CLINICAL TRIALS

EMDR Reprocessing of the Addiction Memory: Pretreatment, Posttreatment, and 1-Month Follow-Up

Michael Hase
Reha-Zentrum Berliner Tor, Hamburg, Germany

Sabine Schallmayer
Hannover Medical School, Hannover, Germany

Martin Sack
Technical University, Munich, Germany

This randomized controlled study investigated the effects of eye movement desensitization and reprocessing (EMDR) in the treatment of alcohol dependency. EMDR was applied to reprocess the addiction memory (AM) in chronically dependent patients. The AM includes memories of preparatory behavior, drug effects (drug use), and loss of control (Wolffgramm, 2002). It is understood to involve extensive brain circuitry, drive part of conscious and unconscious craving, change environmental response at an organic level, and modify circuits that link to feelings of satisfaction, future planning, and hope. Thirty-four patients with chronic alcohol dependency were randomly assigned to one of two treatment conditions: treatment as usual (TAU) or TAU plus two sessions of EMDR (TAU+EMDR). The craving for alcohol was measured by the Obsessive–Compulsive Drinking Scale (OCDS) pre-, post-, and 1 month after treatment. The TAU+EMDR group showed a significant reduction in craving posttreatment and 1 month after treatment, whereas TAU did not. Results indicate that EMDR might be a useful approach for the treatment of addiction memory and associated symptoms of craving.

Keywords: EMDR; adaptive information processing; addiction memory; craving; treatment

Eye movement desensitization and reprocessing (EMDR) is a structured set of protocols and procedures for the treatment of posttraumatic stress disorder (PTSD) introduced in 1987 (Shapiro, 1989). This approach combines brief exposure to a traumatic memory with saccadic eye movements or other forms of alternating bilateral stimuli, for example, auditory or tactile. Over the years, rigorous research has demonstrated the efficacy of EMDR in the treatment of PTSD (Bradley, Greene, Russ, Dutra, & Westen, 2005), and it has consequently been recommended as an effective treatment for PTSD (APA, 2004; Bisson & Andrew, 2007; NICE, 2005).

The theory currently used to explain EMDR is called an adaptive information processing (AIP) model. The AIP model was developed to explain the rapid change toward positive resolution seen in the EMDR treatment process (Shapiro, 2001). The AIP model assumes "an inherent system in all of us that is physiologically geared to process information to a state of mental health" (Shapiro, 2002). The word "information" as used here refers to all external or internal input via all sensory systems, otherwise known as "experience." In EMDR, it is presumed that the AIP system leads to reduction in distress and/or negative emotions that can be stored as a result of upsetting experiences, leading to integrating upsetting information into a more adaptive, positive state. The AIP system may be hindered or blocked by trauma, other severe stress, or the influence of psychoactive drugs.

The primary goal of EMDR treatment is to gain access to and reprocess stored memories with a set of

 DOI: 10.1891/1933-3196.2.3.170

standardized protocols and procedures that include bilateral sets of rapid (saccadic) eye movements, auditory signals, or tactile stimulation. Eye movements have been shown in controlled studies to reduce affect and to increase attentional flexibility and the retrieval of episodic memory (Andrade, Kavanagh, & Baddeley, 1997; Barrowcliff, Gray, Freeman, & MacCulloch, 2004; Christman, Garvey, Propper, & Phaneuf, 2003). EMDR facilitates an association process that may further transform the dysfunctionally stored information and its integration within appropriate contextual memory networks (Shapiro, 1995; Stickgold, 2002).

Shapiro reported on the use of EMDR in the treatment of addictions early in its development (Shapiro, Vogelmann-Sine, & Sine, 1994). PTSD is frequently found to coexist with substance abuse (Brady & Sinha, 2005; Jacobsen, Southwick, & Kosten, 2001) formally known as either a "co-occurring" or "comorbid" disorder. In these cases the client meets full diagnostic criteria for PTSD and substance use disorder simultaneously.

According to Shapiro, the standard EMDR protocol for treating addictions involves reprocessing the earlier (traumatic) memories that set the basis for the dysfunction (including contributing elements to the development of addiction), the present triggers that activate disturbance, and the development of future templates for more adaptive behavior, which is essentially a form of relapse prevention for this population. Strategies for addressing specific targets related to the addiction are a valuable addition.

Other addiction-specific modifications of standard EMDR procedures have been proposed by Vogelmann-Sine et al. (1998) and Popky (2005). Omaha (2004) introduced an approach using bilateral stimulation while bypassing standard EMDR procedures. In Europe, a rationale for applying EMDR in addicted patients that more closely follows the EMDR standard protocol has been proposed. Anecdotal reports on clinical experience have been published (Hase, 2003, 2006).

Addiction Memory

Addiction memory (AM) is a useful concept for the "obsessive–compulsive craving" seen in drug-addicted patients. The addiction memory contains a general memory of loss of control and a drug-specific memory of drug effects. Activating this memory will lead to drug-taking behavior. The recall may be represented in consciousness as craving (Wetterling, Veltrup, & Junghanns, 1996).

The AM concept has gained growing acceptance in the field of addiction research and treatment (Boening, 2001) with regard to its importance in relapse and the maintenance of learned addictive behavior. Based on animal research, Heyne, May, Goll, and Wolffgramm (2000) suggest that the AM may be separate from other kinds of long-lasting consequences of drug experiences. They consider their theory compatible with Boening's observation from a clinician's point of view (Boening, 2001). Some characteristics of human addiction can also be found in a long-term learning model with rats proposed by Wolffgramm and colleagues (Wolffgramm, Galli, Thimm, & Heyne, 2000). Behavioral changes in drug-exposed animals indicate the memory formation that outlasts long periods of drug deprivation (Heyne et al., 2000; Heyne & Wolffgramm, 1998).

