EMDR in the Treatment of Chronic Pain

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Chronic pain can significantly diminish life quality, causing depression, anxiety, and sleep disturbances, and may lead to neuroplastic processes that influence pain modulation. The current study investigated eye movement desensitization and reprocessing (EMDR) treatment of 38 patients suffering from chronic pain with 12 weekly 90-minute sessions. A battery of self-reported questionnaires assessing quality of life, pain intensity, and depression level were administered pre- and posttreatment for objective outcome evaluation. The Structured Clinical Interview for DSM was administered at pretreatment to identify participants' personality traits that may influence pain perception. Patients showed statistically significant improvement relative to baseline after 12 weeks of EMDR treatment. Our findings suggest that EMDR is an effective tool in the psychological treatment of chronic pain, resulting in decrease pain sensations, pain-related negative affect, and anxiety and depression levels. We examine possible theories about the mechanisms by which EMDR achieves these effects. Results were consistent with the underlying EMDR premise that posits the important effect of emotions on pain perception.

Keywords: chronic pain; EMDR; pain modulation; neuroplastic processes

Chronic pain is a complex sensory and emotional experience that causes overwhelming negative effects in every aspect of life and personality (Fishbain, 2002; Harris, Morley, & Barton, 2003). According to a study by the World Health Organization, individuals who live with persistent pain are four times more likely to suffer from depression or anxiety than those without pain and more than twice as likely to have difficulties working and socializing (Gureje, Von Korff, & Simon, 1998; Katz, 1990). Some researchers conducted a survey in different countries to estimate the prevalence of pain at a determined period of time (Buskila, Abramov, Biton, & Neumann, 2000; Català et al., 2002; Gerdle, Bjork, Henriksson, & Bengtsson, 1994). Gerdle et al. (1994) found that 49% of a Swedish sample of 7,637 individuals reported current pain. Similarly, Buskila et al. (2000) observed that 44% of 2,210 individuals in a southern Israel sample indicated pain on the day they were interviewed. In a sample of 5,000 Spaniards interviewed by telephone, Català et al. (2002) found that 30% of respondents had pain the previous day. It is possible to estimate that nearly 40% of the sample evaluated were suffering from any kind of pain at the moment of the interview or in the previous days.

Undertreated pain also has an important impact on direct and indirect medical costs through lost workdays, medical treatments, drugs, surgery, physical therapy, and related expenses. The American Pain Society (2001) concluded that the cost impact of chronic pain was greater than that of cancer and heart disease combined. Despite advances in different pharmacological,

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physical, and surgical pain treatments, a significant number of patients continue to experience pain, disability, and psychological distress (Grant, 1998; Turk, 2003).

Theories of Chronic Pain

Advances in medical technology have allowed testing of revolutionary pain theories and have encouraged the development of different approaches to pain management. These include theories by Melzack and Wall (1965), Melzack and Casey (1968), Rome and Rome (2000), and others.

The gate control theory of Melzack and Wall (1965) and Melzack and Casey (1968) proposed that the brain is an active system that filters, selects, and modulates inputs through a neural network called neuromatrix. It comprises a widely distributed neural network that includes parallel somatosensory, limbic, and thalamocortical components that subserve the sensory-discriminative, affective-motivational, and evaluative-cognitive dimensions of the pain experience. This theory was supported by a number of functional imaging studies that observed the different components of pain processing (Apkarian, 1995; Coghill et al., 1994; Hsieh, Belfrage, Stoneelander, Hansson, & Ingvar, 1995). The sensory-discriminative component of pain originates in the dorsal horn and through the spinothalamic tract moves to the lateral thalamus and subsequently activates the secondary somatosensory cortex and primary somatosensory area. This lateral system processes the sensory-discriminative aspects of pain, such as quality and location. The medial system, which is related to the motivational-affective dimension of pain, processes mainly painful stimulus in the medial thalamic nuclei, the connected anterior cingulated cortex, prefrontal cortices, and limbic structures.

Rome and Rome (2000) proposed that repeated exposure to painful stimuli and/or traumatic experiences may induce a complex series of neuroplastic processes at corticolimbic levels that are able to transduce information coming from the inside one's own body or from the environment into cellular memory. Those previous traumatic or painful memories may result in an augmented pain response to future stimuli, even though these are not painful in nature.

Treatment of Chronic Pain

It has been hypothesized that individual treatment response is highly influenced by the interplay of motivational, emotional, and cognitive factors (Flor, Birbaumer, & Turk, 1990; Melzack & Cassey, 1968; Price, 1999; Turk, 2003). The recognition of the multidimensional aspects of pain has led to the development of several treatments that take into consideration the cognitive, affective, behavioral, social, and physical features of pain. These include cognitive-behavioral therapy (CBT), hypnosis, acupuncture, and biofeedback training.

Among the psychological treatments the most widely implemented is a combination of operant conditioning and CBT (Keefe, Dunsmore, & Burnett 1992; Turk, 2003). CBT approaches consider pain to be a multidimensional experience influenced by behavior, cognition, and emotion and consequently have prioritized behavioral and cognitive techniques to control emotion and improve coping abilities. Specifically, CBT introduces strategies to allow the patients to alter their thoughts of physical reactions to the pain sensation.

Hypnotherapy uses relaxation techniques and suggestions specially designed to alter the affective, cognitive, and discriminative dimensions of pain. In that way, hypnosis can be effective in modulating pain perception through mechanisms of distraction, dissociation, and reinterpretation (Price, 1999; Rainville, Bao, & Chretien, 2005).

Acupuncture is the practice of traditional methods according to the principles of ancient Chinese medicine, which considers that vital energy flows through a set of interconnected channels called meridians, following a circadian rhythm. Excess or deficiencies in the flow of energy are said to cause pain and discomfort. By inserting needles strategically along individual meridians or at their junctures, the acupuncturist attempts to balance the flow of energy throughout the body (Vincent & Richardson, 1986).

Biofeedback training is also used for pain management. It is postulated that feedback from muscles enables the individual to acquire control of muscle activity associated with medical problems. Patients are trained to observe one or more of their ongoing physiological processes, usually presented through electronic circuitry as a varying tone, light, or meter display, and to consciously try to decrease muscle tension to obtain pain relief (Flor, 2002a; Holroyd & Martin, 2000; Jesup, Neufeld, & Merskey, 1979).

Eye Movement Desensitization and Reprocessing

Eye movement desensitization and reprocessing (EMDR) is an integrative psychotherapy approach originally developed as a treatment for posttraumatic stress disorder (PTSD) (Shapiro, 1989, 1995, 1999, 2001, 2002).

Numerous randomized clinical trials have established its efficacy in PTSD treatment (Maxfield & Hyer, 2002). EMDR facilitates the expression of problematic emotional responses, in a controlled fashion, providing the conditions for new learning, resulting in the elimination of distressing symptoms. Bilateral stimulation and dual focus of attention are two of the mechanisms utilized in EMDR to modulate affect.

