stimulation of specific acupuncture points, with or without needles, can plausibly bring about precise, intended outcomes—such as the deactivation of an amygdala-based fear response to a specific stimulus.

Comparing Exposure/Acupoint Protocols with Other Exposure Treatments

The intended effect of exposure treatment is for a traumatic memory or cue that triggers an acute fear response to no longer trigger that response. The substantive procedural differences between the protocols being examined here and other exposure treatments are the use of acupoint stimulation and the shortened exposure times required for attaining the desired outcomes. Conventional exposure treatments produce their effects through the use of repeated or prolonged

exposure. Some approaches also introduce a physical intervention that is incompatible with the stress response during or immediately following exposure to produce reciprocal inhibition and, after memory consolidation, long-term counter-conditioning. This might seem a more powerful strategy than exposure with no psychoactive physical intervention, but conventional approaches for inducing reciprocal inhibition have not proven effective in instances involving severe trauma or arousal. In Wolpe's (1958) systematic desensitization, in fact, exposure rather than the other components of the approach has been shown to be its primary active ingredient (Tryon, 2005), and systematic desensitization was found to be less effective than other exposure protocols. Summarizing investigations of systematic desensitization with trauma victims, Rothbaum and Foa (1996/2007) note that although most of the studies showed some beneficial results, longer exposure treatments that involve "repeated reliving of the trauma in imagination" (p. 496) have been shown to be more efficacious in the treatment of PTSD.

A different order of reciprocal inhibition. Acupoint stimulation seems to generate a distinctly different action than the physical interventions employed in other exposure treatments that attempt to produce reciprocal inhibition. Rather than using a method such as progressive muscle relaxation or diaphragmatic breathing to produce a calm state after exposure to a triggering cue (Hazlett-Stevens, 2008), laboratory evidence suggests that the stimulation of specific acupoints sends deactivating signals directly to the amygdala (Fang et al., 2009). The lateral nucleus of the amygdala is at once activated by memories or cues involving the traumatic event and deactivated by the acupoint-generated signal. Such reciprocal inhibition is the antecedent of extinction and may also bring about the depotentiation of neurological pathways that were sustaining the fear response (e.g., Ruden, in press).

Empirical support. Early studies support this formulation. Unlike what would be predicted by the principles and guidelines developed by practitioners using conventional exposure methods, existing research and clinical reports suggest that brief exposure combined with acupoint stimulation can be effective with high arousal, that prolonged, multiple, or highly focused exposures are not required to extinguish a maladaptive fear response, and that related emotions such as guilt are responsive to the protocol (e.g., Church, 2010; Church, Geronilla, et al., 2009; Church, Hawk, et al., 2010; Sakai et al., in press). The speed by which the procedure can be conducted (each acupoint is stimulated for only a few seconds) allows numerous rounds of exposure/acupoint stimulation to be completed within a very short period of time, so multiple targets may be treated during a single exposure/acupoint session.

In the Rwanda orphanage study cited earlier (Sakai et al., in press), of 188 orphaned survivors of the ethnic cleansing, the 50 who were given the highest scores on a standardized PTSD inventory completed by their caretakers met the study's selection criteria. All 50 exceeded the PTSD cutoff score. The inventory was structured around *DSM IV* (American Psychiatric Association, 1994) criteria for PTSD, designed for parents or other caregivers, and translated into Kinyarwandan in a manner that was approved by the test designers. The inventory scores were corroborated by staff observations that these children suffered with enduring PTSD symptoms such as intrusive flashbacks, nightmares, difficulty concentrating, aggressiveness, bedwetting, and withdrawal during the 12-year period following the ethnic cleansing. After a single treatment session and brief relaxation training, only 6% of the adolescents scored within the PTSD range ($p < .0001$), and the staff reported dramatic observed decreases in PTSD symptoms. On one-year

follow-up, 8% scored within the PTSD range on the caregiver inventory. On a companion inventory administered directly to the orphans, 72% scored within the PTSD range prior to treatment, 18% scored within this range immediately after treatment ($p < .0001$), and 16% scored within the PTSD range on one-year follow-up.

Reports like this have actually compounded rather than attenuated the credibility problems for proponents of exposure/acupoint protocols (Feinstein, 2009). Beyond the field's difficulties in producing coherent explanatory models, such extraordinary claims have led to cognitive dissonance or outright dismissal in many conventionally trained clinicians. How can tapping on the skin possibly help overcome severe psychological disorders, no less account for the speed and power with which positive clinical results are being reported for challenging conditions? Yet a greater proportion of participants in the Rwanda study apparently benefited from the treatment and apparently showed stronger benefits than those in the IOM's most promising outcome studies. For instance, in the Monson et al (2006) investigation of cognitive behavioral therapy with exposure treatments, 60% (18 of the 30 participants) still met the criteria for PTSD after the treatment. In the Rwanda study, only 18% (on the self-inventory; and only 6% on the caregiver inventory) still exceeded the PTSD cutoff after a *single* exposure/acupoint session and brief training in two relaxation techniques.