Several established animal models can reflect certain aspects of addiction in human beings. The "point-of-no-return model" seems to be particularly significant from our point of view. In the point-of-no-return model a free choice of plain water or drinking solutions containing alcohol or the drug to be tested (e.g., d-amphetamine or an opioid) is offered to drug-naïve rats. The animals first develop controlled consumption of the drug (e.g., alcohol). After several months some animals lose their control over drug intake, reflected by excessive consumption and changes in the pattern of activity (Heyne, 1996; Heyne & Wolffgramm, 1998; Wolffgramm & Heyne, 1995).

After a long period of forced abstinence, up to a third of the animal's lifespan, these animals again show an excessive and compulsive drug intake. Adulteration of the drug-containing solution using quinine, normally an aversive taste, reduces the consumption of the controlled consumers but not that of the excessive drinkers. Since this indicates a loss of control, these animals can therefore be regarded as addicted. The addicted rats show a preintake motor restlessness that may be related to craving and that, according to Wolffgramm (2002), may reflect the subconscious quality of such a memory.

Two studies demonstrated the animal models' validity for certain aspects of human alcohol addiction. Wolffgramm et al. treated alcohol-addicted rats with putative anticraving agents (the dopamine D2 receptor agonist lisuride and the D2 receptor antagonist flupentixol) and observed the effects on alcohol intake, alcohol seeking, and brain neurotransmission (Wolffgramm et al., 2000). Their investigations in the animal models paralleled clinical studies on alcohol-addicted humans. In both cases the results of the animal

model correctly predicted the procraving effect of lisuride and flupentixol in the human studies.

Force-feeding a drug in the animal model never leads to an addiction, and alterations in the neurochemical pathways reflecting the continuous presence of the drug in the animals' central nervous system do not differentiate between addicted and nonaddicted animals. Learning and memory formation seem to be an appropriate explanation for the development of addiction (Heyne et al., 2000). As noted previously, Heyne et al. (2000) suggest a separate memory of addiction from other kinds of long-lasting consequences of drug experiences. The memory of addiction develops on the basis of controlled drug consumption, as clearly demonstrated in the animal model. Consequently, a memory of drug effect and drug use must have been previously formed.

Slight differences in the formation of addiction memory with different drugs in the animal model lead to the assumption that the AM consists of both a memory of the specific quality of the addictive drug and a memory of loss of control. Wolffgramm considers this memory a result of an imprinting process and almost unextinguishable under normal circumstances (Wolffgramm et al., 2000). Wolffgramm proposes the idea of a memory network containing components of preparatory behavior, drug effects (drug use), and memory of loss of control. According to Wolffgramm internal or external cues can activate this memory network (Wolffgramm, 2002). Activating this memory will lead to drug-taking behavior. The recall may be represented in consciousness as craving. This seems to bear a striking similarity to Shapiro's AIP model (Shapiro, 2002).

Currently there is no proof for a particular pathological neurobiological structure containing the AM. However, the reward system of the brain, with its neuroanatomical structures, the anterior cingulate gyrus, and the amygdala, may be the structures involved in forming the AM (Hyman, 2005). Though the nature of craving and its role in the addictive process is debated (Tiffany, 1999; Tiffany, Carter, & Singleton, 2000), strategies to reduce craving by altering or extinguishing the AM could add an important component to well-established treatment modalities. Wolffgramm demonstrated this therapeutic option in the animal model where a reimprinting of the AM facilitated by steroids extinguished craving in opiate-addicted rats (Wolffgramm et al., 2000).

Reprocessing the Addiction Memory

The AM is presumed to be an episodic type of memory, and its cue-reactivity and power resemble the maladaptive traumatic memory formation at the core of PTSD (van der Kolk, Burbridge, & Suzuki, 1997). Activating this memory will lead to drug-taking behavior. Thus reprocessing the AM with EMDR should lead to measurable changes of addiction symptoms if the AM qualifies as maladaptive memory within the AIP model. Targets for reprocessing would most likely be memories of relapse or memories of intense craving, as these are likely to indicate an activated AM.

Since activating the AM may be represented in consciousness as craving, it is hypothesized that reprocessing the AM may lead to a reduction in craving. In this study we measured craving reduction with the Obsessive–Compulsive Drinking Scale (OCDS), an instrument that measures alcohol-related craving (Mann & Ackermann, 2000).

Method

Participants

Participants in this study were alcohol-addicted inpatients seeking detoxification treatment in a German regional psychiatric hospital. The inclusion criterion of the selected patients was reported craving for alcohol either at the beginning of treatment, after somatic detoxification, or as a cause for relapse prior to treatment. The patients were diagnosed according to the International Classification of Diseases in its 10th revision (ICD-10; Dilling, Mombour, & Schmidt, 1991). Exclusion criteria included current multiple drug use, continuous use of any drug of abuse in treatment, and organic mental disorders. A history of multiple drug use did not lead to the patient's exclusion.

A stratified randomization procedure was applied so that patients admitted to treatment in odd-numbered weeks (1, 3, 5, etc.) were assigned to treatment as usual (TAU), which was provided according to the standards of qualified detoxification treatment. Patients admitted in even numbered weeks (week 2, 4, etc.) were assigned to TAU plus two sessions of EMDR (TAU+EMDR). After assignment but prior to treatment patients gave informed consent and received additional information about the program and EMDR.