EMDR is based on the adaptive information processing model, which posits that past traumatic experiences are implicated in triggering present pathology represented by different symptoms, such as flashbacks, nightmares, physical sensations, and chronic pain (Shapiro, 1995, 2001). Another distinctive characteristic of Shapiro's model is her hypothesis of the relation between a premorbid traumatic event and chronic pain. Her model is consistent with the previously mentioned neurophysiological research related to sensitization processes and limbically augmented pain syndrome (Rome & Rome, 2000). It is possible that a distinct effect of EMDR treatment may be desensitizing the limbically augmented portion of the pain experience (Ray & Zbik, 2001).

EMDR is a rapid information-processing therapy in which the patient reprocesses traumatic or dysfunctional thoughts, feelings, and somatic perceptions. In the treatment of chronic pain, EMDR interventions seek to alter the patient's cognitive, affective, and somatic symptoms and to identify inner resources that can provide relief.

Although EMDR was originally developed for individuals who had experienced psychological trauma (Shapiro, 1989), the neurobiological similarities found in patients who suffered PTSD and chronic pain disorders (Nicosia, 1994; van der Kolk, 1994, 1995) encouraged several authors to undertake research that explored the utilization of EMDR in the treatment of chronic pain (Bergman, 1998; Grant, 1999; Grant & Threlfo, 2002; Schneider, Hofmann, Rost, & Shapiro, 2008; Shapiro, 1995, 2001; Wilensky, 2000).

Some case studies have provided preliminary evidence that EMDR may be a promising treatment for the chronic pain. Grant and Threlfo (2002) presented some individual case reports on different pain conditions that were successfully treated with EMDR. In a case study of EMDR treatment for severe phantom limb pain following amputation, the phantom limb pain was completely ablated after nine sessions, with the results maintained at 18-month follow-up (Schneider, Hofmann, Rost, & Shapiro, 2007). In another case, presented by Russell (2008), only four sessions of EMDR treatment led to elimination of pain, depression, and phantom limb tingling sensations. Other authors have also demonstrated EMDR's effectiveness for alleviation of painful phantom limb pain after amputation, with results maintained at follow-up (Schneider et al., 2008; Solvey & Solvey, 2006; Tinker, Wilson, & Becker, 1997; Wilensky, 2000).

EMDR treatment, integrated with diaphragmatic breathing and cranial compression, has been used to abort migraine headaches. The results of research conducted by Marcus (2008) with 43 individuals with migraine headaches showed a reduction and in some cases elimination of pain and a significant reduction in painkillers.

Current Study

The aim of the present study was to investigate the use of EMDR in the treatment of 38 patients suffering from chronic pain. EMDR was used to reprocess emotionally charged memories linked to traumatic events or painful memories. It was hypothesized that EMDR can produce changes in the emotional response to pain, thus alleviating the participants' symptoms.

Method

Participants

Fifty newly admitted patients from the FLENI Institute Pain Center were recruited, of whom 38 (32 women and 6 men) completed all 12 weekly 90-minute EMDR sessions. Of the 12 participants who did not complete treatment (4 men and 8 women), 4 discontinued therapy because they found it difficult to miss one working day to attend the clinic. Two women lived far away, and length of travel became a deterrent. One woman who was making great progress phoned at the sixth session to postpone treatment until she obtained her anticipated disability pension (secondary gain). The other five participants dropped out without explanation. Medications used are tracked in Table 1.

Participant selection required first ruling out any prior history of mental retardation, substance abuse, or systemic disease affecting the central nervous system. Detailed history and thorough clinical examination was obtained in all patients by a neurologist, and chronic pain diagnosis was established following the International Association for the Study of Pain classification (Merskey & Bogduk, 1994). Of the 38 patients, 30 (79%) were suffering from headaches, 4 (10.5%) had fibromyalgia, and 4 (10.5%) neuropathic pain. The average length of illness was 12 years.

TABLE 1. Medication Administered Pre- and Posttreatment

Patient	Initial Drug Consumption	Final Drug Consumption
1	Baclofen	Topiramate
2	Amtriptyline	Propanolol
	Ibuprofen	Paracetamol (crisis)
	Sumatriptan	
	Metoclopramide	
	Dexamethasone	
3	Ergotamine	Paracetamol
	Cafeíne	Clonazepán
	Dipyrone	
	Clonazepam	
	Amitriptyline	
4	Ergotamine	Paracetamol
	Cafeíne	Metoclopramide
	Dipirone	Ergotamine
	Ergotamine	Paracetamol (crisis)
5	Cafeíne	
	Dipirone	
6	Zolmitriptan	Naratriptàn (crisis)
	Ergotamine	Verapamil
	Tramadol	Fluoxetin
	Naratriptan	
	Alprazolàn	
7	Ergotamine	Paracetamol
	Paracetamol	Salicilic acid
	Codeíne	
	Salicilic Acid	
	Amitriptyline	
8	Etoricoxib	
9	Ibuprofen (crisis)	
10	Sumatriptan	Propanolol
	Propanolol	Tolfenàmic
	Naratriptan	
11	Dextropropoxifen	Dextropropoxifen
	Ibuprofen	Ibuprofen
	Diazepam	Diazepam
	Ranitidine	Ranitidine
	Chlorpromazine	
12	Propranolol	Amitriptyline
	Amitriptyline	
13	Paracetamol	Diclofenac
	Diclofenac	Clonazepam
	Clonazepam	
	Clonixinte Lysine	
14	Diazepam	Diclofenac
	Betamethazone	
	Diclofenac	
15	Topiramate	Topiramate
	Alprazolam	Alprazolam
16	Ergotamin	Ibuprofen (crisis)
	Cafeíne	Sumatriptan
	Dipirone	Dexamethasone
	Tolfenàmic acid	
	Naratriptan	
	Dexamethasone	
17	Carbamazepine	Carbamazepine
	Valproid acid	Valproid acid

(*Continued*)

Assessment

A battery of self-reported questionnaires assessing quality of life, pain intensity, and depression level was administered at the beginning and at the end of treatment for objective outcome evaluation. The personality traits interview was administered only at the beginning with the purpose to identify participants' personality traits that may influence pain perception or pain behaviors. It was not our purpose in the present study to correlate changes in psychological symptoms, physiological pain, and psychological traits, although it would be an interesting issue for future research. Questionnaires applied included the following.

Short-Form Health Survey. The Short-Form Health Survey (SF-36; Ware, 1993) is one of the most widely used instruments to assess health status. It contains 36 items that yield eight domains. Physical Functioning (10 items) assesses limitations in physical activities, such as walking and climbing stairs. The Role-Physical (four items) and Role-Emotional (three items) domains measure problems with work or other daily activities as a result of physical health or emotional problems. Bodily Pain (two items) assesses limitations due to pain, and Vitality (four items) measures energy and tiredness. The Social Functioning domain (two items) examines the effect of physical and emotional health on normal social activities, and Mental Health (five items) assesses happiness, nervousness, and depression. The General Health perceptions domain (five items) evaluates personal health and the expectation of changes in health. All domains are scored on a scale from 0 to 100, with 100 representing the best possible health state. The scores are standardized with a mean of 50 and a standard deviation of 10.

