CASE REPORT

Annals of Agricultural and Environmental Medicine 2017, Vol 24, No 2, 265–270
www.aaem.pl

Neuromarkers of Post-Traumatic Stress Disorder (PTSD) in a patient after bilateral hand amputation – ERP case study

Anna Chrapusta[1], Juri D. Kropotov[2,3], Maria Pąchalska[3,4]

[1] *Małopolska Burns and Plastic Surgery Centre, Rydygier Memorial Hospital, Krakow, Poland*
[2] *Institute of the Human Brain, Russian Academy of Sciences, St. Petersburg, Russia*
[3] *Chair of Neuropsychology, Andrzej Frycz-Modrzewski University, Kraków, Poland*
[4] *Center for Cognition and Communication, New York, NY, USA*

Chrapusta A, Kropotov JD, Pąchalska M. Neuromarkers of Post-Traumatic Stress Disorder (PTSD) in a patient after bilateral hand amputation – ERP case study. Ann Agric Environ Med. 2017; 24(2): 265–270. https://doi.org/10.26444/aaem/74597

Abstract

Introduction. There is a lack in the worldwide literature of reports on the Neuromarkers of Post-Traumatic Stress Disorder (PTSD) in patients after bilateral hand amputation The aim of this study was to test a hypothesis regarding developing Post-Traumatic Stress Disorder (PTSD) in a patient after bilateral hand amputation with the use of Event Related Potentials (ERPs). On the basis of previous research, the amplitudes of P3 ERP components elicited in the cued GO/NOGO tasks have been chosen as candidates for neuromarkers of PTSD.

Case study. A 24-year-old patient had undergone bilateral hand amputation 12 months previously. The patient was repeatedly operated on (he had undergone successful bilateral hand replantation) and despite the severity of the injuries, he recovered. However, the patient complained of flashbacks, anxiety and sleep difficulties. Specialist tests showed the presence of PTSD. The patient participated in the cued GO/NOGO task (Kropotov, 2009) with recording 19-channel EEG. P3 GO and NOGO waves in this task were found to be significantly smaller, in comparison to a group of healthy control subjects of the same age (N=23) taken from the HBI normative database (https://www.hbimed.com/). This observed pattern of ERP waves in the patient corresponds to the pattern found in PTSD patients.

Conclusions. ERPs in a GO/NOGO task can be used in the assessment of the functional brain changes induced by chronic PTSD.

Key words

replantation, anxiety, flashback

INTRODUCTION

There is a lack of reports in the worldwide literature on the subject of Neuromarkers of Post-Traumatic Stress Disorder (PTSD) in patients after bilateral hand amputation. The main reason is the fact that these are extremely rare cases, as well as often involving only the replantation surgery of the dominant hand. To-date, there has been only three accounts: two from Europe and one from Asia, of patients in whom the replantation of both hands was successfully carried out [1, 2, 3]. In addition to describing the case study, the organizational, surgical and technical difficulties involved in such an operation, the authors of the above-mentioned articles also deal with problems that arose: from the threat to life as a result of the danger of hypovolaemic shock, to the post-operational course of proceedings from the perspective of the varied degree of difficulty in regaining the gripping, manipulative and gnostic functions of the hands.

A review of the MEDLINE databases for the period 1992–2016 show the absence of any description of the impact of the amputation of both hands on the neuropsychological state of the patient, including any reference to Post-Traumatic Stress Disorder (PTSD). The time that elapsed from the accident itself through transportation to hospital, the patient's wait for confirmation of acceptance on the part of the replantation unit for hospitalisation, right up to awakening after the operation(s), all additionally increased the stress experienced. The several hours from injury to vascular linkage, a period perceived as relatively short by the surgeon, for the patient, however, is an incomparably longer period of stress and uncertainty, which is similar to the several or dozen or more days period post-operation, when there is still no guarantee of the success of the surgery itself. As a result of the above, it is essential to test the occurrence of PTSD in patients who have suffered such severe injuries. PTSD significantly impacts on the course of convalescence, rehabilitation and the return of functionality, as well as directly influencing the patient's quality of life.

