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Neurophysiological correlates of eye movement desensitization and reprocessing sessions: preliminary evidence for traumatic memories integration

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Summary

We have investigated the potential role of eye movement desensitization and reprocessing (EMDR) in enhancing the integration of traumatic memories by measuring EEG coherence, power spectra and autonomic variables before (pre-EMDR) and after (post-EMDR) EMDR sessions during the recall of patient's traumatic memory. Thirteen EMDR sessions of six patients with post-traumatic stress disorder were recorded. EEG analyses were conducted by means of the standardized Low Resolution Electric Tomography (sLORETA) software. Power spectra, EEG coherence and heart rate variability (HRV) were compared between pre- and post-EMDR sessions. After EMDR, we observed a significant increase of alpha power in the left inferior temporal gyrus ($T = 3{\cdot}879$; $P = 0{\cdot}41$) and an increased EEG coherence in beta band between C3 and T5 electrodes ($T = 6{\cdot}358$; $P<0{\cdot}001$). Furthermore, a significant increase of HRV in the post-EMDR sessions was also observed (pre-EMDR: $6{\cdot}38 \pm 6{\cdot}83$; post-EMDR: $2{\cdot}46 \pm 2{\cdot}95$; U-Test= 45, $P = 0{\cdot}043$). Finally, the values of lagged coherence were negatively associated with subjective units of disturbance ($r(24) = -0{\cdot}44$, $P<0{\cdot}05$) and positively associated with parasympathetic activity ($r(24)=0{\cdot}40$, $P<0{\cdot}05$). Our results suggest that EMDR leads to an integration of dissociated aspects of traumatic memories and, consequently, a decrease of hyperarousal symptoms.

Introduction

Eye movement desensitization and reprocessing (EMDR) (Shapiro, 1995) is a well-validated therapy for trauma-related emotional disorders, and it is recognized as one of the most effective and fastest recovery treatments for trauma in several practice guidelines worldwide (Foa *et al.*, 2009; World Health Organization, 2013). Indeed, a large amount of controlled empirical studies and meta-analyses demonstrated the effectiveness of EMDR for treating traumatic memories in post-traumatic stress disorder (PTSD) and other clinical outcomes of traumatic experiences (Bisson & Andrew, 2007; Rodenburg *et al.*, 2009). EMDR also showed its effects both at neurobiological (Nardo *et al.*, 2010; Bossini *et al.*, 2011; Pagani *et al.*, 2012) and psychophysiological levels (Elofsson *et al.*, 2008; Sack *et al.*, 2008).

Despite the evidence of its effectiveness and the increasing number of studies investigating the cognitive and neurobiological bases of its effects, the functioning of EMDR, including the crucial role of eye movements (EMs), is still unclear (Bergmann, 2010; Nardo *et al.*, 2010; Pagani *et al.*, 2012).

Different explanations have been proposed to account for EMDR's mechanisms of action (Bergmann, 2010). Several scholars agree in considering that EMDR leads to an adaptive integration of traumatic memories (Shapiro, 1995; van der Kolk, 2002; Stickgold, 2002; Bergmann, 2008). It has been proposed that the neurophysiological substrates of the EMDR's integrative power could be based on an increase of functional connectivity in cortical networks, especially between the two hemispheres (Bergmann, 2008; Propper & Christman, 2008).

Actually, dynamic states of the cerebral cortex, characterized by a high degree of functional connectivity between widespread distributed neurons, have been demonstrated to underpin higher-order integrative mental functions for cognitive and affective stimuli processing (Miskovic & Schmidt, 2010; Santangelo & Macaluso, 2013). On the other hand, EEG connectivity abnormalities have been observed in patients with minimally conscious states (Leon-Carrion *et al.*, 2012), in several psychopathological conditions (Hopper *et al.*, 2002; Sato *et al.*, 2012; Farina *et al.*, 2013), in memory disturbances (Stam *et al.*, 2009; Sankari *et al.*, 2011) and are thought as possible outcomes of psychological trauma (Ito *et al.*, 1998; Cook *et al.*, 2009; Miskovic *et al.*, 2010).

These cortical networking features can be assessed by non-invasive methods such as electroencephalography (EEG) coherence (Miskovic & Schmidt, 2010; Uhlhaas *et al.*, 2010).

Coherence is often interpreted as a measure of 'coupling' and functional association between two brain regions (Nunez *et al.*, 1997). Hence, coherence is a sensitive measure that can reveal subtle aspects of the network dynamics of the brain, which complement the data obtained by power spectral analyses.