Patients were chronically addicted to alcohol, with the dependency lasting an average of 12.1 years in the TAU and 10.7 years in the TAU+EMDR group, respectively. The chronicity of their dependency shows in the average number of previous hospital treatments for detoxification: 12.8 with TAU and 11 with TAU+EMDR. The mean number of previous rehabilitation programs was 1.2 with TAU and 0.9 with TAU+EMDR. The two groups did not show a

statistically significant difference in addiction duration, number of previous treatments, or any other questionnaire measures.

Measures

Questionnaires were administered at pretreatment after the somatic detoxification, at posttreatment, at 1-month follow-up (by mail), and at 6-month follow-up (by mail).

The Münchner-Alkoholismus Test (MALT) provides a self-report and an interview portion. The MALT is a valid diagnostic instrument for diagnosing alcohol dependency (Feuerlein, Ringer, Kufner, & Antons, 1979; Gorenc, Bruner, Nadelsticher, Pacurucu, & Feuerlein, 1984).

The Mini-DIPS served as a second diagnostic tool for alcohol dependency (Margraf, 1994; Schneider et al., 2001). The Mini-DIPS is the short version of the Diagnostic Inventory of Mental Disorders (DIPS). The Mini-DIPS consists of a structured interview enabling the clinician to screen for mental disorders according to DSM-IV and ICD-10 criteria. The Mini-DIPS is an efficient diagnostic tool for anxiety disorders, affective disorders, somatoform disorders, substance abuse, and substance dependency (Margraf, 1994).

The Posttraumatic Stress Scale 10-Items (PTSS-10) was used to screen for PTSD (Eid, Thayer, & Johnsen, 1999). The Dissociative Experiences Scale (DES) and the Somatic Dissociation Questionnaire 5 Items (SDQ-5) were used to screen for dissociative type disorder. The DES is a widely used 28-item self-report scale that quantifies the frequency and intensity of a wide range of experiences indicative of dissociation (Bernstein, Carlson, et al., 1992; Bernstein & Putnam, 1986). The SDQ-5 is the 5-item version of the earlier 20-item version screening for somatoform dissociation (Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1997).

The Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the State-Trait-Anxiety Inventory (STAI; Spielberger, Gorsuch, & Luschene, 1970) served as measures for depression and anxiety to compare treatment as usual (TAU) and TAU+EMDR. The BDI is a 21-item self-report questionnaire widely used in research to evaluate cognitive and vegetative symptoms of depression. As with many EMDR treatment studies, the BDI was also administered pre- and posttreatment in this study. The STAI is a widely used 40-item measure with two scales designed to assess state-anxiety and trait-anxiety.

The OCDS was administered in the German version (Mann & Ackermann, 2000). The OCDS is a 14-item scale designed to assess perceived craving for alcohol (Anton, Moak, & Latham, 1996). The subscores "obsession" and "compulsion," as suggested by Anton and colleagues, were confirmed by principal component analysis but provide little additional information compared to the OCDS total score. The German version of the OCDS is consistent and reliable for alcohol-dependent patients with transsituational and global self-assessment of craving, at least during the time period in which it is administered.

The OCDS monitors craving in alcohol relapse prevention studies, testing pharmacological compounds (Chick et al., 2000). Items 7 and 8 on the scale ask for quantity and frequency of drinking. To avoid any bias in this study, items 7 and 8 were fixed at the time of the initial rating for posttreatment and follow-up assessment. This means that patients always had to answer these particular items in the same way at all points of assessment. The 7-day reporting period covers the days before admittance when patients reported the quantity and frequency of their drinking. At the end of 2 weeks of hospital treatment the participants would be assessed for a second time, and they would have to report abstinence just as an effect of hospitalization, thus lowering the OCDS scale.

Since continuous motivation is a well-known problem with addicted patients, follow-up examination was restricted to the OCDS and a short self-designed questionnaire asking for relapse and psychosocial support within the follow-up period. Patients were provided with the questionnaires and a stamped and addressed envelope at the termination of treatment. They were also instructed to fill out the questionnaires by the date on the back of the envelope and to post the envelope immediately afterward.

Procedures

Patients were recruited and assessment began after somatic detoxification was terminated. This was done for two reasons. First, to obtain informed consent, patients had to be sober and no longer under the influence of psychotropic medications, such as clomethiazol, which is used to combat severe alcohol withdrawal symptoms. Second, when first admitted to treatment, patients were normally under the influence of alcohol. Therefore craving could hardly be measured at that time.

Craving could not be measured under the influence of psychotropic medication during somatic detoxification either. This had the disadvantage that craving could only be initially assessed after admittance and may not reflect craving under real-life circumstances.

As there was no other way to obtain a valid initial measure, some variance had to be tolerated. This can be assumed for measures of depression and anxiety as well.

Treatment as Usual

All patients received TAU, which consisted of standard treatment for qualified detoxification and included detoxification from alcohol, motivational interviewing, assessment of social status and functioning, and addiction-focused group therapy. Relaxation and art therapy were also part of treatment as usual. If possible, patients were referred to a rehabilitation program after qualified detoxification, and contact with 12-steps groups was made. Two weeks was the standard TAU duration, but it could be extended to 3 weeks if necessary. Comorbid psychiatric conditions such as depression or anxiety disorders were treated in accordance with the patient's needs, including appropriate medication.