The eight scales are hypothesized to form two distinct higher-ordered clusters due to the physical and mental health variance that they have in common. Three scales (Physical Function, Physical Role, and Bodily Pain) correlate most highly with the physical component and contribute most to the scoring of the Physical Component Summary measure. The mental component correlates most highly with the Mental Health, Emotion Role, and Social Function scales, which also contribute most to the scoring of the Mental Component Summary measure. The other scales have noteworthy correlations with both components.

State-Trait Anxiety Inventory. The State-Trait Anxiety Inventory (STAI) is suitable to differentiate a state anxiety caused by a specific event from an anxious personality. It is a self-report assessment device that

TABLE 1. (*continued*)

Patient	Initial Drug Consumption	Final Drug Consumption
	Clonazepam	Clonazepam
	Paracetamol	Paracetamol
	Diclofenac	Diclofenac
	Amitriptyline	Amitriptyline
18	Chlorpromazine	Amitriptyline
	Amitriptyline	Sertraline
	Sertraline	
19	Ibuprofen	Paracetamol (crisis)
	Clonazepam	
20	Sumatriptan	Paracetamol
	Diazepam	
	Clonazepam	
21	Amitriptyline	
22	Diazepam	Diazepam
	Topiramato	
23	Baclofeno	Baclofeno
	Metoclopramide	Metoclopramide
	Chlorpromazine	Chlorpromazine
24	Ibuprofen	Ibuprofen
	Ergotamine	Paracetamol
25	Dipirone	
	Cafeína	
26	Naratriptan	Paracetamol
	Amitriptyline	Amitriptyline
27	Paracetamol	Paracetamol
	Amitriptyline	
28	Ibuprofen	Paracetamol
	Metoclopramide	
29	Cloixilato de lisina	Paracetamol
	Ergotamina	
30	Tramadol	Amitriptyline
	Amitriptyline	
31	Naproxen	Naproxen (crisis)
	Alprazolam	
32	Alprazolan	Paracetamol
	Metoclopramide	Metoclopramide
	Paracetamol	
33	Carbamazepine	Carbamazepine
	Pregabalin	Pregabalin
34	Paracetamol	Paracetamol
	Ergotamine	Cafeína
	Dipirone	
	Cafeína	
35	Amitriptyline	Ibuprofen
	Sumatriptan	Clonazepam
	Metoclopramide	
	Naproxen	
36	Ibuprofen	Ibuprofen
	Naproxen	
37	Metoclopramide	Metoclopramide
	Paracetamol	Paracetamol
	Cafeína	Dipirone (crisis)
	Dipirone	
	Ergotamine	
	Ergotamine	Paracetamol
38	Paracetamol	Metoclopramide
	Cafeína	
	Dipirone	

includes separate measures of state and trait anxiety. State anxiety reflects a "transitory emotional state or condition of the human organism that is characterized by subjective, consciously perceived feelings of tension and apprehension, and heightened autonomic nervous system activity" (Spielberger, Gorusch, & Lushene, 1983). It may fluctuate over time and can vary in intensity. In contrast, trait anxiety denotes relatively stable individual differences in anxiety proneness and refers to a general tendency to respond with anxiety to perceived threats in the environment.

Scores on the STAI have a direct interpretation: high scores on their respective scales mean more trait or state anxiety, and low scores mean less. Both percentile ranks and standard (*T*) scores are available for male and female working adults in three age-groups (19–39, 40–49, and 50–69).

Beck Depression Inventory. The Beck Depression Inventory (BDI; Beck, 1987; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a very useful aid in determining the presence and intensity of depression. It consists of a 21-item scale concerning each particular aspect of depression experience and symptomatology (mood, sense of failure, indecisiveness, work inhibition, and appetite). Each item contains four statements of graded severity expressing how a person might feel or think about the aspect of depression under consideration. The standard cutoffs are as follows: a score of 0 to 9 indicates that a person is not depressed, 10 to 18 indicates mild to moderate depression, 19 to 29 indicates moderate to severe depression, and 30 to 63 indicates severe depression. Higher total scores indicate more severe depressive symptoms.

Structured Clinical Interview for DSM. The Structured Clinical Interview for DSM (SCID-II) is a semistructured interview to diagnose personality traits, originally developed for diagnoses according to the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM;* 3rd ed., revised) and later updated for use with the *DSM* (4th ed.).

Visual Analogue Scale. The Visual Analogue Scale (VAS) is an analogue measure used to estimate pain severity. Participants rate current pain severity on a 10-cm horizontal or vertical line with the two endpoints intended to represent pain experience extremes labeled "no pain" and "worst pain," respectively.

Treatment

Participants received pharmacological treatment (Table 1) and EMDR; no other therapies were provided during the study. Pharmacological treatment was prescribed by the treating physician to address the specific needs of each individual.

EMDR treatment consisted of 12 weekly sessions lasting 90 minutes. The sessions were administered using a treatment manual for pain control (Grant, 1999) that was adapted from the basic EMDR trauma protocol developed by Shapiro (1995). The basic protocol addresses past events that set the ground for pathology, present triggers of disturbance, and future adaptative response. Grant's pain protocol differs from the basic protocol in that, from the very beginning, pain sensations become the focus of treatment. If it happened that the participant was not in pain, the focus was directed to the original cause of pain or the worst pain crises he or she remembered along with any additional traumatic information, such as special situations that surrounded the pain, medical interventions, losses, and so on.

During the preparation phase of EMDR, patients were offered an explanation about the effects of pain on the nervous system as a way of introducing them to the information-processing model. The EMDR process and probable effects were explained. Patients were taught some relaxation and visualization techniques to help them to handle any distress that might arise between sessions.

To identify the treatment target, participants were encouraged to describe their pain in the most sensual way, using all the descriptions and metaphors they consider useful. They were also asked to remember the first pain experience and any associated trauma (onset of the illness, accident, medical procedures, and so on) as well as the triggers related to their pain. For example, headaches are commonly associated with responsibility and distress. In addition, negative thoughts, feelings, and physical sensations were also identified. They were asked to rate the level of pain or disturbance associated with their condition on the 11-point Subjective Units of Disturbance Scale (where 0 = no pain and 10 = worst possible pain). After that, they were encouraged to make a positive statement about how they would like to feel in the future, the validity of which was rated on a 7-point Validity of Cognition Scale (where 1 = false and 7 = true).

Reprocessing started by focusing on present pain if the patient was feeling pain at the beginning of the session or by focusing on pain memories such as the onset of the illness or strong pain episodes in the past. In many cases of recurrent headaches, patients were asked to choose the worst crisis, such as when he had to be hospitalized to achieve pain relief or when he had to leave office before an important meeting because of the pain.

