

The Effects of β -Adrenergic Blockade on the Degrading Effects of Eye Movements on Negative Autobiographical Memories

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ABSTRACT

BACKGROUND: Eye movement desensitization and reprocessing (EMDR) is an effective treatment for posttraumatic stress disorder. During EMDR, patients make horizontal eye movements (EMs) while simultaneously recalling a traumatic memory, which renders the memory less vivid and emotional when it is later recalled again. Recalling highly emotional autobiographical memories enhances noradrenergic neurotransmission. Noradrenaline (NA) strengthens memory (re)consolidation. However, memories become less vivid after recall+EMs. Therefore, NA might either play no significant role or serve to strengthen memories that are degraded by EMs. The present study was designed to test the latter hypothesis. We predicted that blocking NA would abolish the memory degrading effects of EMs.

METHODS: Fifty-six healthy participants selected three negative autobiographical memories. One was then recalled while making EMs, one was recalled without EMs, and one was not recalled. Vividness and emotionality of the memories as well as heart rate and skin conductance level during memory retrieval were measured before, directly after, and 24 hours after the EM task. Before the task, participants received a placebo or the noradrenergic β -receptor blocker propranolol (40 mg).

RESULTS: There were no effects of EMs on memory emotionality or psychophysiological measures in the propranolol and placebo groups. However, in the placebo group, but not in the propranolol group, memory vividness significantly decreased from pretest to posttest and follow-up after recall+EMs relative to the control conditions.

CONCLUSIONS: Blocking NA abolished the effects of EMs on the vividness of emotional memories, indicating that NA is crucial for EMDR effectiveness and possibly strengthens the reconsolidation of the degraded memory.

Keywords: Dual tasking, EMDR, Eye movements, Memory reconsolidation, Noradrenaline, Propranolol

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Posttraumatic stress disorder (PTSD) affects about 9% to 18% of trauma-exposed individuals (1). Symptoms involve re-experiencing the traumatic event, avoidance of its reminders, negative cognitions or mood, and hyperarousal (2). A frequently used psychological treatment for PTSD is eye movement desensitization and reprocessing (EMDR). Its core component is that patients recall trauma memories while simultaneously making lateral eye movements (3). While the treatment has met with skepticism, meta-analyses have shown that EMDR for PTSD is as effective as cognitive behavioral therapy (CBT) (4–6). Both EMDR and CBT serve as treatments of choice in clinical guidelines for PTSD in various countries (4,5).

A plausible explanation for the effectiveness of EMDR is provided by the working memory (WM) theory (7–9). WM is a cognitive system for maintaining and manipulating information, and it has limited resources (10). Recalling an emotional memory taxes WM (11), but so does making EMs (12–14). This leaves fewer resources for recall, rendering the memory less

vivid and emotional. Evidence for the WM theory comes from studies that show that memories are degraded not only due to recall+EMs but also by other tasks that require WM resources, like recall plus playing the computer game Tetris (12), mental arithmetic (13,15), copying a complex drawing (16), and other tasks (9). Interestingly, the desensitization of memories by dual tasks persists over time: memories are not only less vivid and emotional during the recall+WM load episodes, but also when the memory is later recalled again, without the WM load (16,17). EMDR presumably exploits the fact that memories become plastic during recall and that future recalls are affected by the nature of earlier recalls (9,18,19). That is, after EMDR, less vivid, less emotional, and less detailed memories may be reconsolidated into long-term storage, reducing their distressing impact.

In line with the WM theory, negative or traumatic memories show reduced vividness and emotionality after EMs and other dual-task procedures, but so do other mental images, like distressing images about possible future events (20,21) and

positive or appetitive memories (22–24). However, in contrast to the WM theory, vivid, autobiographical memories with relatively low emotional intensity have been observed to be insensitive to EM manipulation (25,26). The emotional arousal that is evoked by the retrieval of emotional memories might therefore be crucial for the memory-degrading effects of EMDR and other recall+WM load interventions.

