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Journal of Anxiety Disorders 22 (2008) 622–634

Journal of Anxiety Disorders

Physiological correlates of eye movement desensitization and reprocessing

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Received 15 January 2007; received in revised form 23 May 2007; accepted 31 May 2007

Abstract

Eye movement desensitization and reprocessing (EMDR) is an established treatment for post-traumatic stress disorder (PTSD). However, its working mechanism remains unclear. This study explored physiological correlates of eye movements during EMDR in relation to current hypotheses; distraction, conditioning, orienting response activation, and REM-like mechanisms.

During EMDR therapy, fingertip temperature, heart rate, skin conductance, expiratory carbon dioxide level, and blood pulse oximeter oxygen saturation, were measured in male subjects with PTSD. The ratio between the low and high frequency components of the heart rate power spectrum (LF/HF) were computed as measures of autonomic balance. Respiratory rate was calculated from the carbon dioxide trace.

Stimulation shifted the autonomic balance as indicated by decreases in heart rate, skin conductance and LF/HF-ratio, and an increased finger temperature. The breathing frequency and end-tidal carbon dioxide increased; oxygen saturation decreased during eye movements.

In conclusion, eye movements during EMDR activate cholinergic and inhibit sympathetic systems. The reactivity has similarities with the pattern during REM-sleep.

Keywords: Eye movement desensitization and reprocessing; Post-traumatic stress disorder; Orienting response; Autonomic physiology; Heart rate variability; Respiration

1. Introduction

Since its inception 1988, eye movement desensitization and reprocessing (EMDR) has provoked much discussion. It is, however, an established treatment modality for post-traumatic stress disorder (PTSD), and has been shown to be roughly equally effective in comparison with behavioral exposure treatment (Bradley, Greene, Russ, Dutra, & Westen, 2005). If the treatment is indeed effective, the question arises whether the eye movements *per se* are necessary for the effect. This has lead to investigations where subjects have been treated with the EMDR protocol minus eye movements. This has shown diverging results. Renfrey and Spates (1994), discussed in Cahill, Carrigan, and Frueh (1999), for instance, did not find any added effect of eye movements on therapeutic effect. However, Wilson et al. found that eye movements were effective, compared with two control conditions (Wilson, Silver, Covi, & Foster, 1996).

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0887-6185/$ – see front matter
doi:10.1016/j.janxdis.2007.05.012

The hypotheses regarding the putative effect of eye movements have been manifold; it has been suggested that the eye movements during the procedure fits in a model of ''respondent conditioning, emotional interference with learning, and operant conditioning''; in other words that conditioning and distraction plays a major role during the treatment (Dyck, 1993). Alternatively, it has been proposed that dual attention stimulation elicits the orienting response (Armstrong & Vaughan, 1996) and thus reduces avoidance and allows entry of new trauma-related information into the cognitive processing system. The orienting response is elicited by novel stimuli of any kind, and is characterized physiologically by a lower threshold for sensory stimuli and an initial inhibition of bodily functions that might disturb the perception of stimuli; respiration and heart rate is lowered, skin conductance is increased, and skin temperature goes down due to peripheral vasoconstriction (Öhman, Hamm, & Hugdahl, 2000). The orienting response is also characterized by a fast habituation. In addition, Stickgold has recently suggested that eye movement stimulation, possibly through repeated orienting responses, produce a neurobiological state, similar to that of 'Rapid Eye Movement'-sleep (REM-sleep), which in turn might involve cortical integration of traumatic memories (Stickgold, 2002).

One way of exploring the hypotheses is to study physiological correlates during EMDR sessions. Few previous studies have looked at physiological effects of eye movement stimulation, especially in naturalistic settings. Wilson et al. reported changed respiration, decreased heart rate, and decreased skin conductance during EMDR-treatment compared with two control conditions (Wilson et al., 1996). Similarly, Barrowcliff et al. found that eye movements, compared with two control conditions, significantly reduced skin conductance during exposure to white noise (Barrowcliff, Gray, MacCulloch, Freeman, & MacCulloch, 2003), and Sack has presented a study of heart rate during EMDR-treatment wherein heart rate dropped during eye movements (Sack, 2005).

The present study aimed at studying physiological effects of eye movements during EMDR-treatment of chronic PTSD. Physiological variables to measure were chosen on the basis that they could be non-invasively measured, without severe interference with the treatment, and because they are commonly used as indexes for de-arousal and/or are expected to be involved in the physiological pathways active in the proposed working models of EMDR.

2. Methods and materials

2.1. Participants

The present study included 13 male refugees, with a mean age of 37.5 years (S.D. = 5.5). They were all diagnosed with PTSD, and were distressed by memories of traumatic events, typically after torture and/or war experiences. The Clinician Administered PTSD Scale (CAPS) diagnostic instrument for PTSD (Blake et al., 1995) was used for assessment, and the participants' mean score on the CAPS interview was 72.7 points (S.D. = 13.5). In the assessment of PTSD, DSM-IV criteria were used, as well as the requirement that a symptom was present at least once a week and with an intensity of at least 2 (moderate). Diagnosis and treatment was carried out by the last author.

The patients were included in the study if they had 10 or more previous treatment sessions without recovery (in order to avoid subjects with spontaneous recovery) and were without organic brain disorder, substance abuse, psychosis, or suicidality.

2.2. Procedures

Before the EMDR-treatment began, electrodes and sensors were placed and the physiological variables were allowed to stabilize (ideally a flat trend curve).