In all cases, the psychological impact of the illness, such as low self-esteem, depression, or any related stressful thoughts and fears about their future, was also targeted. At the end of each set of bilateral stimulation, patients were asked what they had noticed, and the responses given were the focus of the following set. Whenever they presented a positive response, they were instructed to notice that, and further sets of bilateral stimulation were introduced. That was repeated until a satisfactory degree of pain and emotional relief was reported. Patients were then assisted to focus on the positive changes of the sensation and to try to build an image or a metaphor representing that sensation; this was then reinforced with more sets of bilateral stimulation. Finally, patients were asked to imagine themselves in 5 years feeling better, recovered, or in a better condition. Whenever negative feelings appeared, reprocessing started again, focusing on the distressful material.

Statistical Methods

The Shapiro–Wilk test (Shapiro & Wilk, 1965) to assess normality was applied for differential variable analysis or vice versa to obtain positive differences. As for most variance analysis, normality was rejected, and a nonparametric method was applied: the Wilcoxon matched-pairs signed-rank test. Eight comparisons were made using the SF-36 scale, a Bonferroni correction was applied, and p values below .00625 were considered significant (Bonferroni, 1936). Software used was SPSS 12.0 and InfoStat (2005 version).

Results

Medication

Table 1 shows the medication used by participants at pretreatment and at posttreatment. The parameter taken regarding the analgesic medication was the number of pills consumed at the beginning and at the end of the treatment.

SF-36

Scores on the SF-36 are standardized in a general population, with a mean of 50 and a standard deviation of 10, with higher scores representing better health. Prior to treatment, participants had scores below 50 in five of the eight SF-36 domains. Scores on Role-Physical and Bodily Pain placed participants at the first percentile in a normal population (Table 2). After treatment, scores in all domains improved. In

TABLE 2. SF-36 Pre- and Posttreatment Scores, Median, and Range Values

Domain	Initial score Median (Range)	Final score Median (Range)	Significance
Physical Functioning	80 (0–100)	87.5 (30–100)	$p = .0027$
Role-Physical	0 (0–100)	75 (0–100)	$p < .0001$
Bodily Pain	26 (0–74)	52 (0–100)	$p < .0001$
General Health	57 (0–87)	64.5 (5–97)	$p = .0015$
Vitality	37.5 (0–60)	47.5 (10–80)	$p = .0003$
Social Functioning	37.5 (0–100)	68.7 (0–100)	$p < .0001$
Role-Emotional	33.3 (0–100)	100 (0–100)	$p < .0001$
Mental Health	46 (8–76)	60 (16–92)	$p = .0019$

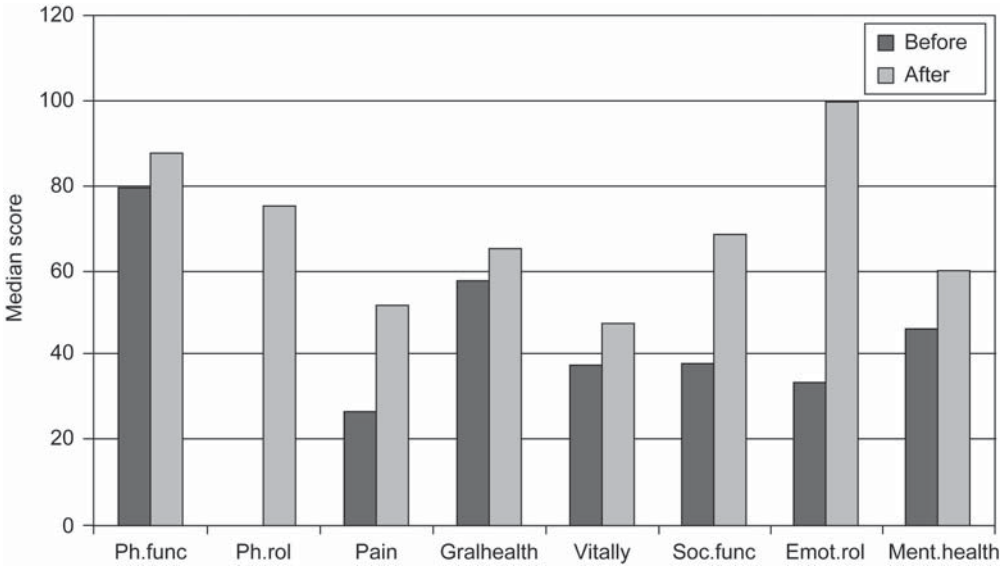

FIGURE 1. Pre- and posttreatment changes in SF-36 scores.

Note. Ph.func = Physiological Functioning; Ph.rol = Role-Physical; Gralhealth = General Health; Soc.func = Social Functioning; Emot.rol = Role-Emotional; Ment.health = Mental Health.

particular, Role-Physical and Role-Emotional showed the largest changes. The median Role-Physical score increased from 0 to 75, and the median Role-Emotional score increased from 33.3 to 100. As Figure 1 shows, participants scored higher in all domains after EMDR treatment, with statistically significant differences observed for all p values.

Symptoms of Depression, Anxiety, and Pain

At pretreatment, the median score of 17 on the BDI was in the mild to moderate depression range. At posttreatment, Beck scores decreased significantly ($p = .002$) (Table 3 and Figure 2). Similarly, scores on the STAI showed anxiety reduction in both state and general anxiety ($p < .001$) (Table 3 and Figure 3). Furthermore, following EMDR treatment, there was a significant reduction on pain levels ($p = .02$) as measured on the VAS (Figure 4).

TABLE 3. Pre- and Posttreatment Scores on the Beck Depression Inventory, State-Trait Anxiety Inventory, and Visual Analogue Scale

	Initial score Median (Range)	Final score Median (Range)	Significance
Depression	17 (1–30)	9 (0–22)	$p = .002$
Trait anxiety	65.0 (0–94)	51.5 (0–94)	$p < .001$
General anxiety	65.0 (0–94)	51.5 (0–94)	$p < .001$
Pain	8 (4–10)	6 (1–9)	$p = .002$

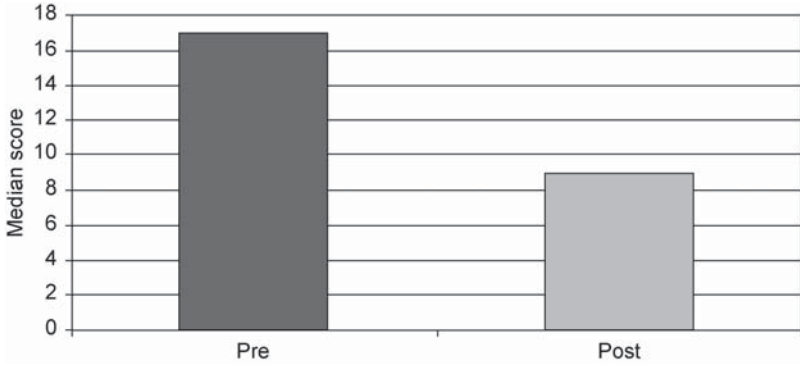

FIGURE 2. Pre- and posttreatment changes on the Beck Depression Inventory.