Generally speaking, emotionally arousing events are better encoded than are emotionally neutral events (27). This emotion-superiority effect is caused by the release of the stress-related neurotransmitter noradrenaline (NA), especially in the basolateral amygdala (28). Many studies have shown that the blockade of noradrenergic β receptors with NA antagonists such as propranolol reduces the specific enhancement of memory for emotionally arousing material, including emotional stories, emotional pictures, or sets of emotional words, relative to memory for neutral material (29). Stimulating noradrenergic activation with NA agonists like yohimbine, on the other hand, increases memory for emotional stories (30), and for emotional and neutral wordlists (31,32). In addition, administering NA agonists during fear learning delays the process of extinction learning and generates a superior recovery of physiological fear responses (33).

Emotional arousal affects not only memory consolidation, but also the reconsolidation of older memories that are reactivated. Under the influence of propranolol, the retrieval of previously learned, emotional wordlists and pictures leads to subsequent memory deficits. These deficits are not observed when neutral memories are retrieved (29). Moreover, blocking noradrenergic activation with propranolol reduces the expression of previously learned cue-elicited fear responses (34,35).

Note that during EMDR, very emotionally arousing, traumatic memories are recollected. Especially during the first recalls, patients remain highly emotional, and therefore the emotion-superior (re)encoding will probably remain intact. At first glance this seems at odds with the common finding that emotional memories become less vivid and emotional during EMDR. So, EMDR might desensitize the memories despite the fact that these are emotional. Alternatively, it might be that the degraded, i.e., the less vivid and emotional memory is subjected to hyperencoding. Stated otherwise, the combination of recall+EMs and NA release may cause the traumatic memory to be reconsolidated as a relatively nonsalient event. If true, EMDR would not work despite the emotional nature of the memory, but rather because of the emotional nature of the memory.

The central goal of the current study was to find out whether noradrenergic mechanisms mediate the effects of EMs on memory. Healthy participants selected three negative autobiographical memories. One memory was recalled while making EMs, one was recalled while keeping eyes still, and one was not recalled. Before recall, participants received a placebo or propranolol. Propranolol is a nonselective noradrenergic β receptor blocker that crosses the blood-brain barrier and exerts both peripheral and central effects. The critical site of action is at central β receptors (36), where propranolol interferes with the protein synthesis that is necessary for memory (re)consolidation (37–39). Here, we expected that propranolol would reduce or abolish the commonly observed

degrading effects of EMs by blocking the reconsolidation of the memory that is attenuated by recall+EMs. Stated otherwise, instead of the reconsolidation of the memory in an inferior form (i.e., less vivid or detailed than the original memory) typically seen after recall+EMs without medication, we expected no changes to the original memories after the administration of propranolol (i.e., no reconsolidation in an inferior form). This would indicate that arousal-related noradrenergic activity is a prerequisite for the desensitization of traumatic memories by EMDR. If NA has beneficial effects on the EM intervention, this will help understand the effectiveness of EMDR and other techniques that serve to alter emotional memories, such as imagery rescripting (40), extinction learning (41), or CBT (42).

As in previous EM studies, the most important outcome measures were self-reported memory vividness and emotionality. However, to provide a more objective measure of the effects of propranolol on memory desensitization by EMs, average heart rate (HR) and skin conductance level (SCL) during memory recall were additionally measured. Previous studies have indicated that patients with PTSD show reduced psychophysiological responding during trauma memory recall after one or more sessions of EMDR (43–45). We expected that participants would show decreased psychophysiological responses to the memories after the EM intervention, and that propranolol would reduce these effects.

METHODS AND MATERIALS

Participants

Sixty participants were tested using a double-blind, placebo-controlled, mixed factorial design. Participation was limited to individuals between 18 and 35 years old without current or previous medical conditions that contraindicate the administration of 40 mg propranolol (cardiovascular disease, lung/kidney/liver disease, substance use, psychiatric/neurological disorders, pregnancy) and without any contraindicative medication intake. Furthermore, individuals with knowledge of EMDR mechanisms were excluded. This was achieved by asking participants to describe four psychological interventions, one of which was EMDR. Participants were assigned to two experimental conditions (propranolol, placebo) by an external person in such a way that differences in age and gender were minimized (46). Four participants were excluded from the analyses because the pretest vividness and/or emotionality scores of their memories deviated >2.5 SD from the mean (47). The final sample comprised 56 participants (mean age = 22.0 years, SD = 2.4 years; 75% women, 25% men). The study was approved by the medical ethical committee of University Medical Center Utrecht and informed consent was obtained from all participants. Participants received financial compensation for their participation.