The EMDR-treatment essentially followed the guidelines outlined by Shapiro (1995). In short, the treatment protocol consists of three phases. During the *Target assessment phase*, the distressing issue or memory to be assessed during the session is presented and the most vivid image of the memory is identified (as well as related emotions and body sensations). The therapist also obtains the client's negative belief about the event together with a desired positive cognition for this same event. A validity of cognition (VoC) rating is used to estimate the validity of the positive cognition (on a 1–7 scale, where 1 represents completely false and 7 completely true). The degree of anxiety/disturbance that the picture/feeling/sensation/cognition evokes is rated by a Subjective Units of Distress Scale (SUDS) from 0 to 10, where 0 represents no distress and 10 represents the highest distress possible. Next the actual EMDR reprocessing procedures are applied during the *Desensitization phase*. The client is instructed to focus on the image, negative thought, and body sensations, while following the therapists upraised two fingers as they move from side to side. The client is then asked to ''Blank it out and take a deep breath'', and pause for a moment to report their experience and level of

disturbance on a SUD scale. Based on patient feedback, focus is directed to new images, feelings, or body sensations as such arise and the stimulation sets are repeated until desensitization is complete, as indicated by a SUD's rating of 0 or 1, or no progress is reported. Finally, during the *Installation phase*, the desired cognition is concentrated upon during the eye movements until a VoC rating of 6 or 7 (completely true) is obtained (or no progress is reported). This cognition and the original issue are then linked together during the eye movements by keeping both in mind, and finally a body scan is completed, checking for any negative body sensations. If there are any signs of tension, the eye movements are again induced, while the client concentrates on these sensations until they have dissipated. If SUDs do not decrease sufficiently, the patient is stabilized with the ''safe place'' procedure before the session is ended.

2.3. Physiological assessment

The physiological variables measured were fingertip skin temperature (FT), heart rate (HR), skin conductance (SC), expiratory carbon dioxide levels (CO_2), and blood pulse oximeter oxygen saturation (SpO_2). Data was sampled using the I-330 DSP12 physiologic monitoring system (J&J Engineering Inc., Poulsbo, WA, USA) and a customized software application developed in the PDS-environment (J&J Engineering Inc.). All variables where stored at 1 s intervals.

CO_2 and SpO_2 were obtained using a Capnocheck® Plus oxycapnometer (Sims Bci Inc., Waukesha, WI, USA). Oxygen saturation was measured photometrically with a sensor on the patient's left middle finger. Expiratory CO_2 was collected from the left nostril using a nasal catheter (6 in. × 1/8 in. × 1/323 in.) which was attached to the distal end of the capnometer sample line.

End-tidal CO_2 ($EtCO_2$) and respiration rate (RR) were computed using the recorded CO_2 wave data.

FT was measured with a thermistor taped to the right little finger tip.

Skin conductance was recorded by means of constant voltage using Ag/AgCl-electrodes placed on the second phalanx of the patient's right index finger and ring finger.

Electrocardiogram (ECG) data were sampled at 256 Hz with two Ag/AgCl electrodes placed on the patient's wrists. The HR was computed by the hardware based on interbeat data obtained from the ECG trace.

Indices of sympathetic and parasympathetic drives were obtained from frequency analysis of the heart rate variability. Frequency analysis was made on mean value subtracted HR data using the periodogram function of the signal analysis tool pack library in the Matlab ver. 6 suite (MathWorks, Natick, MA, USA). Obtained power spectra were divided into spectral bands and the powers in the low frequency band (LF: 0.04–0.15 Hz) and in the high frequency band (HF: 0.15–0.40 Hz) were calculated by integrating the power spectral density in the respective frequency bands. After normalization by dividing the obtained band power with total power minus the very low frequency band power (VLF: 0.003–0.04), LF/HF-ratios were calculated as estimates of sympathovagal balance (Malliani, Pagani, Lombardi, & Cerutti, 1991).

Data were manually controlled for artifacts and with regard to heart rate, ectopic or premature heart beats were identified using an automatized detection method. Artifacts were rejected from the data stream based on individual criteria for each variable, i.e. for oxygen saturation, temperature, and skin conductance, a number of artifacts were caused by glitching sensors that were easily detected through obvious non-physiological changes. For heart rate, corrections were carried out if it was possible to establish a firm rule for the imputation. Regarding heart rata data, an automatic iterative filter was applied that replaced data points deviating more than 20% from the mean of the surrounding values. A maximum of 5% erroneous values at single data points were accepted.

2.4. Data analysis

To be included in the analysis a treatment session had to meet the following criteria: (i) include an at least 50 s long period of stable physiological recordings preceding the assessment phase; (ii) include an at least 50 s long period of physiological recordings occurring at least 60 s after the ending of the installation phase; (iii) contain a minimum of four stimulation sets that are at least 50 s in length and preceded by likewise 50 s long phases of no stimulation; (iv) have at least four stimulation sets with a duration of 50 s or longer that are followed by 50 s or more of no stimulation; and (v) have an installation phase that does not include ''safe place''-procedures.

In order to assess physiological changes during eye movement stimulation, the following measurement periods were defined: (A) a 50 s long sequence occurring after the variables had stabilized but before the assessment phase had begun (Baseline, BL); (B) final 30 s prior to stimulation (Pre-stimulation, PRE); (C) first 50 s of ongoing stimulation (Stimulation, S); (D) final 30 s of stimulation (Stimulation end, SE); (E)

50 s following stimulation (Post-stimulation, POST); (F) a 50 s long sequence occurring at least 60 s after the installation phase had ended (Post-baseline, POSTBL). Periods B–E were further divided into 10 s long segments.

First, mean response time-courses were calculated for each session, from 30 s prior to the onset of stimulation sets (B) until 50 s post-onset (C) and from last 30 s of stimulation (D) until 50 s post-stimulation (E). Treatment sessions means were further combined into individual means for each participant.

Since data are obtained from repeated stimulation sets, measurements from the pre-onset baseline values (B and D) collected during the 30 s prior to stimulation-onset can be contaminated by the tail of the prior stimulation set, and hence baseline values were only calculated when there was at least 20 s between the end of the prior stimulation set and the start of the 30-s pre-onset baseline period. In order to minimize such transitional effects data were only obtained from B/C and D/E pairs with at least 50 s long pre-phases, thus preventing transitional changes shorter than 20 s from affecting the result.