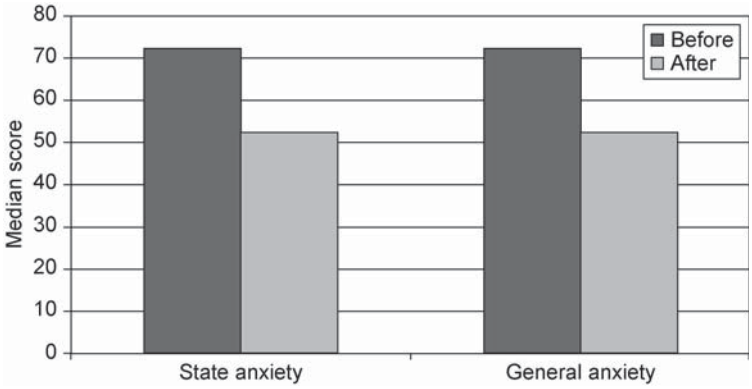

FIGURE 3. Pre- and posttreatment changes on the State-Trait Anxiety Inventory.

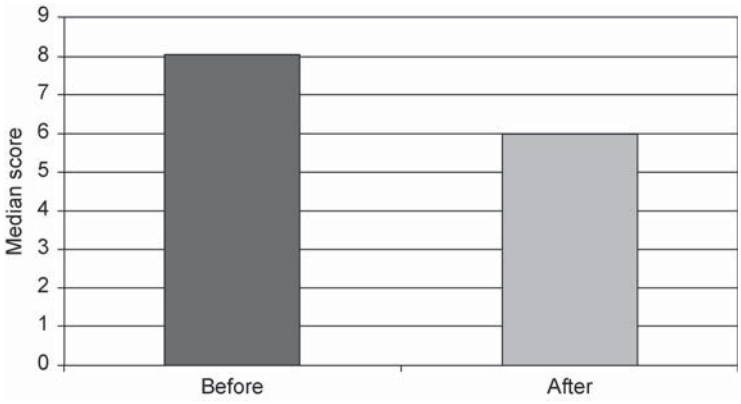

FIGURE 4. Pre- and posttreatment changes on the Visual Analogue Scale.

Personality Disorders

SCID-II results indicated that 73.7% of the patients evaluated in this sample fulfilled criteria for at least one axis II personality disorder, of which obsessive compulsive disorder was the most prominent (44.7%) (Figure 5).

Discussion

Treatment Effectiveness

Participants treated with EMDR showed statistically significant improvement relative to baseline after 12 weeks of treatment. These effects were evident in

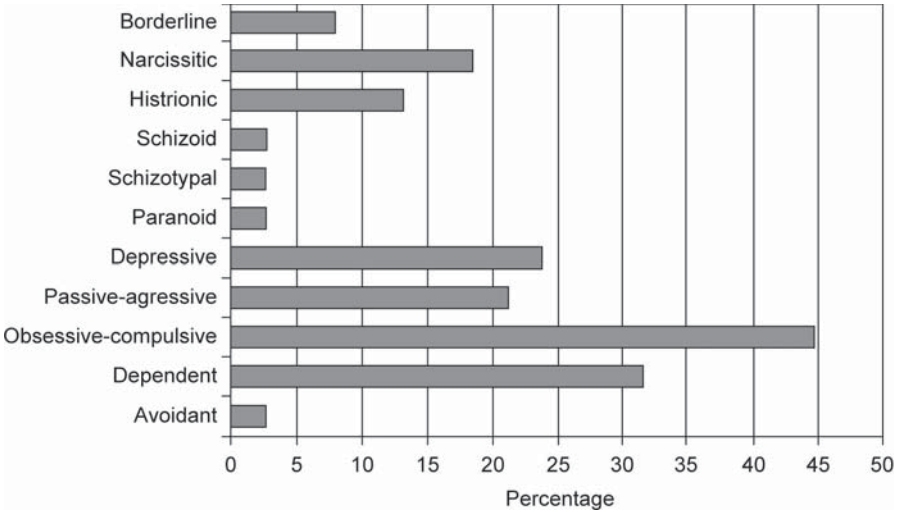

FIGURE 5. Prevalence of personality disorders.

the reduction of medication and symptom improvement. Within the population of 38 patients studied, we could observe a decrease in the consumption of medication of 79.49% (30 patients), while 20.51% (eight patients) showed no change (Table 1). Because of the diversity of analgesic medication taken by patients and the changes of medication made by them, these results have to be evaluated with certain precautions.

Scores on SF-36 improved considerably in all domains, as shown in Table 2, with Role-Physical and Role-Emotional the most outstanding. All domains are scored on a scale from 0 to 100 (with 100 representing the best possible health state) so that an increase in SF-36 subscale scores implies quality-of-life improvement in these areas. Significant improvement following EMDR treatment was also evident in reduced scores of depression, state anxiety, trait anxiety, and pain severity.

Of the 38 patients studied in the present sample, 73.7% met criteria for at least one axis II personality disorder as determined by a SCID-II assessment. These results support previously published research (Fishbain, Cutler, Rosomoff, & Rosomoff, 1998; Fishbain, 2002) that have linked pain to psychological comorbidity. Obsessive-compulsive personality disorder was the most strongly represented category, with characteristics of perfectionism, rigidness, and obsessiveness. These qualities are often seen in headache sufferers, who constituted most of the chronic pain patients evaluated in this research.

Implications for the Treatment of Pain

In the following section, we discuss two possible reasons for EMDR's effectiveness in the treatment of pain. First, we consider the relationship between pain and traumatic memories. Second, we speculate that EMDR may activate neurological mechanisms to reduce pain.

Processing Pain and Trauma

It has been widely accepted that pain is comprised of nociceptive input and emotional reactions that influence the patient's psychological welfare and exacerbate unpleasantness, helplessness, anxiety, depression, pain perception, and pain intensity (Hadjistavropoulos & Hadjistavropoulos, 2000; Rainville et al., 2005). When this condition is chronic, the constant feeling of pain, fatigue, and distress becomes a traumatic experience where the source of danger resides in the body (Shapiro, 1995). Clinical observations and neurobiological evidence suggest that chronic pain disorders and PTSD share important similarities, such as kindling mechanisms, which have been considered in the pathophysiology of both conditions. Those traumatic memories created under stressful conditions can continue to affect a patient even after the disease has been treated when the augmentation of the affective component of the pain signal triggers recall of previous like-stored memory traces (Geisser, Roth, Bachman, & Eckert, 1993; Lenz, 1998; Lenz et al., 1997; Rome & Rome, 2000). The amygdala, anterior cingulate cortex, prefrontal cortex, and hippocampus are the regions of the

central nervous system implicated in the experience of both trauma and pain (Bergman, 1998; Grant & Threlfo, 2002; Price, 1999).