Previous EEG coherence studies testing functional connectivity in EMDR provided controversial findings. Propper *et al.* (2007), investigating the difference of EMs and other forms of sensory stimulation used in EMDR, reported that, compared with a central fixation condition, EMs was associated with a decrease frontal interhemispheric gamma EEG coherence. Propper *et al.* (2007) observed that a decrease in interhemispheric EEG coherence does not necessarily indicate a decrease in functional interhemispheric interaction, suggesting that the communication between the cerebral hemispheres may have an inhibitory nature (Propper & Christman, 2008).

Unfortunately, these findings were not replicated by healthy subjects during a memory task, Samara *et al.* (2011). Recently, Pagani *et al.* (2012) reported significant decrease of connectivity between left visual cortex and right fusiform gyrus in the theta band after the autobiographical recollection of a traumatic event, in patients with PTSD.

The aim of this study was to test the potential role of EMDR in increasing functional connectivity by measuring modifications of EEG coherence and autonomic variables before and after EMDR sessions using a symptom provocation paradigm. To evaluate the specific effect of EMDR on traumatic memories recall, we compared EEG coherence, EEG power spectra and autonomic variables (heart rate variability (HRV)) 5 min before and 5 min after EMDR sessions during the recall of patient's traumatic memory (TM).

Materials and methods

Participants

The present study included six patients (three women, mean age = 45·77 ± 14·20; age range: 18–60 years) who referred to a specialized trauma centre for treatment of trauma-related psychological disorders. All were diagnosed with PTSD according with DSM-IV TR (APA, 2000) criteria, following a detailed clinical interview. No comorbidities were observed. Exclusion criteria were: left handedness; history of medical, neurological diseases; psychiatric comorbidity; head trauma; assumption of central nervous system-active drugs in the 3 weeks before the study; presence of EEG abnormalities at the baseline recording.

After receiving information about the aims of the study, all participants gave their written consent. Diagnosis and treatment were carried out by PCG, AO and ML who are all trained and having more than 10 years clinical experience administering EMDR.

The number of EMDR sessions followed each patient's individual needs and ranged between 1 and 4 sessions. Finally, we have analysed 13 sessions. EMDR treatment strictly followed all the eight phases protocol suggested by Shapiro (1995).

The research was approved by the Catholic University's and Università Europea's ethics review boards.

Procedures

Electroencephalography and ECG were continuously recorded during the experiment. Electrodes and sensors were placed before the EMDR treatment began. Continuous recordings were performed before (pre-EMDR), during, and after (post-EMDR) EMDR sessions.

Subjects in pre-EMDR and in post-EMDR session patients were invited to sit in a comfortable armchair, with eyes closed, and were instructed to recall and concentrate on their traumatic autobiographical event (two gun aggressions; rape; family murder; fire victim; and physical aggression). The patients were asked to remember as many details as possible about that experience. At the end of pre-EMDR and post-EMDR sessions, subjects were asked if they were able to successfully recall their traumatic memories, and SUDs were recorded.

EEG recordings

Electroencephalography was recorded by means of a Micromed System Plus digital EEGraph (Micromed© S.p.A., Mogliano Veneto, TV, Italy). EEG montage included 19 standard scalp leads positioned according to the 10–20 system (recording sites: Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2), EOG and EKG. The reference electrodes were placed on the linked mastoids. Impedances were kept below 5KΩ before starting the recording and checked again at the end. In particular, impedances of the mastoids reference electrodes were checked to be identical. Sampling frequency was 256 Hz, A/D conversion was made at 16 bit, preamplifiers amplitude range was ±3200 μV, and low-frequency prefilters were set at 0·15 Hz.

Artefact rejection (eye movements, blinks, muscular activations or movement artefacts) was performed visually on the raw EEG trace. After artefact rejection, the remaining EEG intervals were exported into American Standard Code for Information Interchange (ASCII) files, and imported into the sLORETA software. At least 120 s of EEG recording were analysed for each condition (pre-EMDR vs. post-EMDR), in all subjects for each EMDR sessions. This procedure has been already used to investigate modifications of EEG power spectra in working memory task (Imperatori et al., 2013) and modification of EEG coherence in patients with dissociative disorders (Farina et al., 2013). All EEG analyses were performed by means of the sLORETA software (Pascual-Marqui et al., 1994).