The accuracy and repeatability of HRV measures in the frequency domain obtained from ultra-short records is questionable (Schroeder et al., 2004; Thong, Li, McNames, Aboy, & Goldstein, 2003), although improved if the mean of several recordings are used (Schroeder et al., 2004). To detect a given frequency, the signal must be observed for at least one period of this frequency and one cannot say exactly at which time the signal had this frequency. Because of this, recordings of the heart rate trace are recommended to last at least 10 times the period of the lower frequency boundary of the investigated component (Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology, 1996). Due to the nature of the EMDR protocol, with relatively short stimulation sets, this was not achievable in the present study. Instead HRV data was analyzed on 50 s long segments of HR recordings. This record length equals two times the lower frequency boundary of the LF-span. However, for the purposes of the present study, this short recording length was acceptable. First, the high number of samplings reduces the error caused by the short recording length, second, since we are not interested in power for a specific frequency but for broader spectra, the consequence of this error on the result is less severe. Accordingly, LF and HF data for all phases (B–E) had to be computed for full 50 s long periods with no further subdivisions, as was the longest period compatible with a sufficient sample size.

Data were analyzed using STATISTICA 6.0 (StatSoft Inc.). Statistical differences were examined by analysis of variance (ANOVA) for repeated measures, followed by homogeneity group *post hoc* comparison (Duncan, significance level set at $p = 0.05$) in order to contrast BL with PostBL, baseline with session phases and stimulation phases with pre- and post-stimulation phases. Log transformations were applied for the variance analysis of HRV power data.

Physiological changes over the entire EMDR session were accessed through the comparison between BL and PostBL levels. Furthermore, for each session and variable a linear regression analysis was performed on mean values of all 30 s long pre-stimulation periods as well as for all stimulation sets from which mean values were obtained for the interval between 20 and 50 s of stimulation (For HRV-measures full 50 s long periods were used). In order to test for session trends the proportion of regression slopes among all sessions of a certain sign was compared with a hypothetical random proportion of 0.5 with an equal number of cases using the 'Difference between two proportions test' (*t*-test; STATISTICA 6.0). Differences between pre- and during-stimulation phases were assessed by applying the same test on the proportions obtained for the pre-stimulation and during-stimulation period, respectively.

3. Results

All in all data from 13 individuals and 30 sessions were eligible and thus included in the analysis. The mean number of sessions included per individual was 2.27 (S.D. = 1.22; range = 1–5), with a mean session duration of 55.0 min (S.D. = 12.96). Because of missing values in some parameters, the degrees of freedom vary, however. The mean number of stimulation sets per session was 19.7 (S.D. = 11.0), with a mean stimulation duration of 44.2 s (S.D. = 10.8) and a mean interval of 58.7 s (S.D. = 21.9). The frequency of eye movements was typically 60 min^{-1} for the movement forth and back.

3.1. Within session trends

Reported SUDs ratings showed a significant decrease from 6.83 (S.D. = 2.57) when scored during the assessment phase to 2.88 (S.D. = 1.87) at the end of the EMDR-session ($n = 17$; $t = 6.07$; $p < 0.0001$), thus indicating at least partial desensitization. However, of 17 treatment sessions from which both pre- and post-EMDR scores where retrieved, complete desensitiza-

tion (as defined by SUDs of 0–1) occurred only in three. VoC scores increased from 4.10 (S.D. = 1.58) to 5.29 (S.D. = 1.59) over the EMDR-session ($n = 14$; $t = -2.65$; $p < 0.05$).

Baseline levels before and after session treatment differed significantly in FT and SC, both showing an increase, and in HR, which decreased (Table 1). However, during exposure to trauma, SC did not show any significant trends while FT and HR did (Table 2). The FT had stabilized before the eye movements began, and increased consistently during the treatment phase, as indicated by the proportion of positive regression slopes (Fig. 1), while the HR appeared to decrease successively more only for each eye movement stimulation set, as indicated by a high proportion of negative regression slopes, and not for the periods in between.

Also the HRV measures showed significant within treatment phase trends (Table 2). The relative balance between LF and HF gradually shifted towards the HF side, shown by the proportions of positive regression slopes which were high for normalized HF and low for normalized LF and LF/HF. As with HR, these regressions were only apparent for stimulation phases.

3.2. Physiological changes during-stimulation

Significant time effects were noticed for all physiological variables (Table 1 and Fig. 2).

RR increased significantly during the stimulation phase. On termination of the stimulation RR showed a sharp drop and returned to baseline level. (Subjects were asked to take a deep breath after eye movements.)

The oxycapnometric measurement indicated a small reduction in SpO_2 and an increase in mean CO_2 as well as $EtCO_2$ during eye movements.

When compared to pre-stimulation phases HR was significantly decreased after 10 s of stimulation. The lowered level remained during the entire stimulation period and was immediately followed by a sharp but transient increase in HR when the eye movements stopped (an example of the HR changes occurring during a stimulation set is given in Fig. 3). As mentioned above, the drop in HR on stimulation start appeared to become more and more pronounced as the session progressed. The deceleration in HR was accompanied by a change in HRV (Table 3). The overall HR variance typically decreased and the high frequency component became more prominent as indicated by a decreased LF/HF.

FT showed an increase during eye movements, indicating an increased peripheral blood flow, and decreased slowly after the stimulation phase ended.

During-stimulation, the SC showed a pattern of decline, indicating sympathetic nervous system de-arousal. The transition from stimulation to post-stimulation was characterized by a sharp increase in SC.

4. Discussion

In the present study, we have found that the eye movements during the EMDR-procedure are accompanied by a number of physiological changes. At the start of eye movements, the sympathetic drive is decreased as indicated by a fall in skin conductance and an increase in skin temperature, while the parasympathetic/vagal influence is increased, shown by heart rate deceleration and a changed balance between high frequency and low frequency heart rate variability. Respiration changes as well, the rate increases, the mean of exhaled as well the end-tidal carbon dioxide levels increase, and with some latency, the oxygen saturation, measured by a photometric method, decreases. Moreover, besides changes in immediate association with the eye movements, within-session trends are also found for the stimulation phases with successively decreasing heart rate, increasing fingertip temperature, and a decreasing LF/HF-ratio.