EMDR

The German version of the EMDR Institute Manual (Shapiro & Hofmann, 1994) served as the basis for EMDR treatment as applied in this study. Two 1-hour sessions of EMDR were provided to participants in the TAU+EMDR group during the second week of TAU. EMDR was modified in the following way: No special stabilization phase prior to reprocessing was included since the target for reprocessing was the AM and not a specific traumatic memory. EMDR requires targeting a certain memory for reprocessing, that is, bringing a certain memory to mind, identifying the worst part of the memory, identifying an image to elicit appropriate negative and positive cognitions, and accessing somatic sensations associated with the memory.

To target the AM, memories of relapse or of intense craving were chosen as target memories for reprocessing. The discomfort of the patient confronted with a memory of traumatic origin is usually measured by the Subjective Units of Disturbance (SUD) on a 0 to 10 scale. In this study, the "Level of Urge" (LoU) was monitored instead. This measure monitors the subjective experience of craving on a 0 to 10 scale. The first three patients in the study received three sessions of EMDR targeting a memory of relapse or intense craving. We observed a strong reduction in the OCDS score with the first three patients receiving EMDR, therefore the number of EMDR sessions was cut to two. This was done to test the efficacy of treatment under more rigorous conditions to help determine a minimum effective dosing level. Compared to the often recommended EMDR sessions lasting up to 90 minutes, treatment sessions for processing the AM were relatively short, with a duration of no more than 60 minutes.

Statistical Analyses

Chi-square tests were used to compare dichotomous variables. After testing for normal distribution, we used *T*-tests for independent groups to compare groups for baseline variables. Treatment effects were tested with GLM repeated measures with a factor by group design and age and gender as covariates. Throughout the study significance level was set at alpha = .05, and two-tailed analyses were carried out.

Results

Completion

Of the 34 patients in the study, 2 from the TAU+EMDR group were excluded because of continuous drug abuse while in treatment. Another 2 patients, both in the TAU group, dropped out during the assessment phase and terminated the treatment prematurely. Thirty patients completed the study. Eleven of the 15 patients completing TAU sent in their assessment measures at 1-month follow-up and only 2 replied at 6-month follow-up. Thirteen of the 15 patients completing the TAU+EMDR treatment group sent in their measures at 1-month follow-up, and 6 sent in their measures at 6-month follow-up (see Figure 1, participant flow chart).

Study Sample

Of the 30 patients who completed the study, 12 were female and 18 male (TAU: 5 female, 10 male; TAU +EMDR: 7 female, 8 male). All 15 patients from the TAU group were diagnosed as addicted to alcohol. In the EMDR group, 14 patients were diagnosed as alcohol-addicted and 1 was diagnosed as multiple-substance dependent, with alcohol being the identified drug of abuse. Ten patients qualified for comorbid psychiatric disorders in the TAU group compared to 12 patients in the TAU+EMDR group (see Table 1).

Baseline Measures

The TAU+EMDR and TAU patients did not differ statistically in any of the following variables: age, number of previous inpatient detoxification treatments, number of previous inpatient rehabilitation treatments, duration of addiction in years, DES, SDQ-5, PTSS-10, BDI, STAI-X1, STAI-X2, MALT, and the duration of current treatment in days (see Table 2). The mean pretreatment OCDS score did not signifi-