The essence of PTSD is the uncontrolled return of traumatic recollections (flashbacks) [4, 5, 6, 7, 8]. The problem results in a disordering of the functioning of the brain and an absence of control possibilities for the disease, as well as the activation of those cognitive functions necessary during the rehabilitation process. In such a case, it is essential to implement new neurotechnologies in order to diagnose the symptoms of PTSD, and therefore implement effective treatment to restore full psychic readiness to combat the disease. For these reasons, the presented case study is innovative in its scope. The evaluation utilised quantitative EEGs (QEEG) and Event Related Potentials (ERPs) [9, 10].

Address for correspondence: Prof. Maria Pachalska, PhD, Chair of Neuropsychology, Andrzej Frycz Modrzewski University, Andrzeja Herlinga-Grudzińskiego 1, 30-750 Krakow, Poland
e-mail: neuropsychologia23@o2.pl

Received: 22 November 2016; accepted: 27 December 2016; *first published on December, 2016*

CASE STUDY

A 24-year-old patient had both hands amputated at the wrists. He was taken by ambulance from the scene of the accident, about 400 km to Kraków, and because of poor weather the journey took about 6 hours. At the scene of the accident, the hands had been secured and protected by the rescue team, in accordance with replantation procedural practice and regulations (Fig. 1A). He was admitted to the Małopolska Burns and Plastic Surgery Centre, at The Ludwig Rydygier Memorial Hospital in Kraków as part of the national replantation emergency service scheme. Thanks to the most effective coordination and communication between the ambulance team, Helicopter-based emergency medical service (EMS) and the centre itself, the patient was admitted and immediately the decision was made for the replantation of both hands. Almost without delay, he was admitted to the operating theatre where the operation was performed by an experienced surgeon and his team, assisted by a 3-person 'young team'.

The surgical procedure began with the identification of the structures in the amputated hands, and Kirchner wires inserted into both appendages as a preparation to fixation. Simultaneously, the anaesthetic team prepared the patient for imminent many-hour operation. The next step, started after introduction of the general anaesthetic, was revision of the stumps and identification of structures: the vascular structures were marked, the nerves and tendons designated for reconstruction. The replantation began with bone stabilisation by using the previously inserted Kirschner wires. Thee replantation began with the replatation of the dominant right hand, in accordance with the generally accepted procedures. At the same time, the second team started reconstruction of the joint capsules and the tendons. A difficulty in the replantation of the right hand was the result of damage to the wrist's proximal row, as well as the distal cut of the tendons of the fingers flexors. This was caused by the flexion of the fingers in the right hand at the moment of amputation. This state resulted in the necessity to open wide the metacarpal together with the carpal tunnel. During the course of the accident, the fingers of the left hand were in extension, and the tendon ends were in line with the cut, which aided the replantation.

The replantation was conducted in accordance with the MCOP procedures in force at the replantation centre. After completion of the arterial and vein anastomosis, The two surgical teams changed places. While this was taking place, the 'young team' continued with suturing the remaining tissues of the right hand. The patient was given 8 units of blood concentrate during the course of the operation, which lasted 10 hours.

After the procedure, the blood-flow and temperature of the fingers in both hands was normal. Typical treatment improving the blood circulation and angiogenesis was applied (Heparyna, Dextran 40.000j, Polfilin) in a constant infusion during 5 days. Treatment in a hyperbaric chamber was carried out. During the course of treatment, the authors' own modifications to the splinting of the hands was applied, which enabled the passive rehabilitation of the fingers, as well as simultaneous stabilization of the fingers in a functional position with the aim of avoiding contracture (Fig. 1B). All these passive rehabilitation activities had to be conducted by another person. The patient's mother was also trained to carry out such exercises, as well as to help the patient in conducting other everyday activities.