Few previous studies have followed physiological changes directly linked to the eye movement stimulation or across EMDR-treatment sessions. Wilson et al. found a pattern of change during EMDR-stimulation in accordance with the present data, with decreased HR (during initial sets) and reduced galvanic skin response (GSR), when applying single session EMDR-treatments to subjects distressed by disturbing memories (Wilson et al., 1996). The sharp increase in parasympathetic tone (as indicated by the root mean square of successive differences of adjacent inter-beat intervals (RMSSD)) and decrease in HR at stimulation-onset reported by Sack are also in accordance with our findings (Sack, 2005). Furthermore, Barrowcliff et al. found that arousal caused by white noise and measured as skin conductance was lowered during eye movements in comparison with a control condition (Barrowcliff et al., 2003), and Montgomery and Ayllon found a non-significant, but consistent, decrease in HR when exposure was combined with saccadic eye movements compared to exposure only in subjects meeting the criteria for PTSD (Montgomery & Ayllon, 1994).

Dunn et al., studying physiological responses to eye movements compared with fixed stare in a non-clinical

Table 1
Mean, standard deviation and homogeneity group indication for all measured physiological variables pre- (B), during (C, D) and post- (E) dual attention stimulation sets and pre- (A) and post- (F) the entire EMDR-session (Baselines)[a]

Variable	Baseline		Pre-stimulation			Stimulation					Stimulation end			Post-stimulation					Main effects (variable × phase)	
	A BL	F PostBL	B1 −30 to 20 s	B2 −20 to 10 s	B3 −10 to 0 s	C1 0–10 s	C2 10–20 s	C3 20–30 s	C4 30–40 s	C5 40–50 s	D1 −30 to 20 s	D2 −20 to 10 s	D3 −10 to 0 s	E1 0–10 s	E2 10–20 s	E3 20–30 s	E4 30–40 s	E4 40–50 s	*F* (d.f.)	*P*-value
CO_2 (%)	2.95 0.41 bc	2.94 0.43 abc	2.86 0.46 abc	2.85 0.46 abc	2.83 0.35 ab	3.00 0.40 bc	2.98 0.50 abc	3.02 0.47 bc	3.02 0.49 bc	3.08 0.49 c[b]	3.02 0.51 bc	2.98 0.52 abc	3.03 0.51 bc	2.76 0.51 a[b,c]	2.90 0.67 abc	2.89 0.61 abc	2.84 0.56 abc	2.79 0.52 ab	1.78 (10)	0.03
$EtCO_2$ (%)	4.62 0.19 abcd	4.48 0.52 a	4.61 0.40 abcd	4.58 0.38 abcd	4.51 0.34 ab	4.57 0.31 abcd	4.72 0.30 bcde	4.78 0.27 cde[b]	4.81 0.34 de[b]	4.90 0.50 e[b,c]	4.81 0.46 de	4.73 0.31 bcde	4.63 0.19 abcd	4.54 0.27 abc[b]	4.49 0.54 ab[b]	4.57 0.32 abcd	4.61 0.22 abcd	4.57 0.40 abcd	2.61 (10)	0.001
RR (bpm)	15.72 4.02 ab	15.38 4.04 abc	15.23 2.80 a	15.71 2.64 a	16.45 2.23 ab	17.44 3.49 abcd	18.44 4.37 bcde[b]	18.49 4.43 bcde[b]	19.51 4.02 cde[b,c]	19.78 3.99 de[b,c]	19.50 4.08 cde[c]	19.82 4.56 de[c]	20.31 4.67 e[c]	19.75 3.79 de[c]	16.13 3.20 ab[b]	15.90 3.37 ab[b]	15.10 3.93 a[b]	16.50 3.69 ab[b]	4.92 (10)	<0.001
SpO_2 (%)	95.27 0.97 cd	95.65 0.90 d	95.10 0.77 abc	95.05 0.86 abc	95.17 0.86 abcd	95.11 0.82 abc	95.07 0.91 abc	94.95 1.19 abc	94.83 1.36 ab[c]	94.97 1.22 abc	94.89 1.26 abc	94.91 1.13 abc	94.92 1.15 abc	94.88 1.14 abc	94.68 1.06 a[c]	95.01 0.64 abc	95.26 0.66 bcd	95.23 0.81 bcd	2.10 (10)	0.008
HR (bpm)	81.52 10.27 a	77.97 7.70 bcde[c]	78.99 9.43 bcd[c]	78.95 9.01 bcde[c]	79.49 9.05 b[c]	78.77 9.52 bcde[c]	77.60 9.20 cde[b,c]	77.79 9.58 bcde[c]	77.30 9.53 de[b,c]	77.68 9.41 bcde[c]	77.45 9.73 de[b,c]	77.37 9.90 de[b,c]	77.14 9.79 e[b,c]	78.87 9.85 bcde[c]	79.30 9.21 bc[c]	77.66 9.28 cde[b,c]	77.46 9.12 de[b,c]	78.71 8.74 bcde[c]	2.86 (12)	<0.001
SC (μMho)	12.70 5.09 a	14.02 6.10 d[c]	14.03 6.13 c[c]	14.00 6.10 c[c]	13.91 5.98 c[c]	13.74 5.83 c[c]	13.62 5.72 bc[c]	13.52 5.66 bc[c]	13.27 5.58 bc[c]	14.02 6.10 b[b,c]	13.46 5.69 bc[c]	13.43 5.72 bc[c]	13.43 5.67 bc[c]	13.54 5.69 bc[c]	13.81 5.84 c[c]	13.85 5.88 c[c]	13.76 5.85 bc[c]	13.94 6.05 c[c]	6.06 (12)	<0.001
FT (°C)	30.60 3.19 a	30.94 2.77 b[c]	31.34 3.01 cde[c]	31.29 3.01 cde[c]	31.25 2.99 c[c]	31.28 2.98 cd[c]	31.35 2.95 cde[c]	31.45 2.96 cdef[c]	31.56 2.97 ef[b,c]	31.65 2.99 f[b,c]	31.44 2.88 cdef[c]	31.46 2.88 cdef[c]	31.51 2.90 cdef[c]	31.54 2.89 def[c]	31.47 2.89 cdef[c]	31.44 2.91 cdef[c]	31.42 2.96 cdef[c]	31.39 2.92 cdef[c]	8.90 (12)	<0.001

Duncan *post hoc* test; significance level set at $p = 0.05$.