Materials

An EM task (48) was used to simulate the EM component of EMDR, and it was computerized. Participants were instructed to track a horizontally moving dot (1 Hz) on a black screen or watch a black screen without a dot. Meanwhile they recalled one of their aversive memories. The moving dots and blank

screens were displayed during six intervals of 24 seconds separated by 10-second breaks. EM interventions this brief have been demonstrated to affect memory quality in previous studies (8,11,16,49–51). Before (pretest) and after (posttest) the EM task, and at a 24 hour follow-up, participants recalled each aversive memory and rated its vividness using 10-cm visual analog scales (VASs) ranging from 0 (not vivid) to 100 (very vivid), and its emotional intensity using a VAS ranging from 0 (not emotional) to 100 (very emotional). The experimental task was programmed in E-Prime, version 1 (Psychology Software Tools, Inc., Sharpsburg, PA). Participants were seated in a comfortable chair approximately 30 cm from the screen in a dimly lit experimental cabin. During memory recall, HR and SCL were recorded. HR was measured with two Ag/AgCl electrodes placed on the chest and left abdomen. SCL was measured with two Ag/AgCl electrodes placed on the palmar side of the distal phalanges of the index and middle finger of the nondominant (left) hand. For both measures a digital Active-Two system (BioSemi, Amsterdam, the Netherlands) was used. Raw signals were 24-bit A/D converted at 1000 Hz. For SCL, a constant-voltage skin conductance coupler was used.

Procedure

Individuals who signed up for the study ($N = 96$) were contacted by phone and screened for inclusion and exclusion criteria. Eligible participants were invited for a medical screening at least 1 week later and were sent an information letter and informed consent form by postal mail. During the medical screening session, participants signed the consent form. Blood pressure (BP) and HR were measured, an electrocardiogram was recorded, and a 2-minute cardiovascular step test was performed. When no anomalies were found, participants ($n = 60$) were included for the main experiment. They were tested on 2 consecutive days, 24 ± 8 hours apart. On the first test day, participants recollected three aversive autobiographical memories following the procedure used by van den Hout *et al.* (48). In line with the Dutch EMDR protocol (52), participants “played” these memories in their minds and made a “screen shot” of the most intense moment. They described the resulting images with keywords and then ranked the images based on emotional intensity for counterbalancing purposes. Then participants recalled all images for 10 seconds and rated vividness and emotionality using two VASs. During the 10-second recall, HR and SCL were measured. After the pretest, BP and HR were assessed with a BP monitor and participants received propranolol (40 mg) or a placebo (double blind). Before medication intake, participants ate a granola bar. Approximately 100 minutes after medication intake, when propranolol presumably had reached peak levels (53), BP and HR were measured again with a BP monitor to ensure participant safety. Then one memory was recalled while making EMs (recall+EMs), one memory was recalled without making EMs (recall only [RO]), and one memory was not recalled (no recall [NR]). The order of the recall+EMs and RO conditions was counterbalanced. After both interventions, an immediate posttest was administered, during which participants recalled each memory again for 10 seconds and rated the VASs for vividness and emotionality. At the end of the task, the memory in the NR condition was recalled and rated. Thirty minutes after

task completion, BP and HR were again assessed with a BP monitor to ensure participant safety, and participants were dismissed. On the second test day, participants recalled and scored their memories for a third time. HR and SCL were measured during memory recall on both the posttest and the follow-up. At the end of the second test day, participants were debriefed and received payment.