Frequency analysis

Electroencephalography frequency analysis was performed by means of a fast Fourier transform algorithm, with a 2 seconds interval on the EEG signal, in all scalp locations. The following frequency bands were considered: delta (0·5–4 Hz); theta (4·5–7·5 Hz); alpha (8–12·5 Hz); beta (13–30 Hz); gamma (30·5–100 Hz). For frequency analysis, monopolar EEG traces (each electrode referred to joint mastoids) were used. Topographical sources of EEG activities were determined using the sLORETA software. The sLORETA software computes the current distribution throughout the brain volume. To find a solution for the 3-dimensional distribution of the EEG signal, the sLORETA method assumes that neighbouring neurons are simultaneously and synchronously activated. This assumption rests on evidence from single cell recordings in the brain that shows strong synchronization of adjacent neurons (Kreiter & Singer, 1992; Murphy et al., 1992). The computational task is to select the smoothest of all possible 3-dimensional current distributions, a common procedure in signal processing (Grave de Peralta-Menendez & Gonzalez-Andino, 1998; Grave de Peralta Menendez et al., 2000). The result is a true 3-dimensional tomography, in which the localization of brain signals is preserved with a low amount of dispersion (Pascual-Marqui et al., 1994).

Connectivity analysis

The connectivity analysis was performed by the computation of lagged coherence. This approach allows to better evaluate 'true' connectivity. Respect to instantaneous coherence, lagged coherence is a much more appropriate measure of electrophysiological connectivity, because it removes the confounding effect of instantaneous dependence due to volume conduction and low spatial resolution (Pascual-Marqui, 2007). The LORETA software computes lagged coherence $\rho_{x\leftrightarrow y}(\omega)$,by the formula (Pascual-Marqui, 2007):

$$\rho^2_{x\leftrightarrow y}(\omega) = 1 - \exp[-F_{x\leftrightarrow y}(\omega)]$$
$$= 1 - \frac{\left\langle \begin{vmatrix} S_{yyw} & S_{yxw} \\ S_{xyw} & S_{xxw} \end{vmatrix} \Big/ \begin{vmatrix} S_{yyw} & 0 \\ 0^T & S_{xxw} \end{vmatrix} \right\rangle}{\left\langle \left| \mathrm{Re}\begin{pmatrix} S_{yyw} & S_{yxw} \\ S_{xyw} & S_{xxw} \end{pmatrix} \right| \Big/ \left| \mathrm{Re}\begin{pmatrix} S_{yyw} & 0 \\ 0^T & S_{xxw} \end{pmatrix} \right| \right\rangle}$$

In this formulas 'ω' is the discrete frequency considered, 'Re' indicates the real part of an element; $S_{xx\omega}$, $S_{yy\omega}$, $S_{xy\omega}$ and $S_{yx\omega}$ denote complex-valued covariance matrices. $F_{x\leftrightarrow y}(\omega)$ is the lagged linear dependence, 'O' is a matrix of zeros and the superscript 'T' means transpose.

The EEG coherence analysis was performed on the same blocks of EEG tracings used for power spectra analysis. Coherence values were computed for each frequency band (delta, theta, alpha, beta, gamma), in the frequency range of 0·5–100 Hz. To evaluate the modifications of connectivity, 19 regions of interests (ROIs) were defined corresponding to the site of the electrode (one for each scalp electrode). We chose the 'single nearest voxel' option: in this way, each ROI consisted of a single voxel, the one which is closest to each seed. Then, the eLORETA computed the coherence values between all these ROIs (total 19 × 19 = 361 connections).

Heart rate and heart rate variability

Heart rate (HR) and heart rate variability (HRV) were carried out on the EKG trace obtained during the EEG registration. The EKG was recorded using a modified lead II derivation (with the right shoulder negative and the left lower torso positive). Sampling rate was 256 Hz, with a digital resolution of 16 bits per sample. Impedance was kept below 5 KΩ. EKG registration was performed at the same times of EEG power spectra and EEG coherence (pre-EMDR versus post-EMDR) in all sessions.

Heart rate is modulated on a beat-to-beat basis by the combined effects of the sympathetic and parasympathetic nervous system on the sino-atrial node. HRV is a measurement of changes in HR over time, which provides information about autonomic functioning (Stein & Pu, 2012). HRV can be analysed both in the time domain and in the frequency domain. Parameters of HRV in the time domain are statistical measures derived from the beat file. In the frequency domain, two major components can be calculated from power spectral frequency analysis performed on a plot of R–R intervals, named tachogram (Task Force of the European Society of Cardiology & the North American Society of Pacing & Electrophysiology, 1996). High-frequency spectral component (HF) is associated with parasympathetic activation; whereas low-frequency spectral component (LF) reflects both sympathetic and parasympathetic activation. The LF/HF ratio is a dimensionless measure which is believed to reflect the sympatho-vagal balance, that is the ratio of sympathetic to vagus nerve traffic to the heart (Eckberg, 1997). Therefore, increases in LF/HF ratio reflect an increase in sympathetic functioning, and that overall decreases in LF/HF ratio reflect an increase in parasympathetic functioning.