[a] ANOVA with repeated measures univariate design. Phases with no common letters are significantly different from each other.

[b] Stimulation phase differs from a pre-stimulation phase OR a post-stimulation phase differs from a stimulation phase.

[c] Phase mean differs from the pre-baseline level.

Table 2
Within session trends estimated by the proportion of positive linear regression slopes among all eligible sessions[a]

Variable	*N*	Phase	*R*, mean (S.D.)	*b*, mean (S.D.)	% +*b*	*P*	*P* (between)
CO_2	22	Pre-stimulation	0.21 (0.16)	−0.01 (0.04)	45.5	0.76	0.53
		Stimulation	0.28 (0.18)	0.03 (0.09)	54.5	0.76	
$EtCO_2$	22	Pre-stimulation	0.29 (0.17)	0.01 (0.09)	54.5	0.76	0.76
		Stimulation	0.36 (0.12)	−0.01 (0.16)	50.0	1.00	
RR	22	Pre-stimulation	0.25 (0.12)	0.02 (0.90)	36.4	0.37	0.37
		Stimulation	0.29 (0.10)	0.06 (1.45)	50.0	1.00	
SaO_2	22	Pre-stimulation	0.35 (0.14)	−0.03 (0.25)	36.4	0.37	0.13
		Stimulation	0.39 (0.23)	0.11 (0.36)	59.1	0.55	
HR	20	Pre-stimulation	0.38 (0.19)	0.02 (1.26)	31.6	0.26	0.04
		Stimulation	0.39 (0.25)	−1.54 (3.22)	5.3	0.004	
SC	30	Pre-stimulation	0.47 (0.24)	0.08 (0.61)	66.7	0.20	1.00
		Stimulation	0.59 (0.23)	−0.05 (0.64)	66.7	0.20	
FT	25	Pre-stimulation	0.54 (0.20)	0.16 (0.14)	80.0	0.03	0.71
		Stimulation	0.50 (0.22)	0.18 (0.12)	84.0	0.01	
Tot. power	20	Pre-stimulation	0.33 (0.25)	−1.47 (5.27)	47.4	0.87	0.34
		Stimulation	0.39 (0.23)	−2.16 (5.67)	31.6	0.27	
LF	20	Pre-stimulation	0.30 (0.21)	−0.74 (2.43)	36.8	0.42	0.48
		Stimulation	0.38 (0.00)	−1.22 (4.24)	26.3	0.14	
HF	20	Pre-stimulation	0.31 (0.25)	−0.63 (3.04)	47.4	0.87	1.00
		Stimulation	0.28 (0.21)	−0.22 (0.67)	47.4	0.87	
NLF	20	Pre-stimulation	0.29 (0.21)	0.54 (3.86)	31.6	0.27	0.04
		Stimulation	0.36 (0.20)	−2.82 (5.84)	5.3	0.004	
NHF	20	Pre-stimulation	0.29 (0.22)	−0.69 (3.75)	63.2	0.42	0.02
		Stimulation	0.32 (0.21)	2.64 (6.53)	94.7	0.004	
LF/HF	20	Pre-stimulation	0.28 (0.21)	0.10 (0.74)	47.4	0.87	0.006
		Stimulation	0.39 (0.22)	−0.,50 (0.56)	5.3	0.004	

N = number of sessions included in the analysis; *R* = mean linear regression coefficient ± the standard deviation of the mean; *b* = mean slope of the regression line ± the standard deviation of the mean; % +*b* = proportion of positive slopes; *P* = significance level for the comparison with a hypothetical 50% proportion; *P* (between) = significance level for the comparison between the proportions obtained for pre-stimulation and stimulation regressions, respectively. (Difference between two proportions *t*-test).

[a] Within session trends for the last 30 s before the onset of dual attention stimulation (pre-stimulation) and for a 30 s long period starting 20 s into the stimulation phase (stimulation).

sample of college students, did not present data linked directly to stimulation sets but reported a decreased HR from pre- to post-treatment and no significant trend in GSR (Dunn, Schwartz, Hatfield, & Wiegele, 1996). The same pattern of change in HR and GSR over repeated eye movement sets was also reported by Wilson et al. (1996). In contrast to our study, Dunn et al. did not find a significant trend in FT (Dunn et al., 1996). However, increasing FT across eye movement sessions have been reported both by Wilson et al. (1996) and by Friedberg (2004), the latter studying patients treated for fibromyalgia with EMD (i.e. a treatment protocol using eye movements but which differs from EMDR in some ways). The ambiguous results regarding the FT within-session trend could possibly be attributed to differences in clinical background of the subjects studied and/or to the temporal distance between the measure taking and the actual stimulation. As was noted in the present study, the increase in FT was closely linked to the saccadic eye movements and almost immediately started to decrease when stimulation ended. Dunn et al. compared FT readings from pre-treatment with post-treatment measures, both obtained when the subjects focused on a negative image, and did not report the elapsed time from the last stimulation set to the post-session recording. Thus, increased finger temperature seems to be a general effect of eye movements.

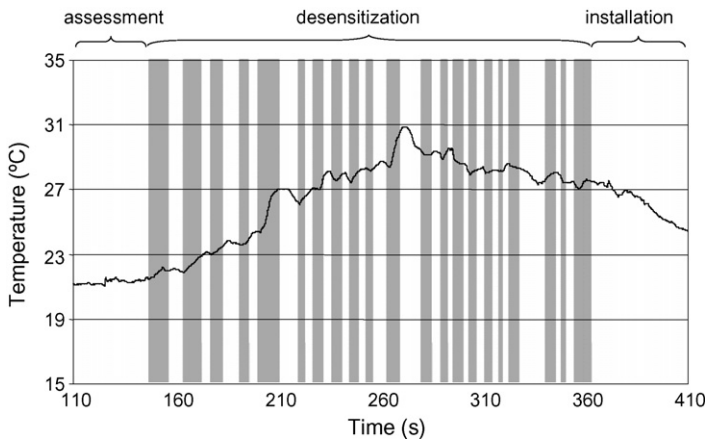

Fig. 1. Finger temperature changes over part of an EMDR-session including the ending of the assessment phase, the entire desensitization phase, and the beginning of the installation phase. Grayed areas represent periods of eye movement stimulation.