According to Le Doux (1992, 1994) and van der Kolk (1994), this is due to the secretion of endogenous stress hormones occurring under severe stress conditions that overconsolidate traumatic memory storage and inhibit cognitive evaluation and semantic representation of the experience. Traumatic memories can be stored in sensorimotor modalities as somatic sensations and visual images and can initiate response memories without conscious participation. The recognition that unresolved traumatic memories may augment the emotional dimension of the pain experience further emphasizes the importance of psychological interventions in the treatment of pain.

The adaptative information processing model (Shapiro, 1995, 2001) that guides EMDR treatment posits that, as with traumatic experiences, chronic pain may be a result of unassimilated neurobiologically stored memories related to the source of the pain itself (accident, onset of the illness, and so on), the long-standing state of pain, medical procedures, or other unresolved distressing events (Bergman, 1998; Flor, 2002b, 2002c; Schneider et al., 2007; Shapiro, 1995, 2001). The model views chronic pain as involving not only the automatic emotional response to the pain sensation but also the somatic component of the stored memories. Cognitive, emotional, and perceptual dimensions of pain work in parallel and are interrelated. That means that positive changes in the patients' cognitive and/or affective experience can activate thalamospinal nociceptive inhibitory fibers that modify the sensory discriminative dimension of pain (Price, 1999).

EMDR treatment of chronic pain includes the processing and desensitization of both the automatic emotional response to the pain sensation and the somatic component of the stored memories related to the etiology of pain. It seems to have a direct effect on desensitizing the limbically augmented portion of the pain experience. Thus, it may reset the circuit breaker for emotion, allowing a more normal affective response to pain signals and to stressful events (Ray & Zbik, 2001).

In a successful EMDR treatment, it is possible to disengage the connections between traumatic memories and painful associations, making it possible to experience pain with less disturbing feelings and distress (Ray & Zbik, 2001; Shapiro, 1995, 2001; Welch & Beere, 2002). Clients are observed to learn from the past, change their negative cognitions and feelings of helplessness, and develop more adaptive strategies to improve their condition.

Possible Neurobiological Mechanisms

Another interesting support for the efficacy of EMDR comes from recent neuropsychological research on interhemispheric and intrahemispheric interactions during pain processing. Right-cerebral activation is reported in response to noxious stimuli and appears to be related to the right hemisphere's role in experiencing negative emotion and processing aversive events such as pain (Alvarez & Shpko, 1991; Chamandromouth, Kanchan, & Ambadevi, 1993; Coghill, Giltron, & Iadarola, 2001; Levin, Lazrove, & van der Kolk, 1999; Mollet & Harrison, 2006; Rauch et al., 1996).

Previous cognitive models of PTSD (Chemtob, Tolin, van der Kolk, & Pittman, 2000) also support the lateralization hypothesis. They have suggested that high levels of emotional arousal linked to overactivation of the right hemisphere interferes with adequate cognitive processing and augments threat expectancies. When left-hemisphere activation increases, there are more coping resources available, making reorganization of the traumatic experience in a more adaptative manner feasible (Gainotti & Zoccolotti, 1993; Tucker & Frederick, 1989; van der Kolk, 1994; Wittling, 1995).

Levin et al. (1999), Rauch et al. (1996), and van der Kolk (1996) speculate that EMDR may correct neurological abnormalities by stimulating both hemispheres and promoting an integration of hemispheric functioning as well as a normalization of brain activity. Using quantitative electroencephalography, Nicosia (1994) observed that after EMDR treatment, cerebral hemispheres were more synchronized and exhibited slower brain waves.

According to various authors (Bakan & Svorad, 1969; Bergman, 1998; Christman & Garvey, 2001; Davidson & Fox, 1994; Levin et al., 1999; Ray & Zbik, 2001; van der Kolk, 1994), EMDR treatment could help decrease or eliminate pain sensations by enhancing interhemispheric communication and cortical integration of traumatic memories. As a result, there could be a decrease in the negative emotional arousal with a concomitant reduction in hypervigilence that may also lead to a decrease in pain perception. In particular, Bergman (1998) hypothesized that EMDR results in increased activation of the anterior cingulate cortex and the left prefrontal area, enabling the capacity of

higher-brain functions to override input from limbic structures. This then is thought to facilitate limbic down-regulation, reduce kindling, and enhance integration of thalamic, amygdaloid, hippocampal, and cortical functioning.

Further, bilateral stimulation in EMDR has been found to induce a relaxation response (Bergman, 1998). It is likely that this allows the release of natural opiates, which, when combined with the inhibition of the amygdale, leads to the amelioration of symptoms and body sensations (Bergman, 1998; Grant & Threlfo, 2002).

Conclusions

The usefulness of EMDR has been investigated in the treatment of 38 patients suffering chronic pain during 12 sessions over a 12-week period. Treatment focused on desensitizing the emotional and somatic aspects of the pain experience.

In the present study, we found a general decrease in pain reports and medication intake. The SF-36 test results showed improvements in all domains with Role-Emotional improving the most. This finding is consistent with the underlying EMDR premise that posits the important effect of emotions on pain perception. Altering the emotional dimension of pain might have implications in the way it is perceived and reproduced within the patients' nervous system.

Our research on the application of EMDR with chronic pain patients demonstrated that EMDR can produce significant improvements within a limited number of sessions compared to mean average of 15 years of pain. Not only was a decrease in pain sensations and pain-related negative affect observed, but there was also an important decrease in anxiety and depression levels following EMDR treatment. As previously suggested by other authors (Ray & Zbik, 2001; Welch & Beere, 2002), EMDR may function by desensitizing emotional aspects of the pain experience, allowing the patient to separate painful somatic perception from emotionally linked memories and allowing changes in the way pain is perceived and remembered. Following EMDR treatment, a considerable increase in perceived ability to cope with pain was observed as well.

Limitations of the present study include the lack of a follow-up, which we acknowledge is an important issue. Other limitations include homogeneity with respect to gender (32 women and 6 men), pathology (79% headaches, 10.5% fibromyalgia, and 10.5% neuropathic pain), and the presence of comorbid personality disorder diagnoses. No administration of the SCID-II was conducted at the end of the treatment. Future research could assess whether the reduction of physical and psychological symptoms was also associated with a corresponding decrease in the diagnosis of personality disorders. Nevertheless, we hope our results will encourage further research of EMDR in the treatment of chronic pain. Our results suggest that this psychotherapeutic approach has the potential to be a useful intervention for the treatment of chronic pain patients.

References

Alvarez, W., & Shpko, S. (1991) Alexithymia and PTSD. *Journal of Clinical Psychiatry, 52,* 317–318.