The Rwanda outcomes are corroborated by accounts from other relief teams utilizing similar protocols in a variety of post-disaster situations (Feinstein, 2008b) as well as in studies with combat veterans. In the RCT by Church, Hawk, et al. (2010) cited earlier, for instance, 49 veterans received treatment (the wait-list group received treatment after the waiting period). All 49 exceeded the PTSD cutoff on the military version of the Post-Traumatic Stress Checklist prior to treatment while only 7 (14%) exceeded the cut-off after six one-hour sessions. In another single-session treatment using acupoint stimulation with traumatized orphans, also in Rwanda but conducted by an independent research team, decreases in symptoms as reflected by a standardized self-inventory were significant at the .0001 level (Stone, Leydon, & Fellows, 2009). In an RCT with 16 abused male adolescents in Peru, which like the Rwanda studies used only a single exposure/acupoint session, 100% in the treatment group ($n = 8$) went from above to below PTSD thresholds thirty days after treatment while none in the wait-list control group ($n = 8$) showed significant change (Church, Piña, Reategui, & Brooks, 2009).

Retraumatization and spontaneous recurrence. A concern in the use of exposure techniques is the risk of retraumatizing the client (Gaffney, 2009). Conventional protocols often depend upon the vivid activation of and extended focus upon traumatic memories (Rothbaum & Foa, 1996/2007), making retraumatization a potential risk (Chu, 1998). With exposure/acupoint protocols, only brief exposure is required. Acupoint stimulation also appears to provide rapid in-session relief of the distress. This seems to make retraumatization less of an issue with exposure/acupoint protocols. Several studies, in fact, have commented on the lack of retraumatization when painful memories were invoked during interventions that involved acupoint stimulation in individual treatments (Mollon, 2007; Schulz, 2009) as well as group formats (e.g., Church & Brooks, in press; Flint, Lammers, & Mitnick, 2005). Another concern following successful exposure treatment is the spontaneous recurrence of symptoms (McNally, 2007; Monfils et al., 2009). Because of the ease of application, exposure/acupoint clients are routinely taught to self-apply the method if symptoms recur (Feinstein, 2004). The purported

ability of acupoint stimulation to permanently depotentiate the neurological pathways between the conditioned stimulus and the fear response (Ruden, in press) may also mitigate against the spontaneous recurrence of symptoms. In any case, existing studies of exposure/acupoint treatments for PTSD (e.g., Church, 2010; Sakai et al., in press) have found therapeutic gains to be durable on one-year follow-up.

An Explanatory Model for Exposure/Acupoint Protocols

Two interrelated, testable propositions can be derived from the above comparisons between conventional and acupoint-assisted exposure protocols:

> *1. Stimulating specified areas of the skin while mentally accessing a traumatic memory or cue that triggers hyperarousal in the limbic system sends signals to areas of the brain that, with repetition, may within minutes allow the memory or cue to be accessed without triggering limbic hyperarousal.*
>
> *2. Systematically addressing PTSD-related traumatic memories and cues as above has the effect of reducing the occurrence of flashbacks, intrusive memories, hypervigilance, insomnia, nightmares, exaggerated startle responses, emotional numbing, concentration difficulties, and other* DSM IV *symptoms of PTSD.*

In the practice of energy psychology, acupoint stimulation is generally preceded by establishing rapport, identifying treatment goals, detecting and resolving ambivalence or internal resistance to achieving the treatment goals, and selecting memories or cues to be accessed during the acupoint stimulation based on their clinical salience and the client's readiness to address them (Connolly, 2004; Craig, 2008; Diepold, Britt, & Bender, 2004; Feinstein, 2004; Gallo, 2002; Mollon, 2008). The above propositions assume that the acupoint stimulation was applied within that context.

In brief, the explanatory model for the mechanisms in exposure/acupoint protocols in the treatment of PTSD derived from comparisons with conventional exposure treatments suggests that (a) exposure/acupoint protocols rapidly reduce limbic system hyperarousal caused by traumatic memories and cues via deactivating signals sent to the amygdala, and (b) systematically neutralizing such memories and cues in this manner can result in a rapid and lasting resolution of many PTSD symptoms and render others more amenable to treatment.

Unresolved Issues

This explanatory model attempts to integrate clinical and neurological data into a plausible explanation for the reported reduction of PTSD symptoms when acupoint stimulation is combined with the mental activation of traumatic memories or cues. Several issues, however, remain unresolved:

The role of acupoint stimulation in regulating the limbic system. Research showing that the stimulation of acupoints sends deactivating signals to the limbic system is based on relatively few acupoints with relatively few subjects and is still in a preliminary phase. Even if these early

findings are confirmed by further research, it is yet to be established that the *manual* stimulation of specific acupoints sends signals to the limbic system that are equivalent to those sent with the use of acupuncture needles. A recent double blind study comparing penetration by acupuncture needling with non-penetrating pressure did, however, find equivalent clinical improvements for each intervention (Takakura & Yajima, 2009), and the acupressure outcome studies cited earlier are also suggestive of an equivalency between the effects of acupressure and acupuncture.