On the 10th day after the operation, the patient was discharged from hospital in a good local and general condition. Both hands had the correct blood-flow, had the correct temperature and vascular peristalsis. The wounds were healing extremely well. The stitches were removed on the 16th day after the operation.

The stabilising Kircher wires were removed after 4 weeks and there was immediately commenced a cycle of passive and active rehabilitation (physiotherapy exercises and following compression in a hyperbaric chamber) conducted 5 days a week. The rehabilitation lasted from 2 – 5 hours daily.

Neuropsychological examination. 12 months after the operation, a neuropsychological diagnosis of the brain was conducted. A structured interview was used, directed toward the diagnosis of PTSD, which was ascertained during a comprehensive clinical diagnosis in accordance with the DSM 5 criteria (Tab. 1).

(A)

(B)

Figure 1 (A). Patient's hands severed by a metal-cutting guillotine. (B) State of patient's hands a week after replantation: stabilizing forearm splints were used (as modified by the first author of the article)

Table 1. Criteria (categories) and DSM 5. [Diagnostic criteria 309.] (F43.10).

Code	Criteria (categories) and symptoms of PTSD according to DSM-5	Description of the symptoms of PTSD in the given patient
A	**Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:**	
A1	Directly experiencing the traumatic event(s).	Exposure to threatened death.
A2	**Witnessing, in person, the event(s) as it occurred to others.**	None.
A3	Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.	None.
A4	Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains: police officers repeatedly exposed to details of child abuse).	None
B	**Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:**	
B1	Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).	Recurrent daily presence of involuntary, and intrusive distressing memories
B2	Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).	Recurrent (almost every night) distressing dreams in which the content and sometimes the effect of the dream are related to the traumatic events.
B3	Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.	Flashbacks in which the patient feels as if the traumatic event(s) were recurring. Sometimes, the patient looses awareness of present surroundings.
B4	Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).	Prolonged psychological distress at exposure to external cues that symbolize or resemble an aspect of the traumatic event.
B5	Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).	None
C	**Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:**	
C1	Avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).	Avoidance of distressing memories about the traumatic event.
C2	Avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).	Avoidance of external reminders (conversations and objects) that arouse distressing memories about the traumatic event.
D	**Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:**	
D1	**Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors, such as head injury, alcohol, or drugs).**	Inability to remember an important aspect of the traumatic event (due to dissociative amnesia).
D2	Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., 'I am bad', 'No one can be trusted', 'the world is completely dangerous', 'my whole nervous system is permanently ruined').	Persistent and exaggerated negative beliefs or expectations about oneself, and the world: often claiming that: 'One cannot do anything'.
D3	Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself, or others.	Persistent, distorted cognitions about the consequences of the traumatic event that lead the individual to blame himself.
D4	Persistent negative emotional state (e.g.fear, horror, anger, guilt, or shame).	Persistent negative emotional state (anger, and less frequently fear).
D5	Markedly diminished interest or participation in significant activities.	The patient does not participate in significant activities at all.
D6	Feelings of detachment or estrangement from others.	Feelings of detachment from others.
D7	Persistent inability to experience positive emotions (e.g. inability to experience happiness, satisfaction, or loving feelings).	Persistent inability to experience happiness, satisfaction, or love to his closest family.
E	**Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:**	
E1	Irritable behaviour and angry outbursts (with little or no provocation), typically expressed as verbal or physical aggression towards people or objects.	Irritable and angry outbursts (with no provocation), typically expressed as verbal aggression toward people.
E2	Reckless or self-destructive behaviour.	None
E3	Hypervigilance.	Hypervigilance, mainly during the day.
E4	Exaggerated startle response.	None
E5	Problems with concentration.	The patient is not able to concentrate on a topic of conversation or during a long task.
E6	Sleep disturbance (e.g. difficulty in falling asleep, staying asleep, or restless sleep).	Sleep disturbance (difficulty falling asleep and restless sleep).