Artefact rejection was performed visually; periods of EKG recording characterized by ventricular extrasystoles, movements and muscular or other artefacts were excluded from the analysis. A dedicated software (Rembrandt SleepView, Medcare®, Broomfield, CO, USA) recognized the individual electrocardiographical R wave peaks and calculated the R–R intervals (tachogram). Successively, the tachogram, an Excel file, was converted into an ASCII file and analysed by means of a dedicated software, freely available from the Web (HRV Analysis Software, Biomedical Signal analysis Group, Department of Applied Physics, University of Kuopio, Finland) (Niskanen et al., 2004).

Heart rate variability analysis was performed both in a time domain and in a frequency domain. Since many of the measures correlate closely with others, the following parameters were considered in the time domain: mean and standard deviation of heart rate (HR); mean and standard deviation of R–R intervals. In the frequency domain, HRV was analysed using the parametric autoregressive model analysis which allowed for an accurate estimate of power spectral density (PSD) when analysing short time intervals during which the signal is supposed to maintain stationarity. The frequency bands considered were the low-frequency (LF, 0·04–0·15 Hz) and the high-frequency (HF, 0·15–0·4 Hz) ones. In the frequency domain, the power of LF and HF bands were expressed in normalized units (nu), and the LF/HF ratio was calculated. Normalization was performed using the formula: $Z = \frac{X-\mu}{\sigma}$ where $\mu = E[X]$ is the mean and $\sigma = \sqrt{Var(X)}$ is the standard deviation of the probability distribution of X. A detailed description of HRV analysis, standards of measurement, physiological interpretation and clinical use is available in the report by the Task Force of the European Society of Cardiology & the North American Society of Pacing & Electrophysiology (1996).

Statistical analysis

Power spectra analysis and EEG lagged coherence were compared between pre-EMDR and post-EMDR in both experiments for each frequency band. Comparisons were performed using the statistical non-parametric mapping (SnPM) methodology supplied by the sLORETA software (Nichols & Holmes, 2002). This methodology is based on the Fisher's permutation test: a subset of non-parametric statistics. In particular, this is a type of statistical significance test in which the distribution of the test statistic under the null hypothesis is obtained by calculating all possible values of the test statistic under rearrangements of the labels on the observed data points. Correction of significance for multiple testing was computed for the two comparisons between pre-EMDR and post-EMDR for each frequency band: for the correction, we applied the non-parametric randomization procedure available in the sLORETA program package (Nichols & Holmes, 2002).

T-level threshold was computed by the statistical software implemented in the sLORETA, which correspond to threshold of statistical significance ($P<0·05$ and $P<0·01$) (Friston et al., 1991).

Heart rate, HRV and SUD comparison between pre-EMDR and post-EMDR were performed with a non-parametric test, Mann–Whitney U-test. Significance level was set at $P<0·05$. Correlation was tested between SUD, EEG coherence values, and HR and HRV parameters by means of Pearsons's correlation coefficient (r).The critical value of the Pearson's product–moment correlation coefficient was set to $r(24) = 0·388$, corresponding to a significance level $P<0·05$. Statistics were performed using the Statistical Package for Social Science (SPSS®, Armonk, NY, USA) software version 19.

Results

Electroencephalography and EKG recordings suitable for the analysis were obtained in all 13 sessions. Visual evaluation of the EEG recordings showed no relevant modifications of the background rhythm frequency, focal abnormalities or epileptic discharges. No subject showed evidence of drowsiness or sleep during the recordings.

Power spectra analysis

In the comparison between pre-EMDR and post-EMDR sessions, the threshold for significance was $T = 3·754$, corresponding to $P<0·05$, and $T = 4·492$, corresponding to $P<0·01$.

Significant modifications were observed reported in the alpha band ($T = 3·879$, corresponding to $P = 0·41$). In the post-EMDR condition, increased power of alpha activity was observed in the left temporal lobe and sLORETA software localized these modification in the left inferior temporal gyrus (Brodmann Area, BA 20) (Fig. 1). No significant differences were observed in the other frequency bands.

Lagged coherence analysis

In the comparison between pre-EMDR and post-EMDR, the threshold for significance was $T = 5·098$, corresponding to $P<0·05$, and $T = 6·202$, corresponding to $P<0·01$. In the post-EMDR condition, significant modifications were observed in the beta band ($T = 6·358$ corresponding to $P<0·001$). This modification was associated with an increase of lagged coherence in the left hemisphere, in particular between the cortical areas explored by C3 and T5 electrodes (Fig. 2a). No significant differences were observed in the other frequency bands for other electrode pairs.