Data Reduction

Electrophysiological data were processed with BrainVision Analyzer 2 (Brain Products GmbH, Munich, Germany). HR data were additionally analyzed with custom-made software. Baseline-corrected, log-transformed SCL and HR (beats/min) data were averaged per memory recall condition (EM, RO, NR) for the 10-second memory recall period. See the [Supplement](#) for a detailed description of data-reduction procedures.

Statistical Analyses

There were two groups (placebo and propranolol) and three conditions: one experimental (recall+EMs) and two control (RO, NR) conditions. Assessment took place three times: before the EM intervention and medication intake (pretest), immediately after the EM intervention (posttest), and 24 hours later (follow-up).

Four 2 (group: placebo, propranolol) \times 3 (condition: recall+EMs, RO, NR) \times 3 (time: pretest, posttest, follow-up) mixed factorial repeated measures analyses of variance were conducted to assess whether image vividness, image emotionality, and HR and SCL during 10-second memory recall were more reduced after recall+EMs than after RO and NR in the placebo group, but not in the propranolol group. Group was a between-subjects factor, while condition and time levels had been varied within subjects. Wherever sphericity assumptions were violated as indicated by Mauchly's test, Greenhouse-Geisser corrections were applied to adjust the number of degrees of freedom for within group effects. An alpha level of .05 was used for all statistical tests.

RESULTS

See the [Supplement](#) for randomization and manipulation checks. See [Table 1](#) for mean vividness, emotionality, HR, and SCL values for separate groups and conditions at pretest, posttest, and follow-up.

Memory Vividness

[Figure 1](#) shows posttest and follow-up values, relative to pretest values (i.e., decreases in vividness over time). Overall, memory vividness scores decreased from pretest to posttest and follow-up. This was reflected in a significant main effect for time ($F_{2,108} = 6.64, p < .01, \eta^2 = .10$), with significantly lower vividness scores at follow-up than pretest ($p = .001$). There was also a significant main effect of condition ($F_{2,108} = 3.94, p = .02, \eta^2 = .07$). Overall, memory vividness scores were lower in the EM condition than in the RO condition ($p = .05$). Furthermore, data ([Figure 1](#)) suggest that the placebo/propranolol difference was higher in the EM condition than in the other conditions. Indeed, the crucial higher-order interaction of time \times condition \times medication was significant ($F_{4,216} = 2.54$,

Table 1. Vividness and Emotionality VAS Scores (Range 0–100), SCLs, and HRs for Memories in the Recall + EMs, RO, and NR Conditions at Pretest, Immediate Posttest, and 24-Hour Follow-Up

	Placebo			Propranolol		
	EMs	RO	NR	EMs	RO	NR
Vividness						
Pretest	67.25 (19.76)	67.11 (15.90)	60.96 (13.65)	61.07 (18.07)	66.43 (18.26)	67.57 (13.99)
Posttest	57.46 (21.62)	63.96 (16.47)	62.79 (18.82)	56.43 (21.21)	65.11 (17.86)	62.89 (17.75)
Follow-up	54.18 (18.01)	57.0 (17.03)	62.29 (13.62)	58.86 (17.77)	64.07 (19.50)	61.75 (22.20)
Emotionality						
Pretest	61.07 (17.74)	65.14 (14.80)	65.04 (12.92)	64.14 (14.34)	67.04 (9.99)	65.36 (13.46)
Posttest	53.75 (21.42)	55.0 (20.25)	58.29 (18.85)	54.68 (22.13)	60.79 (17.86)	61.71 (15.62)
Follow-up	52.39 (19.02)	56.18 (18.57)	59.21 (14.29)	58.75 (19.24)	60.82 (17.47)	63.18 (19.79)
SCL (Log-Transformed)						
Pretest	2.34 (0.52)	2.26 (0.49)	2.15 (0.56)	2.07 (0.44)	2.08 (0.45)	2.12 (0.44)
Posttest	1.94 (0.69)	1.99 (0.71)	2.15 (0.62)	2.19 (0.71)	2.16 (0.67)	2.20 (0.70)
Follow-up	2.22 (0.59)	2.29 (0.64)	2.22 (0.69)	2.22 (0.57)	2.21 (0.60)	2.22 (0.56)
HR						
Pretest	−0.54 (4.45)	−1.04 (4.19)	0.04 (4.65)	−0.12 (4.11)	−0.59 (4.52)	−0.58 (4.51)
Posttest	−0.64 (4.16)	−3.15 (6.76)	−1.76 (6.33)	−1.72 (4.22)	0.11 (4.76)	0.77 (4.18)
Follow-up	−2.30 (4.61)	−2.04 (7.72)	−2.35 (4.56)	−1.02 (3.84)	−0.91 (4.48)	−1.09 (4.38)