Table 1. Criteria (categories) and DSM 5. [Diagnostic criteria 309.] (F43.10) (Continuation)

Code	Criteria (categories) and symptoms of PTSD according to DSM-5	Description of the symptoms of PTSD in the given patient
F	Duration of the disturbance (criteria B, C, D, and E) is more than 1 month.	Duration of the disturbance (Criteria B, C, D, and E) is 12 months.
G	The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.	The disturbance causes clinically significant distress and impairment in social areas of functioning.
H	The disturbance is not attributable to the physiological effects of a substance (e.g. medication, alcohol), or another medical condition.	The disturbance is not attributable to the physiological effects of medication or alcohol).
With dissociative symptoms:	Depersonalization: persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g. feeling as though one were in a dream; feeling a sense of unreality of one's self or body or of time moving slowly).	None
	Derealization: persistent or recurrent experiences of unreality of one's surroundings (e.g. the world around the individual is experienced as unreal, dreamlike, distant, or distorted).	The patient, from time to time, feels recurrent experiences of unreality of surroundings; the world around is experienced as unreal and distant.

ERP CASE STUDY

Cued GO/NOGO task. To test brain correlates of cognitive control a specific variant of the cued GO/NOGO task was used [11, 12]. In this task, images of animal (*a*) and plant (*p*) categories served as relevant stimuli. The trials consisted of the presentations of paired stimuli *s1-s2* with inter-stimulus intervals of 1,000 ms and inter-trial intervals of 3,000 ms. Four categories of trials were used: *a-a, a-p, p-p and p-h+novel sound,* where *h* is an image of a human. The duration of stimuli was 100 ms. The subject's task was to respond by pressing a button with the right hand to a-a trials (GO trials), and to withhold from responding in a-p trials (NOGO trials) [10].

The pictures were selected from textbooks for children in such a way that the overall luminance and the image sizes of animals and plants were approximately equal. To avoid habituation to repeating stimuli, 20 different images of animals, plants and humans were randomly presented in various combinations. To maintain a certain level of alertness, novel sounds were occasionally presented simultaneously with the images of a human in ignore trials. They produced an orientation reaction, confirmed by the elicitation of the P3 novelty ERP wave.

The trials were grouped into 4 blocks with 100 trials each. In each block, a unique set of 5 *a*, 5 *p*, and 5 *h* stimuli were selected. Each block consisted of a pseudo-random presentation (requiring equal number of trials in 4 categories) of 400 trials, with 100 trials within each trial category. The patient practiced the task before the recording started. Subjects rested for a few minutes after each 200 trials. The patient sat upright in a comfortable chair, looking at a computer screen. Stimuli were presented on 17-inch CRT computer screens which were positioned 1.5 meters in front of the subjects, and occupied 3.8° of the visual field.

Data recording. The patient responses were recorded in a separate channel. An average for response latency as well as its standard deviation were calculated. Omission errors (failure to respond in GO trials) and commission errors (failure to suppress a response to NOGO trials) were also computed. EEG was recorded from 19 scalp sites according to the 10–20-system, bandpass-filtered between 0.3–50 Hz, and digitized at a rate of 250 samples per second per channel.

The EEG was recorded referentially to linked ears, allowing computational re-referencing of the data (re-montaging). The EEG computationally was re-referenced to the common average montage. EEG was recorded with a 19-channel electroencephalographic PC-controlled system, the 'Mitsar-201' (CE 0537) (Mitsar Co., Ltd., St. Peterberg, Russia), and electrodes applied using caps (Electro-Cap International, Inc., Eaton, OH, USA). The tin recessed electrodes contacted the scalp using ECI ELECTRO-GEL. Quantitative data were obtained using WinEEG software [11, 13].