HR, HRV and SUD results

In the time domain, no significant differences were reported for all considered parameters. However, significant tendency was observed in RR mean (pre-EMDR: 0·78 ± 0·08; post-EMDR: 0·84 ± 0·06; U-Test=122, $P = 0·054$) and in HR mean (pre-EMDR: 79·28 ± 10·76; post-EMDR: 72·48 ± 5·44; U-Test=48, $P = 0·061$). In the frequency domain, increased HF component was observed in post-EMDR experiment (pre-EMDR: 20·05 ± 14·03; post-EMDR: 35·61 ± 15·13; U-Test=127, $P = 0·029$). No modifications of the LF component were observed. The sympatho-vagal balance, expressed by the LF/HF ratio, was significantly decreased in the post-EMDR sessions (pre-EMDR: 6·38 ± 6·83; post-EMDR: 2·46 ± 2·95; U-Test=45, $P = 0·043$). Detailed results of HR and HRV parameters are shown in Table 1.

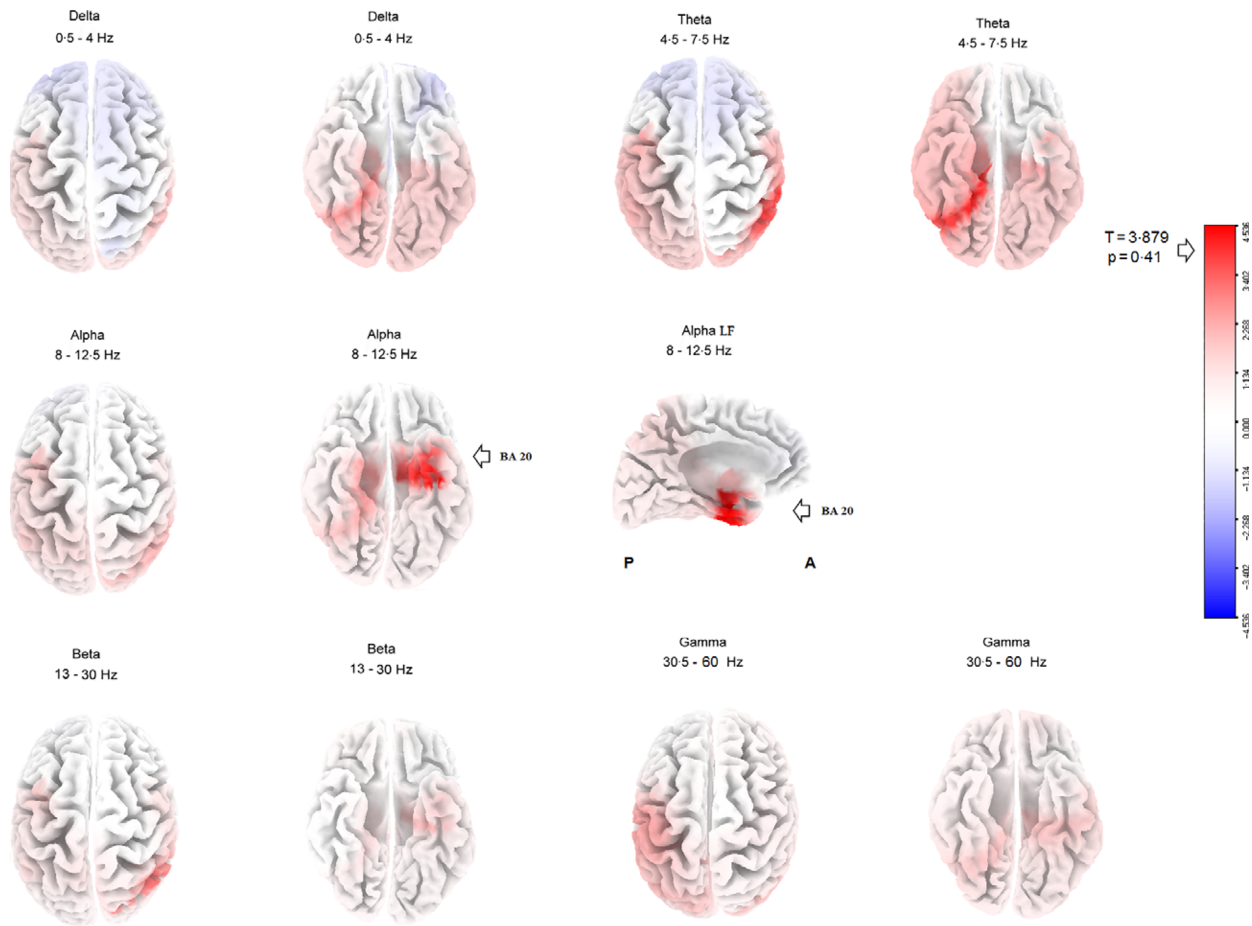


Figure 1 Results of the sLORETA comparison of EEG power spectra in all bands (pre-EMDR versus post-EMDR). Threshold values (T) are reported in the lower right corner. In the post-EMDR sessions, a significant increase power of alpha activity was observed in BA 20. A, anterior; P, posterior; LF, left hemisphere; BA, Broadmann Area.

In the post-EMDR condition, a significant reduction of SUD was also observed (pre-EMDR: 7·54 ± 2·03; post-EMDR: 2·38 ± 2·60; U-Test=169, $P<0·001$).

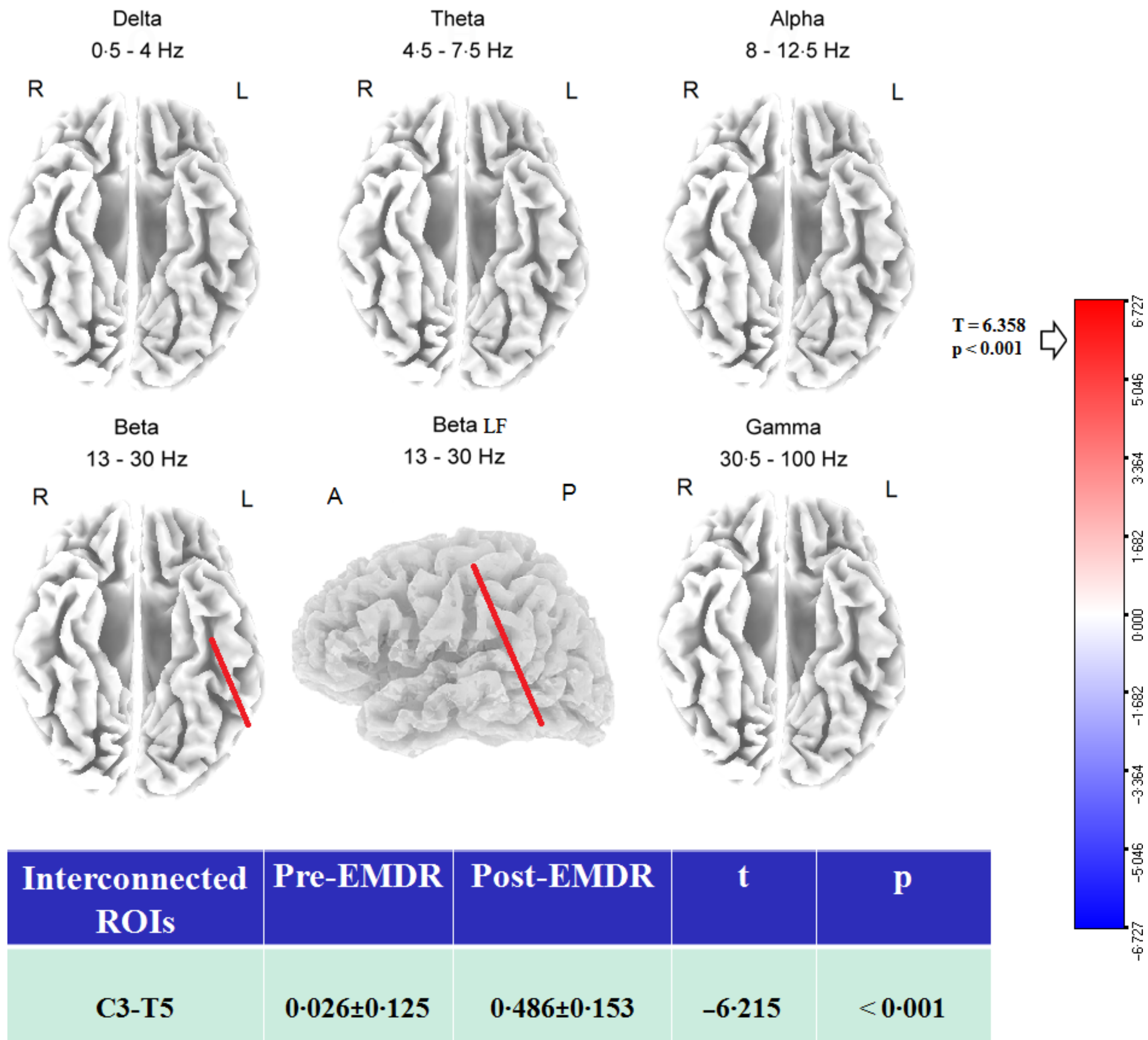
Association between SUD, significant interconnected ROIs, HR and HRV parameters