The physiological changes measured in our and previous studies indicate that the effects of the eye movements are beneficial and are coupled with a relaxation response. A clear-cut de-arousal in a narrow time frame around the eye movements, indicated by increased FT, decreased SC, and apparently vagal shifts in HR and HRV, and a within-session physiological habituation, evidenced by progressively decreasing HR, increasing FT and decreasing LF/HF-ratios across the treatment sessions, is meaningful from a therapeutic point of view, since it may help to uncouple stimulus and response elements during exposure treatment (Jaycox, Foa, & Morral, 1998; Nishith, Griffin, & Weaver, 2002). In contrast to the changes in heart rate, skin conductance and finger temperature, is the observed respiratory pattern, with an increased breathing frequency during-stimulation, not in agreement with a relaxation response. However, a more shallow and rapid breathing during eye movement stimulation was also noted by Wilson et al. who interpreted it as pacing of the breathing to the rhythm of the saccadic stimulation (Wilson et al., 1996). A changed breathing caused by involuntary pacing may lead to hypoventilation, which could explain the increased mean CO_2 and $EtCO_2$ levels, as well as reduced SpO_2, which were correlated with eye movement in this study.

One of the main hypotheses about the working mechanism of EMDR is that dual attention stimulation causes de-arousal by eliciting an orienting response (Armstrong & Vaughan, 1996). The initial sharp drop in HR and shift in autonomic balance in a vagal direction support this idea (Öhman et al., 2000). However, the orienting response hypothesis does not apply to all physiological trends associated with the eye movement phase in our data. For example, the skin conductance typically increases with the orienting response (Öhman et al., 2000); in our data it decreased during eye movements, a pattern also observed by Wilson et al. (1996) and Barrowcliff et al. (2003). Wilson et al. did report an initial increase of galvanic skin response within the first 10 s of stimulation before the decrease. However, they also suggested that this increase was distinct from an orienting response as eye movements alone without instruction to focus on a traumatic memory failed to produce this increase. Moreover, the orienting response is coupled to a slowing of breathing, probably as an adaptation to reduce respiratory noise. In our data respiratory rate increases immediately in response to eye movements, something which was noted by Wilson et al. (1996) as well. Furthermore, the increased skin temperature during eye movements found in the present study does not fit with the orienting response model. While no other studies report finger temperature in direct association with eye movements, it is noteworthy that Wilson et al. (1996) as well as Friedberg (2004) found that finger temperature increased over sessions. The vasoconstriction associated with the orienting response would instead lead to decreased finger temperature. Another characteristic pattern of the orienting response is a relatively fast habituation to repeated stimuli (Öhman et al., 2000). Thus, with repeated stimulations the decrease in heart rate could be expected to diminish. However, in the present study, the magnitude of the drop in heart rate actually increased over time with repeated sets of eye movements. Thus, an orienting response does not explain the physiological changes associated with dual attention stimulation observed in the present study.

Another of the main hypotheses of the putative mechanisms explaining the effects of EMDR posits that