Artifact correction. Eye blink artifacts were corrected by zeroing the activation curves of individual independent components corresponding to eye blinks. These components were obtained by application of Independent Component Analysis (ICA) to the raw EEG fragments, as described by Jung et al. [14]. Comparison of the method applied in the current study with an EOG regression technique is described in Tereshchenko et al. [15]. In addition, epochs with excessive amplitude of filtered EEG and/or excessive faster and/or slower frequency activity were automatically marked and excluded from further analysis. The exclusion thresholds were set as follow: 1) 100 μV for non-filtered EEG; 2) 50 μV for slow waves in 0–1 Hz band; and 3) 35 μV for fast waves filtered in the 20–35 Hz band.

RESULTS

Behavioural data. The parameters of the patient's performance in the cued GO/NOGO task, compared with the normative data, are presented in Table 1. The normative data were obtained from a group of healthy subjects (N=23) selected from the HBI (Human Brain Index) database (see description of the database in Kropotov [10, 13]. There was a statistically significant ($p<0.01$) increase of reaction time, while the other parameters did not deviate from the normative data.

Table 1. Behavioural data from task carried out under sight control

Data	Omission errors	Commission errors	Reaction time (RT)	Standard deviation of RT
Patient	4	0	559	119
Norm	2.5	0.7	377	78
p-value	0.63	0.52	0.01	0.08

ERPs. Figure 2 depicts ERPs in the cued GO/NOGO task in the patient, compared with to the grand averaged ERP for the group (N=23) of healthy controls (HC). It can be seen that P3 GO and P3 NOGO waves in the patient are significantly smaller than those in the group of healthy subjects of corresponding age. Simultaneously, the maps of the P3 waves show the P3 NOGO anterization phenomenon similar to that found in healthy controls.

DISCUSSION

Post-Traumatic Stress Disorder (PTSD) as an anxiety disorder following a traumatic event is characterized by 3 symptom clusters, such flashbacks and nightmares, emotional numbing, and hyperarousal symptoms (DSM-5). From the neuropsychological perspective, patients with PTSD show dysfunction of cognitive control [16, 17, 18], which is reflected in the decrease of P300-like ERP components in PTSD patients (for review, see: Johnson et al., 2013 [19]). The cued GO/NOGO task is designed specifically to measure the P300-like components of cognitive control [10, 11, 12]. In the presented study, the hypothesis was tested whether the P300 components of cognitive control in the cued GO/NOGO task are impaired in the patient after bilateral hand amputation.

The patient performed the cued GO/NOGO task while 19-channel EEG was recorded. The reaction time was shown to be statistically longer in the patient, compared to a group healthy subjects. However, omission and commission errors were similar to those found in the healthy control, indicating no behavioural correlates of impaired cognitive control. A different pattern was found in the ERP measures extracted from the EEG. In particular, the P300 GO and NOGO were found to be significantly smaller at p<0.01 than those in the group of healthy controls. This finding supports the initial hypothesis of PTSD in the patient.

The presented case study of a patient who had undergone a successful replantation of both hands is unique in nature in terms of the multi-discipline treatment employed. The application of unique replantation procedures, splinting, movement rehabilitation and ERP measures of cognitive control was applied in a pioneering fashion in the world. The patient, as a result of this programme of surgical treatment and comprehensive rehabilitation, after only three months of the replantation, was able too partially regain the ability