Significant correlations were also observed. The values of lagged coherence between the C3 and T5 ROIs were negatively associated with SUD ($r(24) = -0·44$, $P<0·05$) and positively associated with HF component ($r(24)=0·40$, $P<0·05$). Furthermore, significant negative correlations were also observed between SUD and RR mean ($r(24) = -0·435$, $P<0·05$) and between SUD and HR mean ($r(24) = -0·42$, $P<0·05$). Detailed correlations are listed in Table 2.

Discussion

The main aim of this study was to explore the modifications of EEG coherence associated to EMDR sessions test the potential role of EMDR in enhancing functional connectivity. Every EMDR sessions in our sample was effective in improving the symptoms, as demonstrated by SUD score reduction. After EMDR during TM recall, we observed increase of left intra-hemispheric EEG coherence, between fronto-parietal and temporal cortical areas (explored by C3 and T5 electrodes), in the beta frequency band. Moreover, we also observed a significant increase of EEG alpha power in the left inferior temporal gyrus.

It is possible to hypothesize that the increase in lagged coherence observed in our study is a direct measure of the increased functional connectivity of reprocessing aspect of EMDR and an indirect measure of the desensitization aspect of EMDR as indicated by the reduction in SUDs. Recent data indicate that dynamic and widespread cortical connectivity networks, explored by means of EEG coherence in the high-frequency range, have been found to play a crucial role in high-level integrative cognitive functions such as processing of affective stimuli (Miskovic & Schmidt, 2010), working memory (Santangelo & Macaluso, 2013), autobiographical memory (Imperatori *et al.*, 2014) and state of consciousness (Leon-Carrion *et al.*, 2012; Farina *et al.*, 2013).



Interconnected ROIs	Pre-EMDR	Post-EMDR	t	p
C3-T5	0·026±0·125	0·486±0·153	−6·215	< 0·001

Figure 2 Results of the sLORETA comparison of EEG lagged coherence in all frequency bands (pre-EMDR vs post-EMDR). Threshold values (T) are reported in the right of the figure. In the post-EMDR sessions, a significant increase of coherence was observed in beta band between C3 and T5 electrodes A, anterior; P, posterior; R, right; L, left; A, anterior; P, posterior; LF, left hemisphere.

Table 1 The comparison of SUD, HR and HRV parameters between pre- and post-EMDR.

	pre-EMDR (N = 13)	post-EMDR (N = 13)	Mann–whitney U-test	P
SUD	7·54 ± 2·03	2·38 ± 2·60	12	0·001***
RR mean	0·78 ± 0·08	0·84 ± 0·06	122	0·054
RR SD	0·08 ± 0·13	0·07 ± 0·06	114·5	0·124
HR mean	79·28 ± 10·76	72·48 ± 5·44	48	0·061
HR SD	6·41 ± 6·22	5·78 ± 5·26	81·5	0·878
LF	61·23 ± 19·56	55·28 ± 20·63	71	0·489
HF	20·05 ± 14·03	35·61 ± 15·13	127	0·029*
LF/HF	6·38 ± 6·83	2·46 ± 2·95	45	0·043*

*P<0·05; **P<0·001;
Note: SUD=Subjective Units of Disturbance; RR, R wave to R wave interval; SD, Standard deviation; HR, heart rate; LF, low-frequency spectral component; HF, High-frequency spectral component.

Table 2 Values of Pearsons's correlation coefficient between values of lagged coherence (C3-T5 Rois) and SUD, HR and HRV parameters during pre- and post-EMDR sessions. Significant correlations are in bold font with stars (*).

	SUD	RR mean	RR DS	HR mean	HR DS	LF	HF	LF/HF
Lagged coherence between ROIs C3-T5	**−0·44***	0·37	−0·12	−0·37	−0·10	0·10	**0·40***	−0·27
SUD		**−0·44***	0·08	**0·42***	0·04	0·13	−0·22	0·22

*$P<0.05$.

Note: SUD, Subjective Units of Disturbance; ROIs, Regions of Interest; RR, R wave to R wave interval; SD, Standard deviation; HR, heart rate variability; LF, low-frequency spectral component; HF, High-frequency spectral component.

Nevertheless, we could not demonstrate any significant modification of inter-hemispheric connectivity, as suggested by several authors (Stickgold, 2002; Propper *et al*., 2007; Bergmann, 2008; Propper & Christman, 2008).