Values are presented as mean (SD). Skin conductance levels (SCLs) and heart rates (HRs) were corrected for baseline levels (10 to 5 seconds prerecall), resulting in mostly negative values for HRs.

EM, eye movement; NR, no recall; RO, recall only; VAS, visual analog scale.

$p = .04$, $\eta^2 = .05$). This interaction was broken down by medication.

In the placebo group, the crucial time \times condition interaction was significant ($F_{4,108} = 3.63$, $p < .01$, $\eta^2 = .12$). In line with previous studies, follow-up t tests showed that the EM intervention caused a decrease in memory vividness from the pretest (mean = 67.25, SD = 19.76) to posttest (mean = 57.46, SD = 21.62; $t_{27} = 2.16$, $p = .04$) and from the pretest to follow-up (mean = 54.18, SD = 18.01; $t_{27} = 4.31$, $p < .001$; see Figure 1, left panel). After RO (see Figure 1, middle panel),

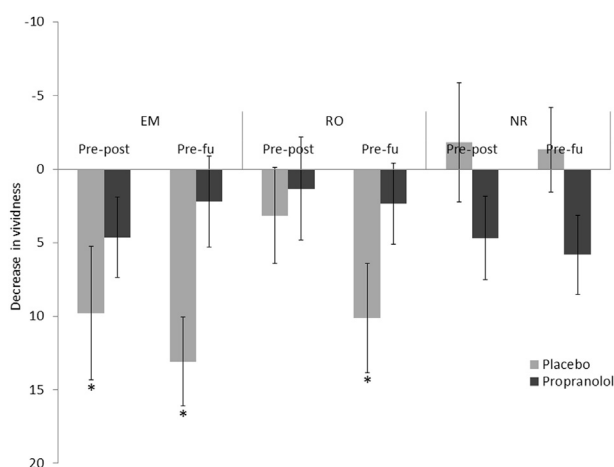


Figure 1. Average (\pm SEM) decreases in vividness scores from pretest (pre) to posttest (post) and pretest to follow-up (fu), for the recall + eye movements (EMs), recall only (RO), and no recall (NR) conditions depicted for the placebo and propranolol groups. Asterisks indicate significant ($p < .05$) decreases between measurements (from pretest to posttest [pre-post] or from pretest to follow-up [pre-fu]; see x axis).

memory vividness did not decrease from the pretest (mean = 67.11, SD = 15.90) to posttest (mean = 63.96, SD = 16.47; $t_{27} = 0.97$, $p = .34$). However, it did decrease from pretest to follow-up (mean = 57.00, SD = 17.03; $t_{27} = 2.72$, $p = .01$). The vividness of memories that were not recalled (NR condition) did not significantly change from pretest to posttest ($t_{27} = 0.45$, $p = .66$) or from pretest to follow-up ($t_{27} = 0.46$, $p = .65$; see Figure 1, right panel).

In the propranolol group, the crucial time \times condition interaction was not significant ($F_{4,108} = 0.43$, $p = .73$, $\varepsilon = .73$). See the Supplement for additional line-graphs depicting raw pretest, posttest and follow-up scores (Supplemental Figure S1).