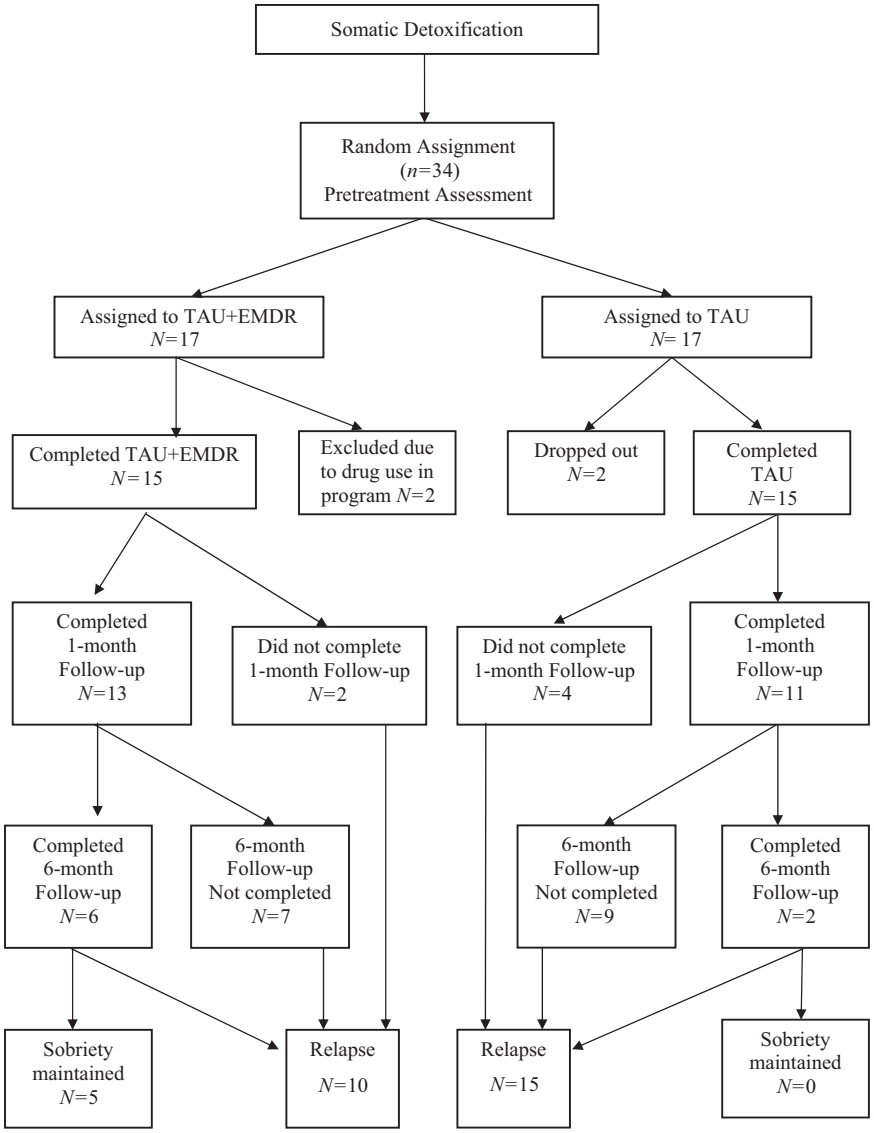

FIGURE 1. Flow of participants through the study.

TABLE 1. Comorbid Diagnoses in EMDR and TAU

ICD-10 F	32.0	32.1	32.2	40.1	41.0	43.1	43.21	60.31	60.80
EMDR	1	1	1	0	1	4	1	3	0
TAU	0	0	0	1	0	5	1	2	1

Note. ICD-10 F 32.0: mild depressive episode, 32.1: moderate depressive episode, 32.2: severe depressive episode, 40.1: social phobia, 41.0: panic disorder, 43.1: posttraumatic stress disorder, 43.21: adjustment disorder, 60.31: borderline personality disorder, 60.8: other specific personality disorders.

cantly differ in the TAU+EMDR (mean = 20.4, *SD* = 4.6) group compared to TAU (mean = 20.3, *SD* = 6.3).

Change From Pretreatment to Posttreatment

The hypothesis in this study was that reprocessing the AM might reduce cravings in alcohol-addicted patients; therefore the most relevant measure for analysis was the OCDS. Compared to pretreatment, posttreatment scores of OCDS revealed a significant improvement in the TAU+EMDR treatment group (9.5 *SD* 4.2, $T = 10.7$, $p < .001$), while only a small reduction in craving was noticed in TAU (18.7 *SD* 6.9, $T = 1.1$, $p = .29$). Between TAU+EMDR and TAU, the difference in OCDS scores posttreatment ($p < .001$) was statistically significant.

TABLE 2. Description of the Sample by Questionnaire Data

	EMDR $N = 15$		TAU $N = 15$			
	M	*SD*	*M*	*SD*	Test Value	Significance
Age (years)	45.7	5.2	42.5	8.5	1.314	.281
Duration (years)	10.7	7.4	12.1	7.4	0.198	.660
Number of detoxifications	11	14.4	12.8	17.1	0.097	.757
Number of rehabilitation treatments	0.9	0.8	1.2	0.9	1.159	.291
DES	9.9	7.4	12.9	8.3	1.108	.302
SDQ-5	5.7	1.4	6.7	3.4	1.083	.307
PTSS-10	25.9	13.8	25.3	9.8	0.019	.892
BDI	20.1	10.4	17.1	9.8	0,10	.802
STAI–state	54.5	12.9	54.9	12.3	0.008	.931
STAI trait	68.9	9.3	65.5	9.8	0.035	.342
MALT	27.9	4.7	25.0	7.5	1,627	.213
OCDS	20.4	4.6	20.3	5.4	0.004	.948
Duration of treatment (days)	17.2	4.7	16.5	5.3	0.159	.634

Note. TAU: treatment as usual; Age: age in years; Duration: duration of addiction in years; Detoxification: number of previous inpatient detoxification treatments; Rehabilitation: number of previous rehabilitation programs; SDQ-5: Somatoform Dissociation Questionnaire 5 Items; PTSS: Posttraumatic Stress Scale-10; BDI: Beck Depression Inventory; STAI X1: State-Trait Anxiety Inventory Scale Form 1; STAI X2: State-Trait Anxiety Inventory Scale Form 2; MALT: Munich Alcoholism Test; OCDS: Obsessive–Compulsive Drinking Scale; Treatment: duration of treatment in days; Test-value: Chi^2 for categorical variables, *T* for continuous variables.

Since depression is a common comorbid condition in clients suffering from addictive disorders, BDI scores served as a secondary measure of treatment outcome in our study. Compared to pretreatment, posttreatment scores of the BDI revealed a significant improvement in the TAU+EMDR treatment group (8.7 *SD* 6.7, $T = 4.0$, $p = .001$), while only a small reduction was seen in TAU (15.4 *SD* 7.8, $T = 0.9$, $p = .37$).

1-Month Follow-Up Data

A total of 24 patients (TAU: 11, EMDR: 13) responded to follow-up 1 month after treatment. The mean OCDS score in patients receiving EMDR was 13.7 (*SD* 5.7) compared to 20.9 (*SD* 10.7) for TAU. In contrast to pretreatment data this meant a significant reduction in the EMDR group's scores even if the OCDS measured 4.2 points higher than the posttreatment score. With regard to pretreatment, OCDS score reduction in patients receiving EMDR 1 month after treatment compared to pretreatment was still statistically significant (13.7 *SD* 5.7, $T = 6.2$, $p < .001$). Patients receiving TAU reported slightly higher levels of craving (OCDS) compared to pretreatment (20.9 *SD* 11, $T = 0.10$, $p = .44$). Between-group differences of TAU+EMDR versus TAU in OCDS scores posttreatment at 1-month follow-up ($p < .05$) were also statistically significant (see Figure 1).