Are auxiliary methods active ingredients in exposure/acupoint protocols? A number of approaches which are described quite differently by their proponents (e.g., TFT, EFT, and TAT) use the stimulation of acupoints during brief psychological exposure. Are such approaches essentially equivalent, or do their auxiliary procedures play an active role (or *the* active role) in the reported outcomes? Also complicating attempts to identify active ingredients is the fact that acupoint tapping is most often applied as a tool within a broader clinical framework. Its purpose is usually to redress neurological patterns, such as elevated affect or maladaptive conditioning, rather than to serve as an independent, self-contained psychotherapy (Feinstein, 2009).

Explanations focused on reduced limbic system arousal do not address possible complementary mechanisms or the range of clinical outcomes reported following exposure/acupoint protocols. The model presented here was developed to explain how exposure/acupoint stimulation in the treatment of PTSD may affect established neural pathways, but it does not attempt to address possible complementary mechanisms. For instance, based on the first marker of PTSD that has been detected using existing instrumentation—the measurement of the brain's magnetic fields—an objective diagnosis of PTSD is now possible (Georgopoulos et al., 2010). This formulation would seem consistent with the earliest explanations used by proponents of energy psychology, which postulated disturbances in the brain's energy fields (Callahan & Callahan, 1996). The model developed here is also too limited to explain reported outcomes with diagnoses other than PTSD. A small number of studies (e.g., Brattberg, 2008; Church & Brooks, in press) and a plethora of anecdotal accounts (more than 2,000 such accounts can be found at http://www.emofree.com) suggest effectiveness with a range of conditions such as phobias, generalized anxiety disorder, reactive depression, obesity, substance abuse, unrealized goals, and physical maladies. Although strong preliminary evidence exists for the deactivating effects of specific acupoints on conditioned fear, it is unknown whether other acupoints impact additional problematic emotions such as anger or jealousy, disinhibit positive emotions, or activate motivational centers (e.g., by triggering the release of dopamine). Traditional acupuncture theory would, however, predict such relationships (Gach & Henning, 2004; Kaptchuk, 2000).

The need for further research. Although systematic observation using standardized measures and a few RCTs supporting the efficacy of exposure/acupoint protocols in the treatment of PTSD are available, additional RCTs are needed (a) to firmly establish that the efficacy of the procedure is based on components that actually augment the empirically-established component of psychological exposure, and (b) so the favorable comparisons of acupoint protocols in relation to other exposure protocols that can be made based on preliminary studies (discussed above) can be confirmed or disconfirmed. Controlled comparisons with other PTSD treatments are also required to ascertain the relative advantages of each approach. In

addition, dismantling studies comparing the elements used in the various exposure/acupoint protocols would help establish the active ingredients in successful clinical applications.

Conclusions

PTSD is a debilitating condition that has proven resistant to most forms of psychotherapy (Johnson, Fontana, Lubin, Corn, & Rosenheck, 2004). Exposure techniques have had the greatest success in treating the disorder, yet conventional forms of psychological exposure typically fail to benefit a substantial portion of those who utilize them and sometimes cause retraumatization. Although CBT combined with psychological exposure is still considered the treatment of choice for PTSD (Bryant et al., 2008), "half of patients do not respond" (p. 555). In addition, as noted by Cahill, Foa, Hembree, Marshall, and Nacash (2006), "Despite all the evidence for the efficacy of exposure therapy and other CBT programs, few therapists are trained in these treatments and few patients receive them" (p. 597). Reasons for this presumably include the fact that prolonged, intense exposure is a disquieting and often arduous process which holds only mixed promise for problem resolution (Gaffney, 2009). Problems with conventional treatment approaches are reflected in a recent finding that of 49,425 veterans of the Iraq and Afghan wars with newly diagnosed PTSD, less than one in ten who sought care from facilities run by the Department of Veterans Affairs actually completed the treatment as recommended (Seal et al., 2010).

Preliminary evidence suggests that by combining acupoint stimulation with brief psychological exposure, PTSD symptoms and underlying neurological patterns may be targeted with unusual speed, power, and lasting effects while minimizing the likelihood of retraumatization. In the Church, Hawk, et al. (2010) study, 49 of 50 participants—all of whom had scored above the PTSD cut-off on a standardized measure before treatment—completed the 6-session protocol, with 86% scoring below the PTSD cut-off following the treatment. The treatment targeted traumatic memories or maladaptive stimulus-response pairings, one at a time, using brief imaginal exposure while simultaneously stimulating acupoints. The acupoint stimulation is believed to send deactivating signals to the amygdala and other brain structures, rapidly reducing hyperarousal and extinguishing threat responses to innocuous triggers. If clinical reports and early research evidence are confirmed, the combination of brief psychological exposure and acupoint stimulation may enhance the ability of psychotherapists to treat PTSD more rapidly and effectively.

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