Memory Emotionality

There was a significant main effect for time ($F_{2,108} = 15.56$, $p < .001$, $\eta^2 = .22$), with significantly lower emotionality levels at posttest and follow-up than at pretest (both $ps < .001$). No other significant main or interaction effects were observed. Also, the crucial time \times condition \times medication interaction was not significant ($F_{4,216} = 0.52$, $p = .72$). See the Supplement for a graphical depiction of posttest and follow-up values, relative to pretest values (Supplemental Figure S2).

SCL During Memory Recall

No significant main effects were observed for SCL during memory recall. The time \times medication interaction was significant ($F_{2,106} = 3.46$, $p = .04$, $\eta^2 = .06$), with (a trend toward) lower SCLs at posttest than at pretest ($t_{26} = 2.32$, $p = .07$) and follow-up ($t_{27} = 3.21$, $p < .01$) in the placebo group, but not in the propranolol group, all $ts < 1.44$, all $ps > .47$. The crucial higher-order interaction of time \times condition \times medication was not significant ($F_{4,212} = 1.72$, $p = .15$).

HR During Memory Recall

There were no significant main or interaction effects. The crucial time \times condition \times medication interaction was also not significant ($F_{4,208} = 1.50, p = .21$).

Explorative Analyses

To check whether emotionality, SCL, and HR were affected by the EM intervention in the absence of propranolol intake, explorative analyses of variance were conducted. Correlational analyses were conducted to check whether SCL/HR were indicative of self-reports. See the [Supplement](#) for all explorative analyses.

DISCUSSION

Previous studies have shown that emotionally neutral memories do not become less vivid after recall+EMs, whereas equally vivid, emotional memories do (25,26). This finding suggests that emotional arousal is a prerequisite for the memory degrading effects of EMDR. The present study was designed to critically test this hypothesis and examine underlying working mechanisms.

There were no effects of recall+EMs versus control conditions on memory emotionality or psychophysiological measures in the propranolol and placebo groups. Only memory vividness was uniquely attenuated by recall+EMs, allowing further testing of the modulating effects of propranolol.

While memory vividness decreased after recall+EMs relative to RO and NR in the placebo group, memory vividness did not decrease in the propranolol group in either of the conditions. Thus, the blockade of noradrenergic activation abolished the commonly observed effects of EMs on the vividness of emotional memories. These effects were found at the posttest (immediately after the EM intervention) and at the follow-up (24 hours later). Since propranolol is known to impair memories by interfering with (re)consolidation (37–39), the present effects can be accounted for by attenuation of noradrenergic neurotransmission induced by propranolol, thereby blocking superior reconsolidation of the degraded memory.

Alternatively, the observed effects might be explained by WM taxation. After propranolol intake, general physiological arousal decreases. Perhaps memory recall during EMs was less taxing due to this reduced arousal, which might have decreased competition with EMs, which might have prevented substantial memory desensitization. However, it is unclear whether retrieving a memory while being less aroused diminishes its cognitive load. Furthermore, propranolol has impairing effects on WM itself (53,54). With such WM impairment, there would be more competition for WM resources, leading to increased memory desensitization.

In sum, the findings with respect to memory vividness confirm that noradrenergic neurotransmission is a prerequisite for memory desensitization by EMs. The underlying mechanism is likely to be NA-modulated, superior encoding of the less vivid image, although an alternative WM taxation explanation cannot be ruled out.

The finding that recall+EMs reduced memory vividness relative to RO in the placebo group is in line with numerous previous studies investigating the effects of EMs in a laboratory setting (9,11,17). In addition, the observed 24-hour delayed effects of EMs are in line with four studies investigating long-term effects

(16,55–57), but see (58). It must be noted that in the current study memory vividness also decreased from pretest to follow-up after RO. Although a recent meta-analysis (59) demonstrated a significant advantage for recall+EMs over RO across studies, it is still possible that such a decrease is also observed for RO, albeit weaker than for recall+EMs. This is in line with two laboratory studies (56,57) showing that the EMs outperformed RO in the short run, but that after a 1-week delay beneficial effects were observed in both conditions. The inconsistent findings on delayed effects might be inherent to the use of laboratory models of EMDR, which use only very brief sessions of recall and EMs (here: 6×24 seconds). It is expected that after more and/or prolonged sessions of EMs, as provided in EMDR therapy, recall+EMs will outperform RO [see clinical studies in (59)] also in the long run. Note that all studies consistently found that immediate memory degrading effects (pretest to posttest decreases in vividness) only arise after recall+EMs.