American Pain Society. (2001). *Pain assessment and treatment in the managed care environment: A position statement from the American Pain Society.* Retrieved November 5, 2001, from http://www.ampainsoc.org

Apkarian, A. V. (1995). Functional imaging of pain: new insights regarding the role of the cerebral cortex in human pain perception. *Seminars in Neuroscience, 7,* 279–293.

Bakan, P., & Svorad, D. (1969). Resting EEG alpha asymmetry of reflective lateral eye movement. *Nature, 223,* 975–976.

Beck, A. T. (1987). *Beck Depression Inventory.* San Antonio, TX: The Psychological Corporation.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

Bergman, U. (1998). Speculations on the neurobiology of EMDR. *Traumatology, 4*(1), 1–13.

Bonferroni, C. E. (1936). Teoria statistica delle classi e calcolo delle probabilità. *Pubblicazioni dell' Istituto Superiore di Scienze Economiche e Commerciali di Firenze, 8,* 3–62.

Buskila, D., Abramov, G., Biton, A., & Neumann, L. (2000). The prevalence of pain complaints in general population in Israel and its implications for utilization of health services. *Rheumatology, 27,* 1521–1525.

Català, E., Reig, E., Artés, M., Aliaga, L., López, J. S., & Segú, J. L. (2002). Prevalence of pain in the Spanish population: Telephone survey in 5000 homes. *European Journal of Pain, 6,* 133–140.

Chamandromouth, R., Kanchan, B., & Ambadevi, B. (1993). Right left asymmetry in tonic pain perception and its modification by simultaneous contralateral noxious stimulation. *Neuropsychologia, 31,* 687–694.

Chemtob, C. M., Tolin, D. F., van der Kolk, B., & Pitman, R. (2000). Eye movement desensitization and reprocessing In E. A. Foa, T. M. Keane, & J. M. Friedman (Eds.), *Effective treatments for PTSD: Practice guidelines from the International*

Society for Traumatic Stress Studies (pp. 139–155, 333–335). New York: Guilford Press.

Christman, S., & Garvey, K. (2001). *Bilateral saccadic eye movements increase Stroop interference: A possible role of interhemispheric interaction.* Paper presented at the 31st annual meeting of the International Neuropsychological Society, Honolulu, HI.

Coghill, R. C., Giltron, I., & Iadarola, J. (2001). Hemispheric lateralization of somatosensory processing. *Journal of Neurophysiology, 85*(6), 2602–2612.

Coghill, R. C., Talbot, J. D., Evans, S. C., Meyer, E., Gjedde, A., Bushnell, M. C., et al. (1994). Distributed processing of pain and vibration in the human brain. *Journal of Neuroscience, 14,* 4095–4108.

Davidson, R. J., & Fox, N. A. (1994). Cerebral asymmetry and emotion: Developmental and individual difference. In D. Melfese & S. Segalowitz (Eds.), *Brain lateralization in children: Developmental implications* (pp. 191–206). New York: Guilford Press.

Fishbain, D. (2002). Pain and psychopathology. *Science, 3,* 763–775.

Fishbain, D. A., Cutler, B. R. B., Rosomoff, H. L., & Rosomoff, R. S. (1998). Comorbidity between psychiatric disorders and chronic pain. *Current Review of Pain, 2,* 1–10.

Flor, H. (2002a). The modification of cortical reorganization and chronic pain by sensory feedback. *Applied Psychophysiology and Biofeedback, 27*(3), 215–227.

Flor, H. (2002b). Painful memories. Can we train chronic pain patients to forget their pain? *EMBO Reports, 31*(41), 288–291.

Flor, H. (2002c). Phantom-limb pain: Characteristic, causes and treatment. *Lancet Neurology, 1,* 182–189.

Flor, H., Birbaumer, N., & Turk, D. (1990). The psychobiology of chronic pain. *Behavioral Research and Therapy, 12,* 47–84.

Gainotti, G., Caltafirone, C., & Zoccolotti, P. (1993). Left/right and cortical subcortical dichotomies in the neuropsychological study of human emotions. *Cognition and Emotions, 7,* 71–93.

Geisser, M. E., Roth, R. S., Bachman, J. E., & Eckert, T. A. (1993). The relationship between symptoms of posttraumatic stress disorder and pain, affective disturbance and disability among patients with accident and non accident related pain. *Pain, 66,* 207–221.

Gerdle, B., Bjork, J., Henriksson, C., & Bengtsson, A. (1994). Prevalence of current pain and their influences upon work and healthcare-seeking: A population study. *Journal of Rheumatology, 31,* 1399–1406.

Grant, M. (1998). *Pain control with EMDR.* Denver: Mentor Books.

Grant, M., & Threlfo, C. (2002). EMDR in the treatment of chronic pain. *Journal of Clinical Psychology, 59,* 1050–1502.

Gureje, O., Von Korff, M., & Simon, G. E. (1998). Persistent pain and well-being: A World Health Organization study in primary care. *Journal of the American Medical Association, 280,* 147–151.

Hadjistavropoulos, H., & Hadjistavropoulos, A. (2000). Health anxiety moderates the effects of distraction versus attention to pain. *Behavioral Research and Therapy, 38,* 425–438.

Harris, S., Morley, S., & Barton, S. (2003). Role loss and emotional adjustment in chronic pain. *Pain, 105,* 363–370.

Holroyd, K. A., & Martin, P. R. (2000). Psychological treatments for tension-type headache. In J. Olesen, P. Tfelt-Hansen, & K. M. A. Welch (Eds.), *The headaches* (2nd ed., pp. 643–649). Philadelphia: Lippincott, Williams and Wilkins.

Hsieh, J. C., Belfrage, M., Stoneelander, S., Hansson, P., & Ingvar, M. (1995). Central representation of chronic ongoing neuropathic pain studied positron emission tomography. *Pain, 63,* 225–236.

Jesup, B. A., Neufeld, R. W. J., & Merskey, H. (1979). Biofeedback therapy for headache and other pain: An evaluative review. *Pain, 7,* 225–269.

Katz, N., & Melzack, D. (1990). Pain memories in phantom limbs: Review and clinical observations. *Pain, 43,* 319–336.

Keefe, F., Dunsmore, J., & Burnett, R. (1992). Behavioural and cognitive behavioural approaches to chronic pain: Recent advances and future directions. *Journal of Consulting and Clinical Psychology, 60,* 528–536.

Le Doux, J. (1992). Emotions and the limbic system concept. *Concepts in Neuroscience, 2,* 169–199.

Le Doux, J. (1994). Emotion, memory and the brain. *Scientific American, 270,* 50–57.

Lenz, F. A. (1998). Painful stimulus evoke potential recording over the human anterior cingulate gyrus. *Journal of Neuropsychology, 79,* 2231–2234.

Lenz, F. A., Gracely, H., Zirh, A. T., Tomanoski, A. J., Staats, P., & Dougherty, P. M. (1997). The sensory-limbic model suggests testable hypotheses about the learned component of the affective dimension of pain. *Pain Forum, 6,* 41–43.