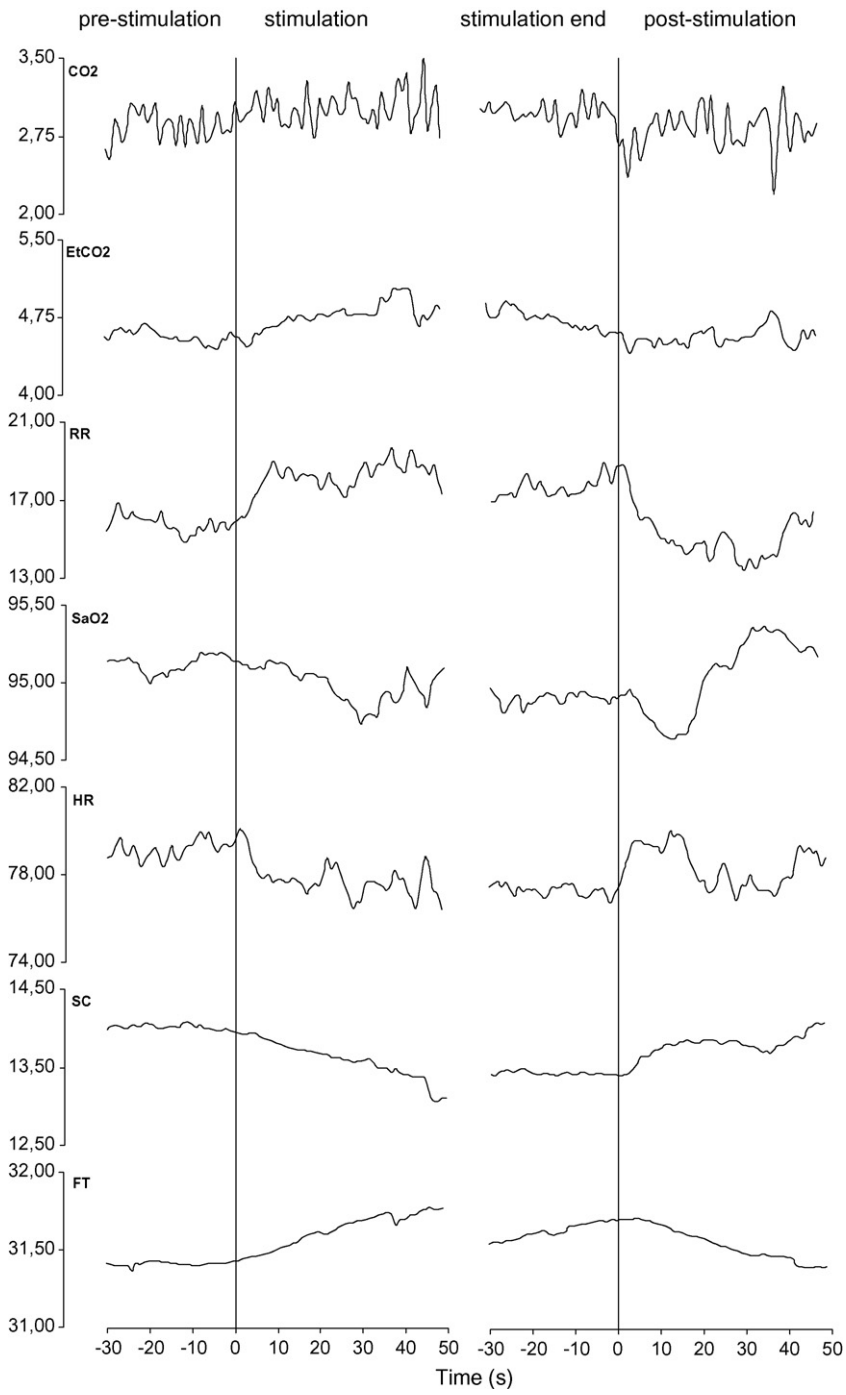

Fig. 2. Trend data for all measured physiological variables over the onset and ending of dual attention stimulation. Data represents the average of individual mean values for alignment points relative the starting and ending positions of eye movement stimulation sets with a resolution of 1 s.

the orienting response might lead to REM-like states (Stickgold, 2002). According to Stickgold the eye movements might kick-start the innate memory processing system in the brain activated during REM-sleep (Stickgold, 2002). This hypothesis is plausible because PTSD patients show REM-sleep deprivation due to extreme arousal caused by nightmares, which tend to wake them up during REM-sleep. Even if such a response might not be explained by the orienting response – as assumed by Stickgold – there are

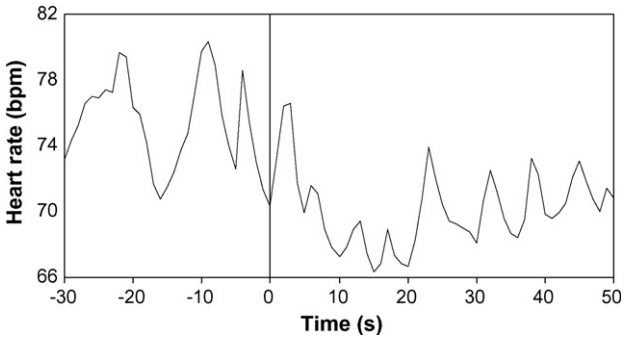

Fig. 3. A typical example of the heart rate (HR) pattern before (−30 to 0 s) and during (0–50 s) a dual attention stimulation set. As the stimulation start the mean HR and overall variance decrease, and the HR variability changes.

other possibilities such as reciprocal activation by eye movements of brain areas involved in REM-sleep. However, apparently the patients receiving dual attention stimulation are not sleeping, so even if dual attention stimulation would induce a neurophysiologic state similar to REM-sleep, it cannot be expected to yield the full range of autonomic changes associated with REM-sleep. Moreover, in contrast to the orienting response, which is a well defined reflex, testable using a few autonomic measures, REM-sleep is a complex state defined in relation to other sleep stages and has no well defined static autonomic profile. Although REM-sleep is a predominantly parasympathetic (vagal) state (Murali, Svatikova, & Somers, 2003; Stickgold, 2002) – reflected in a decreased cardiac activity, as well as by lowered SC compared with wakefulness (Kobayashi, Koike, Hirayama, Ito, & Sobue, 2003) – it is also characterized by rapid fluctuations in autonomic

Table 3
Mean value, standard deviation and homogeneity group indication for heart rate variability parameters before, during and after saccadic eye movement stimulation sets and before and after the entire EMDR-session

Measure	Band	Baseline		Stimulation		Post-stimulation		Main effects	
		Pre	Post	−50 to 0 s	0–50 s	−50 to 0 s	0–50 s	*F*	*P*-value
Power, Bpm^2	Total	18.59	18.31	20.30	13.74	13.72	19.98	1.74	0.14
		19.10	13.70	12.42	10.34	10.81	11.80		
		n.p.	n.p.	n.p.	n.p.	n.p.	n.p.		
	LF	10.62	8.54	11.04	6.78	7.05	10.46	2.92	0.02
		10.33	7.18	7.72	6.34	7.16	6.99		
		ab	ab	b	a[a]	a[a]	b[a]		
	HF	4.61	1.74	2.99	2.62	2.66	3.28	1.42	0.23
		8.38	1.23	3.72	4.07	4.23	4.46		
		n.p.	n.p.	n.p.	n.p.	n.p.	n.p.		
NU	LF	72.29	73.49	77.32	70.27	72.78	77.45	2.07	0.08
		13.75	14.45	10.58	13.44	10.81	10.50		
		n.p.	n.p.	n.p.	n.p.	n.p.	n.p.		
	HF	24.77	23.50	20.01	25.96	23.31	19.96	1.74	0.14
		13.10	12.70	9.65	12.02	8.78	9.92		
		n.p.	n.p.	n.p.	n.p.	n.p.	n.p.		
Ratio	LF/HF	5.01	5.70	6.61	4.27	4.94	6.15	2.51	0.04
		4.43	5.20	4.49	2.46	2.54	3.33		
		a	ab	b[b]	a[a]	ab	b[a]		

Abbreviations: NU = normalized units; LF = low frequency span between 0.04 and 0.15 Hz; HF = high frequency span between 0.15 and 0.4 Hz. ANOVA with repeated measure multivariate design (d.f. = 12). Phases with no common letters are significantly different from each other. (Duncan *post hoc*-test; significance level set at $p = 0.05$). n.p. = no *post hoc*-test performed.