Interestingly, the decrease in vividness after RO was also attenuated by propranolol. This might indicate that possible beneficial effects of repeated memory exposure are also strengthened by NA release.

There was no effect of propranolol on changes in memory emotionality. However, in contrast with most previous studies, memory emotionality was not uniquely attenuated by recall+EMs to begin with, preventing further testing of the effects of propranolol on this unique effect. It is unclear why there were no effects of recall+EMs relative to the control conditions on memory emotionality in the placebo group. Although speculative, an explanation might lie in the relatively low emotionality scores at pretest (mean = 64.63, SD = 9.54 on a scale from 0 to 100). In previous studies, average pretest emotionality scores ranged between 70 and 85 (11,13,60). It is, however, not entirely uncommon that effects materialize on vividness only (7,8,14,61). Because research indicates that memory emotionality is reduced after memory vividness has dropped (62), it is expected that with prolonged sessions of EM effects will start to materialize on emotionality as well. It is important to note that not only high levels of memory emotionality, but also extreme levels of vividness are a distinctive feature of traumatic memories in PTSD. Patients experience their memories as if they are happening in the here and now (63). Being able to reduce memory vividness is therefore highly beneficial in itself.

The current study was the first to use HR and SCL during memory retrieval as an outcome measure of memory degradation by EMs. However, no effects of the EM intervention were found for these measures at posttest or follow-up. An explorative analysis testing whether pretest psychophysiological responses were correlated with self-reported memory emotionality and vividness only demonstrated a trend for a correlation between SCL and vividness. Therefore, SCL might be more responsive to autobiographical memory retrieval than HR. In line with this, we observed significant SCL decreases after recall+EMs and, to a smaller extent, after RO, when analyzing placebo group data only. However, these effects did not last until the follow-up. It may be that psychophysiological changes in response to memory retrieval are too subtle to detect in participants without PTSD, who selected aversive but not traumatic memories. Furthermore, although both HR and SCL generally increase with emotional intensity of memory imagery, an extensive review by Kreibitz (64) indicates that

some emotions cause decreases in HR and SCL (i.e., certain types of sadness and fear). Aversive memories in the present study could have induced a great range of emotions that might have responded differently to the interventions.

Because of possible carryover effects of emotional arousal, we were not able to include a neutral memory control condition. To meet this limitation, and to further investigate the possible role for NA release in EMDR, we are currently conducting a study testing whether the administration of NA agonists (e.g., yohimbine) will stimulate the degradation of neutral memories by EMs.

To summarize, results of the present study indicate that the emotionally arousing properties of the memories retrieved during EMDR contribute to the therapy's effects. NA release likely strengthens the reconsolidation of memories that have become less vivid by recall+EMs or other dual-task interventions. More research is necessary to draw more firm conclusions about the underlying mechanisms.

The present study helps to elucidate the working mechanisms of EMDR: a therapy that has been proven effective, but has long been the topic of debate because of its lack of a theoretical rationale. In addition, it helps to delineate boundary conditions of the experimental paradigm. Moreover, the finding that arousal-related noradrenergic activity appears to be crucial to the reconsolidation of attenuated memories helps to understand the effectiveness of other techniques that aim to update or rewrite clinically relevant memories, including imagery rescripting (40) or CBT (42). Another specific example might be postretrieval extinction training, which combines fear memory reactivation and extinction training (i.e., learning that a feared stimulus is safe) to update fear memory with safety information through reconsolidation processes (41,65). Finally, the present study paves the path for research on the use of pharmacological interventions to enhance effects of memory updating techniques. Results suggest that if adrenergic β receptors are stimulated during the application of these techniques, this could further strengthen the reconsolidation of attenuated images, and, additionally, boost the ease of recollection of positive memories and cognitions.

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