Levin, P., Lazrove, S., & van der Kolk, B. (1999). What psychological testing and neuroimaging tell us about the treatment of posttraumatic stress disorder by eye movement desensitization and reprocessing. *Journal of Anxiety Disorders, 13,* 159–72.

Marcus, S. (2008). An abortive treatment for migraine headaches. *Journal of EMDR Practice and Research, 2,* 15–25.

Maxfield, L., & Hyer, L. A. (2002). The relationship between efficacy and methodology in studies investigating EMDR treatment of PTSD. *Journal of Clinical Psychology, 58,* 23–41.

Melzack, R., & Wall, P. (1965). Pain mechanisms: A new theory. *Science, 150,* 971–979.

Melzack, R., & Casey, K. (1968). Sensory, motivational and central control determinants of pain: A new conceptual model. In D. Kenshalo (Ed.), *The skin senses.* Springfield IL: Charles C. Thomas.

Merskey, N., & Bogduk, N. (1994). *Classification of chronic pain* (2nd ed.). Seattle: IASP Press.

Mollet, G., & Harrison, D. (2006). Emotion and pain: A functional cerebral system integration. *Neuropsychological Review, 16,* 99–121.

Nicosia, G. (1994). *A mechanism for dissociation suggested by the qualitative analysis of electroencephalography*. Paper presented at the annual meeting of the EMDR International Association, Sunnydale, CA.

Price, D. (1999). Psychological and neural mechanisms of the affective dimension of pain. *Science, 288*(5472), 1769–1772.

Rainville, P., Bao, Q. V., & Chretien, P. (2005). Pain related emotions modulate experimental pain perception and autonomic responses. *Pain, 118*(3), 306–318.

Rauch, S. L., van der Kolk, B., Fisler, R. E., Alpert, S. P., Orr, S. P., Savage, C. R., et al. (1996). A symptom provocation study of posttraumatic stress disorder using positron emission topography and script driven imagery. *Archives of General Psychiatry, 53,* 380–387.

Ray, A., & Zbik, A. (2001). Cognitive behavioural therapies and beyond. In *Practical management* (3rd ed., pp. 189–208). Philadelphia: Lippincott.

Rome, H., & Rome, J. (2000). Limbically augmented pain syndrome (LAPS): Kindling, corticolimbic sensitization and the convergence of affective and sensory symptoms in chronic pain disorders. *Pain Medicine, 1,* 7–23.

Russell, M. C. (2008). Treating traumatic amputation-related phantom limb pain: A case study utilizing EMDR within armed services. *Clinical Case Studies, 7,* 136–153.

Schachter, S., & Singer, J. E. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review, 69,* 379–399.

Schneider, J., Hofmann, A., Rost, C., & Shapiro, F. (2007). EMDR and phantom limb pain: Theoretical implications, case study and treatment guidelines. *Journal of EMDR Practice and Research, 1,* 31–45.

Schneider, J., Hofmann, A., Rost, C., & Shapiro, F. (2008). EMDR in the treatment of chronic phantom limb pain. *Pain Medicine, 9,* 76–82.

Shapiro, F. (1989). Eye movement desensitization: A new treatment for post-traumatic stress disorder. *Journal of Behavior Therapy and Experimental Psychiatry, 20,* 211–217.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols procedures*. New York: Guilford Press.

Shapiro, F. (1999). Eye movement desensitization and reprocessing (EMDR) and the anxiety disorders: Clinical and research implication of an integrated psychotherapy treatment of traumatic memories. *Journal of Anxiety Disorders, 13,* 35–67.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols procedures* (2nd ed.) New York: Guilford Press.

Shapiro, F. (2002). *EMDR as an integrative psychotherapy approach: Expert of diverse orientations explore the paradigm prism*. Washington, DC: American Psychological Association Books.

Shapiro, S. S., & Wilk, M. B. (1965). An analysis of variance test for normality (complete samples). *Biometrika, 52,* 591–611.

Solvey, P., & Solvey, R. (2004). Tratamiento del miembro fantasma. *Emdria Latinoamérica Noticias, 7*(1), 1–3.

Solvey, P., & Solvey, R. (2006). Dolor en miembro fantasma. *Terapias de Avanzada, 25,* 89–105.

Spitzer, R. L., William, J. B. W., Gibbon, M., & First, M. B. (1990). *Structured clinical interview for DSM III-R*. Washington, DC: American Psychiatric Press.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58,* 67–75.

Tinker, R. H., Wilson, S. A., & Becker, L. (1997, July). *Treatment of phantom limb pain with EMDR*. Paper presented at the annual meeting of the EMDR International Association, San Francisco, CA.

Tucker, D. M., & Frederick, S. L. (1989). Emotion and brain lateralization. In H. Wagner & A. Manstead (Eds.), *Handbook of social psychophysiology* (pp. 27–70). New York: Wiley.

Turk, D., & Meichenbaum, D. B. (1989). A cognitive behavioural approach to pain management. In P. Wall & R. Melzack (Eds.), *Textbook of pain.*

Turk, D. (2003). Cognitive-behavioural approach to the treatment of chronic pain patients. *Regional Anesthesia and Pain Medicine, 28*(6), 573–579.

Turner, J., Jensen, M., Warms, C., & Cardenas, D. (2002). Catastrophizing is associated with pain intensity: Psychological distress, and pain related disability among individuals with chronic pain after spinal cord injury. *Pain, 98,* 127–134.

van der Kolk, B. (1994). The body keeps a score: Memory and evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry, 1*(5), 253–265.

van der Kolk, B. (1996a). *Trauma and memory: Psychobiological processes and therapeutic interventions*. Paper presented at Westmead Hospital, Sydney, Australia.

van del Kolk, B. (1996b). *Traumatic stress: The effects of overwhelming experience on mind body and society*. New York: Guilford Press.

Vincent, C. A., & Richardson, P. H. (1986). The evaluation of therapeutic acupuncture: Concepts and methods. *Pain, 24,* 1–13.

Ware, J. E. (1993). *S.F.36 Health Survey: Manual and interpretation guide*. Boston: Health Institute, New England Medical Center.

Welch, K., & Beere, D. (2002). EMDR: A treatment efficacy model clinical psychology and psychotherapy. *Clinical Psychology and Psychotherapy, 9,* 165–176.

Wilcoxon, F. (1945). Individual comparisons by ranking methods. *Biometrika, 1,* 80–83.

Wittling, W. (1995). Brain asymmetry in the control of autonomic physiologic activity. In R. J. Davidson &

K. Hugdahl (Eds.), *Brain asymmetry* (pp. 305–358). Cambridge, MA: MIT Press.
Worchel, S., & Brown, E. H. (1984). The role of plausibility in influencing environmental attributions. *Journal of Experimental Social Psychology, 20,* 86–96.
Wilensky, M. (2000). Phantom limb pain. *EMDRAC Newsletter, 4,* 2–3.

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