Furthermore, EMDR was followed by increase of the HF component of HRV; as HF is known to reflect vagal activity, this finding suggests an increase of parasympathetic tone after EMDR sessions. Coherence values, in the C3-T5 cortical ROIs, were directly related with HF values, and inversely related with SUD score.

Taken together, our results could support the hypothesis that EMDR fosters an adaptive integration of the traumatic memory by enhancing cortical connectivity and consequently decreasing hyperarousal symptoms. Indeed, integration, defined as the '*the capacity of a system to collect information of different nature and combine it to produce new, useful, information*' (Zamora-Lopez *et al*., 2011, pp. 2), seems to be dramatically altered in patients with PTSD (Shapiro, 1995; van Der Kolk *et al*., 1997; Bergmann, 2008) and in other trauma-related disorders (Farina *et al*., 2013).

Inducing the TM after EMDR increased functional connectivity between left fronto-parietal and temporal ROIs. Functional connectivity between these cortical areas is considered to play an important role in consciousness (Leon-Carrion *et al*., 2012), in multisensory integration (Bergmann, 2008; Zamora-Lopez *et al*., 2011) and in the consolidation of newly acquired declarative memories and their integration for long-term storage (Buzsaki, 1989; Steriade & Timofeev, 2003). In addition, the increase of EEG coherence was observed in the beta band (13–30 Hz), which is supposed to be involved in sensorimotor integration (Kisley & Cornwell, 2006; Senkowski *et al*., 2006).

Furthermore, it is interesting to note that the increase of alpha power after EMDR sessions was localized in the left inferior temporal gyrus, which is believed to play an important role in different multisensory processes, such as faces and object perception (Grill-Spector, 2003; Aggelopoulos & Rolls, 2005). These cortices seem to be functionally altered in patients with PTSD (Shaw *et al*., 2002; Kroes *et al*., 2011). Therefore, the increase of alpha power observed in the present study could reflect the action of EMDR in fostering adaptive multisensory integration of dissociated aspects of traumatic event (Bergmann, 2008).

Our results are not consistent with previous findings from Propper *et al*. (2007) and Pagani *et al*. (2012). Propper *et al*. (2007) reported that the EMs reduce frontal interhemispheric gamma EEG coherence, and Pagani *et al*. (2012) observed a decrease in connectivity between left visual cortex and right fusiform gyrus in the theta band. The discrepancies between these results could be explained by differences in their study designs and methods. Propper *et al*. (2007) used a measure of coherence that depends mostly on the consistency of phase differences between electrodes (Nunez *et al*., 1997). Furthermore, these authors analysed phase coherence after 30 s of bilateral saccadic EMs recorded from only one pair of (prefrontal) electrodes; therefore, their experimental designs only allowed us to evaluate frontal interhemispheric coherence. Several methodological differences exist also between the present study and the one described by Pagani *et al*. (2012). For example, Pagani *et al*. (2012) used a different task (script listening) and adopted different approach to selects ROIs: initially the authors analysed the connection between 42 pairs of BAs, successively they clustered the BAs into wider ROIs until they reached statistical significance in the comparison.

The present study has several limitations. The most important limitation is the absence of control group (no treatment, waitlist or control treatment) to specifically evaluate the effect of EMDR. Second, the small sample size (we analysed only 13 EMDR sessions) is a problem in the generalization of our results. Finally, we use scalp EEG recordings, which have an intrinsic limit in space resolution, particularly in the identification of deep subcortical sources. The same kind of limitation, obviously, is reflected by the sLORETA software, which is by definition a low-resolution electric source analysis software.

Although our data are promising, they must be considered only as preliminary results. Future researches are needed to test if these modifications are selectively associated with EMs in EMDR, comparing EMDR sessions with and without EMs. Indeed, it must be underlined that both Propper *et al*. (2007) and Samara *et al*. (2011) studies investigated EEG coherence of EMs, outside the context of a clinical procedure utilizing EMDR, while the present research and the Pagani *et al*. (2012) examined the modification of EEG coherence after EMDR whole sessions, in which EMs are only one component of a more complex process. This leads to be very cautious when comparing results from studies with different aims and designs because there are no evidence that the neural underpinnings of EMs, when done within EMDR sessions, could be the same as when done outside of EMDR procedural methodology.

In conclusion, taken together, our findings seem to suggest that the complex action of EMDR leads to an integration of dissociated aspects of traumatic memories, as a reflection of functional connectivity, and consequently a decrease of hyperarousal symptoms.

Conflict of interest

The authors have no conflicts of interest.

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