6-Month Follow-Up Data

Six patients treated with TAU+EMDR and 2 patients from the TAU group reported at the 6-month follow-up. Statistical evaluation of the OCDS is impossible because of the small amount of data. Six months after treatment was terminated, 15 TAU patients relapsed or failed to report, while only 10 patients in the TAU+EMDR group relapsed or failed to report. The patients failing to report at 6-month follow-up were counted as relapsers (DGS, 2001) in accordance with the standards (DGSS-4) of the German Society for Addiction Research–DGS). Applying Fisher's exact test revealed a statistically significant difference between TAU+EMDR and TAU regarding relapse ($p < .05$) (see Figure 2).

Discussion

The most important study finding is that reprocessing the AM using a set of modified EMDR procedures was followed by a significant decrease in craving for alcohol posttreatment and at 1-month follow-up as measured with the OCDS. Compared to TAU, patients who received two sessions of EMDR in addition to TAU reported a significantly greater decrease in craving after termination of inpatient treatment as

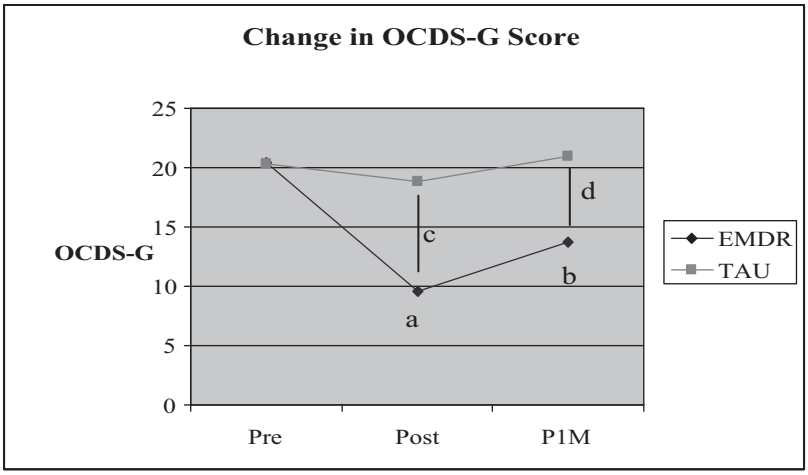

FIGURE 2. Changes in Obsessive–Compulsive Drinking Scale.

a = Statistically significant difference to pretreatment score ($p < .05$); b = Statistically significant difference to pretreatment score ($p < .05$); c = Statistically significant difference between EMDR and TAU posttreatment ($p < .05$); d = Statistically significant difference between EMDR and TAU P1M ($p < .05$); OCDS = Obsessive–Compulsive Drinking Scale; P1M = 1-month follow-up data.

well as during 1-month follow-up. This finding is also reflected in the between-group difference in relapse rates, as fewer patients receiving EMDR relapsed. EMDR treatment was also associated with a significant decrease in depressive symptoms, while patients receiving TAU showed no improvement in this area.

EMDR was relatively short (two sessions) and only directed at reprocessing of the AM. This fact might give us insight about the need for more extensive treatment sessions focused on the AM to determine if the impact on that specific type of target can be further reduced.

Despite these limitations, the OCDS score did not reach pretreatment level during follow-up in patients treated with EMDR, while TAU had no appreciable impact on craving. Use of the standard EMDR protocol for treating addictions would provide a more comprehensive treatment plan, including the reprocessing of earlier (traumatic) memories, setting the basis for the dysfunction, the present triggers, and future templates of adaptive behavior. This would be expected to enhance overall treatment outcomes, but future research is needed to determine that information.

A comprehensive, addiction-specific EMDR treatment plan needs to include earlier memories of craving or relapse in addition to earlier distressing events and experiences that laid the groundwork for dysfunctional negative beliefs. Present-day triggers (related to earlier traumatic memories and current substance use cravings and triggers) as well as specific future templates aimed at stable abstinence would probably be needed to maximize robust and lasting treatment effects with this complex population.

Surprisingly, targeting and reprocessing the AM did not lead to a destabilization of patients. During one EMDR session a patient developed a strong feeling of panic, which was completely reprocessed during the routine EMDR process. This patient had been diagnosed with comorbid panic disorder prior to the start of the study. Since a number of patients in this study also had a comorbid PTSD diagnosis, traumatic memory activation during EMDR processing could be expected to arise in a considerable number of patients.

Contrary to expectations, we did not observe activation of traumatic memories during EMDR treatment in our study. Our preliminary conclusion is that processing of the AM may be independent from processing of traumatic memories even in patients with a comorbidity of trauma-related disorders. It is hoped that our positive experiences using EMDR in addicted patients can be replicated in more rigorous studies. There may be a significant advantage to integrating psychotherapeutic interventions such as EMDR to process addiction memory at an early stage during inpatient treatment for alcohol-addicted patients.