[a] A during-stimulation phase differs from a pre-stimulation phase or a post-stimulation phase differs from a stimulation phase.

[b] A phase mean differs from the pre-baseline level.

tone (Murali et al., 2003) associated with the rapid eye movement bursts. It is hardly surprising that studies of heart rate variability during REM-sleep have shown conflicting results (Monti, Medigue, Nedelcoux, & Escourrou, 2002). Thus, the rejection or acceptance of the REM-sleep hypothesis of EMDR is not within the reach of the present study. Still, as a working model, the REM-sleep hypothesis does have explanatory power when applied to our data. One of the most consistent pattern of REM-sleep is a depressed thermoregulatory response, with a decreased difference between core and peripheral temperature (Glotzbach & Heller, 2000). The immediate increase of skin temperature during eye movements and the decrease immediately after eye movements support the REM-related hypothesis because the thermoregulatory control of core-to-peripheral temperature is suspended in REM-sleep, and episodes of phasic REM-sleep is closely linked to rapid temperature increases in the extremities, at least during non-extreme ambient temperatures (Dewasmes, Bothorel, Candas, & Libert, 1997; Henane, Buguet, Roussel, & Bittel, 1977). Furthermore, the eye movements during REM-sleep are associated with hypoventilation and rapid shallow breathing (Douglas, White, Pickett, Weil, & Zwillich, 1982; Gould et al., 1988; Millman et al., 1988), and due to a decrease in minute ventilation the levels of end-tidal PCO_2 is increased (Schäfer & Schläfke, 1998). Also end respiratory PO_2 decreases during REM-sleep (Douglas, White, Pickett et al., 1982), and at least patients with respiratory disorders become more hypoxemic during bursts of eye movements (Douglas et al., 1979; Douglas, White, Pickett et al., 1982). The ventilatory responses to both hypoxemia (Douglas, White, Weil, Pickett, Martin et al., 1982) and hypercapnea (Douglas, White, Weil, Pickett, & Zwillich, 1982) are also decreased during REM-sleep, so the normal defenses against the development of such states are impaired. Thus, when comparing the typical autonomic pattern for REM-sleep with the physiological changes observed during induced eye movements in the present study, we find similarities in several measured variables, including a vagal shift as indicated by decreased heart rate and skin conductance, a change in the respiratory pattern with an increased frequency and a tendency for the subject to become more hypercapnic and hypoxemic, and finally a change in finger temperature.

Since the present study followed a naturalistic design, without control conditions, other explanations than the eye movements *per se* to the observed physiological changes cannot be excluded; redirection of inner focus may have physiological manifestations and one must also consider the possibility of placebo effects. On the other hand, placebo effects tend to habituate; in contrast with the effects of eye movements found in this study. At least some of the measured parameters are likely to be affected by changes in bodily and behavioral functions directed by the treatment protocol. For example, between stimulation sets, the subjects were talking, while they were silent during the actual eye movements. Speaking is known to interfere with cardiovascular functions as well as respiration. Bernardi et al. found that free talking, compared to spontaneous breathing, increased HR and LF power and a non-significant tendency to increased total variability (Bernardi et al., 2000). They attributed these changes to increased respiratory rate. However, in our study the respiratory rate increased during eye movements, making the putative effect of silence during-stimulation sets on at least the HRV less likely to have contributed to the result.

This study and previous studies has demonstrated a number of effects of eye movements during EMDR or other conditions. Eye movements might not be necessary for desensitization, as shown by the equally good treatment results during behavioral exposure (Bradley et al., 2005). However, eye movements might involve another mechanism of desensitization, and in consequence, might be applicable in different patient groups who cannot tolerate behavioral exposure. Further, the added burden of many hours of homework associated with behavioral exposure should be considered as well (Rothbaum, Astin, & Marsteller, 2005). It is also interesting to note that Christman et al. found that bilateral eye movements enhance the retrieval of episodic memories (Christman, Garvey, Propper, & Phaneuf, 2003). This is consistent with the clinical experience that eye movements often are accompanied by retrieval of previously forgotten or disregarded information. New information activated during treatments sessions often serves to reframe the traumatic experience. Other studies of the eye movement component have indicated a reduction in negative affect, and decrease in the vividness of negative imagery, which may also be viewed as concomitants of the desensitization effect observed in this study (e.g., Andrade, Kavanagh, & Baddeley, 1997; Barrowcliff et al., 2003; Kavanagh, Freese, Andrade, & May, 2001; van den Hout, Muris, Salemink, & Kindt, 2001).

In future research, it seems important to replicate this study and see whether the eye movements can be tied more directly to treatment effects in other groups with PTSD. With regard to the increased breathing, increased CO_2, and decreased SpO_2, it would also be interesting to

study metabolic activity in the brain during EMDR in order to see whether the effects are related to increased oxygen consumption during eye movements due to increased brain activity, or if they are explained better by the cholinergic shift caused by the eye movements.

The present study shows the importance of further studies of eye movements as well as dismantling studies of EMDR. The association of treatment effects and eye movements or alternative stimulation have been addressed by Wilson et al., who found no effect of a tapping or a time interval condition on symptoms as well as physiology (Wilson et al., 1996). Further studies are needed to extricate the precise roles of alternative forms of bilateral stimulation. In future studies, a control condition leading to a shift of attention should be included in order to examine the possibility that eye movements are confounded by shift of attention. Brain activity during eye movements and EMDR sessions should be explored, however difficult that might be. It is also desirable in future studies to compare physiological measurements during behavioral exposure and EMDR.

Acknowledgements

Fredrik Garpe, Ulf Hansson, Solveig Eriksson Öhman och Ingrid von Schèele are gratefully acknowledged for valuable help during data collection. This investigation was supported by a grant from The Swedish Foundation for Health Care Sciences and Allergy Research (The Vårdal Foundation).

No funders or sponsors were involved in the planning, design, or interpretation of the study.

The corresponding author had full access to all of the data in the study and takes full responsibility for the integrity of the data and the accuracy of the data analysis.

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