This pilot study had several limitations. The sample size was small, thus reducing statistical validity. Treatment was applied by the same person evaluating the study, which might have biased the outcome. The study, however, was placed in a naturalistic psychiatric inpatient setting with the only modification of two additional sessions of EMDR treatment. Patients participating in the study were chronically dependent, and the majority of them had previously received a significant amount of traditional inpatient treatment

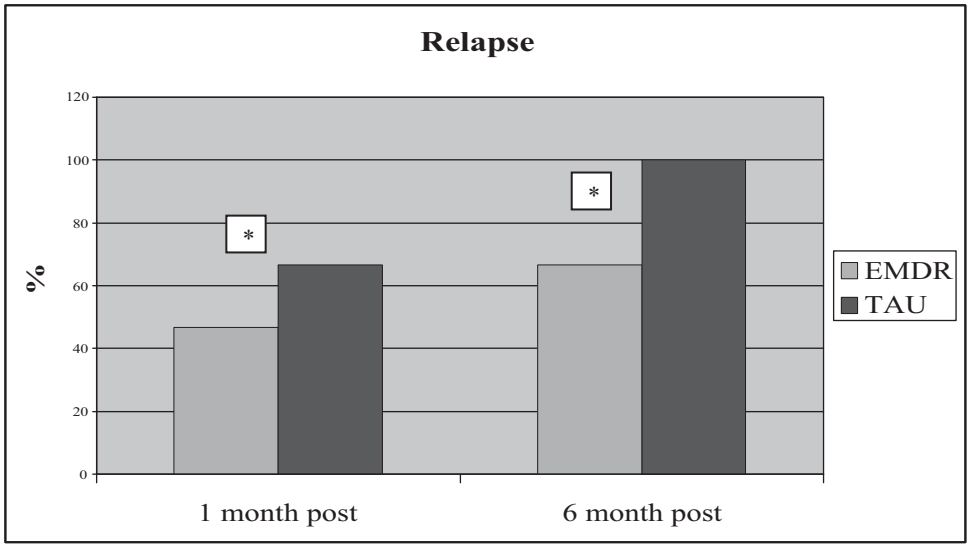

FIGURE 3. Number of relapses in EMDR and TAU.

* Statistically significant difference according to Fisher's exact test ($p < .05$).

and rehabilitation before being included in the study. This fact makes improvement by uncontrolled minor influential factors unlikely.

The results of our study support the animal model of AM (Wolffgramm et al., 2000) as well as the theory of AIP (Shapiro, 2002). The modified EMDR treatment employed in this study might facilitate the integration of addiction-related implicit memories into networks of consciously manageable explicit memories, therefore reducing involuntary craving for alcohol. The findings of this preliminary study need to be reproduced under more rigorous research conditions including a larger sample of patients suffering from addiction disorders. If the results of our study hold, a modified EMDR protocol for processing addiction memory could potentially improve relapse prevention in treating patients suffering from alcohol dependency and other forms of addictive behavior.

References

American Psychiatric Association. (2004). *Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder*. Arlington, VA: Author.

Andrade, J., Kavanagh, D., & Baddeley, A. (1997). Eye-movements and visual imagery: A working memory approach to the treatment of post-traumatic stress disorder. *British Journal of Clinical Psychology, 36*, 209–223.

Anton, R. F., Moak, D. H., & Latham, P. K. (1996). The obsessive compulsive drinking scale: A new method of assessing outcome in alcoholism treatment studies. *Archives of General Psychiatry, 53*(3), 225–231.

Barrowcliff, A. L., Gray, N. S., Freeman, T. C. A., & MacCulloch, M. J. (2004). Eye-movements reduce the vividness, emotional valence and electrodermal arousal associated with negative autobiographical memories. *Journal of Forensic Psychiatry and Psychology, 15,* 325–345.

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.

Bernstein, E. M., Carlson, E., et al. (1992). *Manual for the Dissociative Experience Scale*. Beloit, WI.

Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *The Journal of Nervous and Mental Disease, 174*(12), 727–735.

Bisson, J., & Andrew, M. (2007). Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews, 2007*(3).

Boening, J. A. (2001). Neurobiology of an addiction memory. *Journal of Neural Transmission, 108*(6), 755–765.

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry, 162*(2), 214–227.

Brady, K. T., & Sinha, R. (2005). Co-occurring mental and substance use disorders: The neurobiological effect of chronic stress. *American Journal of Psychiatry, 162,* 1483–1493.

Chick, J., Anton, R., Checinski, K., Croop, R., Drummond, D. C., Farmer, R., et al. (2000). A multicentre, randomized, double-blind, placebo-controlled trial of naltrexone in the treatment of alcohol dependence or abuse. *Alcohol Alcohol, 35*(6), 587–593.

Christman, S. D., Garvey, K. J., Propper, R. E., & Phaneuf, K. A. (2003). Bilateral eye movements enhance the retrieval of episodic memories. *Neuropsychology, 17,* 221–229.

DGS. (2001). Dokumentationsstandards III für die Evaluation der Behandlung von Abhängigen. *Sucht, 47* (Sonderheft 2).

Dilling, H., Mombour, W., & Schmidt, M. H. (1991). *Internationale Klassifikation psychischer Störungen: ICD-10, Kapitel V (F)*. Bern, Göttingen, Toronto: Huber.

Eid, J., Thayer, J. F., & Johnsen, B. H. (1999). Measuring posttraumatic stress: A psychometric evaluation of symptom- and coping questionnaires based on a Norwegian sample. *Scandinavian Journal of Psychology, 40*(2), 101–108.

Feuerlein, W., Ringer, C., Kufner, H., & Antons, K. (1979). The diagnosis of alcoholism—the Munich Alcoholism Test (MALT). *International Journal of Rehabilitation Research, 2*(4), 533–534.

Gorenc, K. D., Bruner, C. A., Nadelsticher, A., Pacurucu, S., & Feuerlein, W. (1984). A cross-cultural study: A comparison of German, Spanish and Ecuadorian alcoholics using the Munich Alcoholism Test (MALT). *American Journal of Drug and Alcohol Abuse, 10*(3), 429–446.

Hase, M. (2003). You'll never walk alone—Trauma und Sucht Über die Konvergenz scheinbar divergierender Konzepte. In C. Jellinek, G. U. Bellmann, & B. Westermann (Eds.), *Mehr als abhängig? Schlafstörungen: Trauma, Betäubung, Behandlung* (pp. 127–145). Freiburg im Breisgau: Lambertus-Verlag.

Hase, M. (2006). EMDR in der Behandlung der stoffgebundenen Abhängigkeit. *Praxisbuch EMDR*. Stuttgart, Klett-Cotta: F. H. Lamprecht.

Heyne, A. (1996). The development of opiate addiction in the rat. *Pharmacology, Biochemistry, and Behavior, 53*(1), 11–25.

Heyne, A., May, T., Goll, P., & Wolffgramm, J. (2000). Persisting consequences of drug intake: Towards a memory of addiction. *Journal of Neural Transmission, 107*(6), 613–638.

Heyne, A., & Wolffgramm, J. (1998). The development of addiction to d-amphetamine in an animal model: Same principles as for alcohol and opiate. *Psychopharmacology (Berl), 140*(4), 510–518.

Hyman, S. E. (2005). Addiction: A disease of learning and memory. *American Journal of Psychiatry, 162*(8), 1414–1422.

Jacobsen, L. K., Southwick, S. M., & Kosten, T. R. (2001). Substance abuse disorders in patients with posttraumatic stress disorder: A review of the literature. *American Journal of Psychiatry, 158,* 1184–1190.

Mann, K., & Ackermann, K. (2000). Die OCDS-G: Psychometrische Kennwerte der deutschen Version der Obsessive Compulsive Drinking Scale. *Sucht, 46*(2), 90–100.

Margraf, J. (1994). *Diagnostisches Kurz-Inventar bei psychischen Störungen*. Berlin: Springer.

NICE. (2005). *Post traumatic stress disorder (PTSD): The management of adults and children in primary and secondary care*. London: National Institute of Clinical Excellence.

Nijenhuis, E. R., Spinhoven, P., van Dyck, R., van der Hart, O., & Vanderlinden, J. (1997). The development of the somatoform dissociation questionnaire (SDQ-5) as a screening instrument for dissociative disorders. *Acta Psychiatrica Scandinavica, 96*(5), 311–318.

Omaha, J. (2004). *Psychotherapeutic interventions for emotion regulation: EMDR and bilateral stimulation for affect management*. New York: W. W. Norton.

Popky, A. J. (2005). DeTUR, an urge reduction protocol for addictions and dysfunctional behaviors. In R. Shapiro (Ed.), *EMDR solutions*. New York: W. W. Norton.

Schneider, U., Altmann, A., Baumann, M., Bernzen, J., Bertz, B., Bimber, U., et al. (2001). Comorbid anxiety and affective disorder in alcohol-dependent patients seeking treatment: The first Multicentre Study in Germany. *Alcohol and Alcoholism, 36*(3), 219–223.

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

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

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

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

Shapiro, F., & Hofmann, A. (1994). *Einführungsseminar in die EMDR-Methode—Manual Teil 1 von 2*. Watsonville, CA, & Bergisch-Gladbach: EMDR-Institute Inc. zusammen mit EMDR-Institut Deutschland.

Shapiro, F., Vogelmann-Sine, S., & Sine, L. F. (1994). Eye movement desensitization and reprocessing: Treating trauma and substance abuse. *Journal of Psychoactive Drugs, 26*(4), 379–391.

Spielberger, C. D., Gorsuch, R. L., & Luschene, R. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58*(1), 65–72.

Tiffany, S. T. (1999). Cognitive concepts of craving. *Alcohol Research & Health, 23*(3), 215–224.

Tiffany, S. T., Carter, B. L., & Singleton, E. G. (2000). Challenges in the manipulation, assessment and interpretation of craving relevant variables. *Addiction, 95*(Suppl. 2), S177–187.

van der Kolk, B. A., Burbridge, J. A., & Suzuki, J. (1997). The psychobiology of traumatic memory—clinical implications of neuroimaging studies. In A. Yehuda & A. C. McFarlane (Eds.), *Psychobiology of posttraumatic stress disorder* (p. 821). New York: Academy of Sciences.

Vogelmann-Sine, S., Sine, L. F., Smyth, N. J., & Popky, A. J. (1998). *EMDR chemical dependency treatment manual*. Honolulu, HI: Author.

Wetterling, T., Veltrup, C., & Junghanns, K. (1996). Craving—ein ausreichend fundiertes Konzept? *Fortschritte der Neurologie Psychiatrie, 64*, 142–152.

Wolffgramm, J. (2002). *Bildung und löschung des suchtgedächtnis*. Reinstorf: Forensische Tage Lüneburg.

Wolffgramm, J., Galli, G., Thimm, F., & Heyne, A. (2000). Animal models of addiction: Models for therapeutic strategies? *Journal of Neural Transmission, 107*(6), 649–668.

Wolffgramm, J., & Heyne, A. (1995). From controlled drug intake to loss of control: The irreversible development of drug addiction in the rat. *Behavioral Brain Research, 70*(1), 77–94.

Correspondence regarding this article should be directed to Michael Hase, Department of Psychosomatic Medicine, Reha-Zentrum Berliner Tor, Jungestr. 10 20535, Hamburg, Germany. E-mail: hase@berliner-tor.de