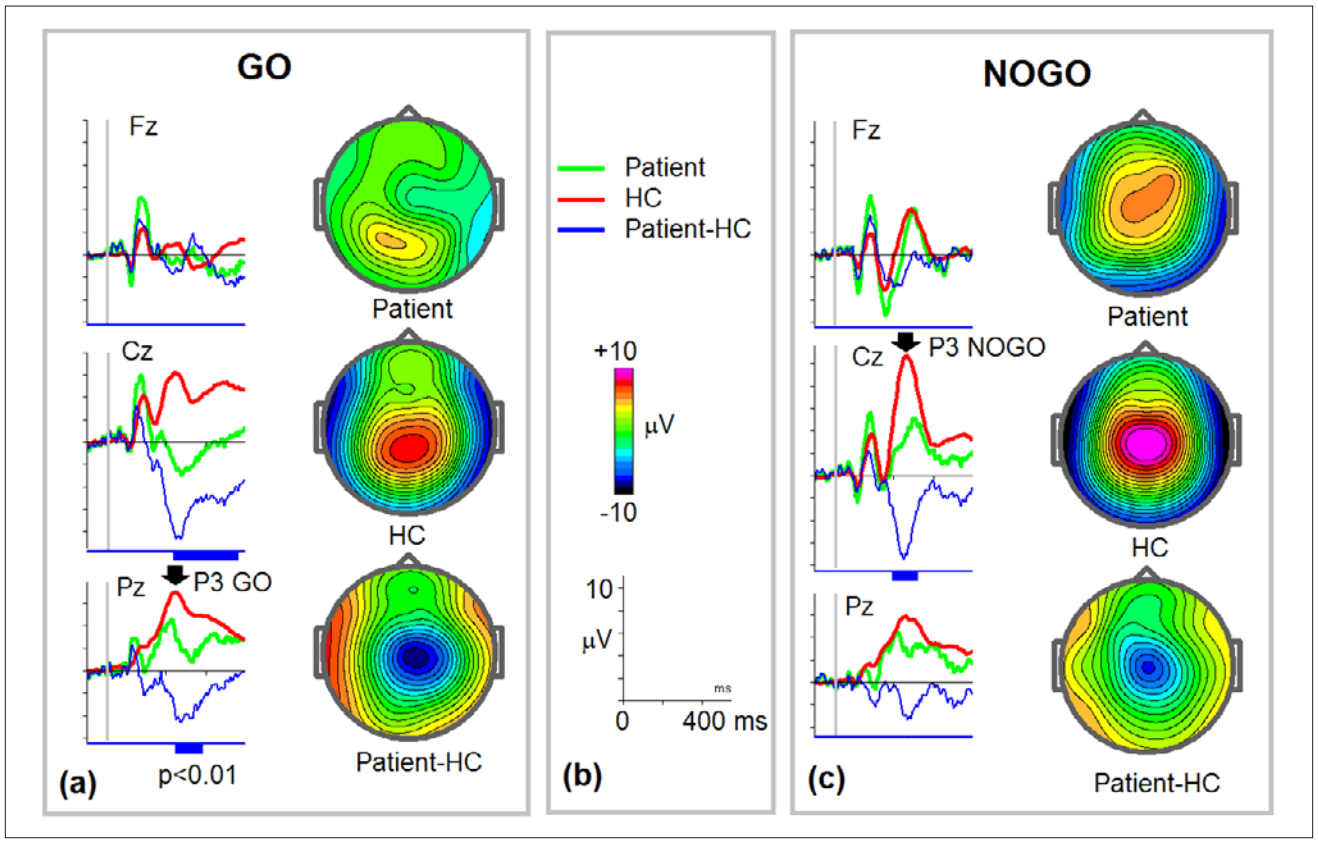

Figure 2. ERPs in cued GO/NOGO task in the patient, compared to the grand averaged ERP for the group (N=23) of healthy controls.
(a) Left: ERPs recorded at Fz, Cz and Pz for GO trials for the patient (green lines), the group of healthy controls (HC) (red lines) and the ERP differences (patient- HC) (blue lines), with marks of statistical significance (p<0.01) of the difference below the curves. Right: maps of the ERP and ERP differences at the latency corresponding to the peak of P3 GO for HC taken at Pz.
(b)Scales and line indicators.
(c) Left: ERPs recorded at Fz, Cz and Pz for NOGO trials for the patient (green lines), the group of healthy controls (HC) (red lines) and the ERP differences (patient- HC) (blue lines), with marks of statistical significance (p<0.01) of the difference below the curves. Right: maps of the ERP and ERP differences at latency, corresponding with the peak of P3 NOGO for HC taken at Cz.

to grip, manipulate, and regained even the gnostic functions of both hands.

The study shows that the ERP measures in the cued GO/NOGO task could serve as a reliable functional neuromarkers for the detection of cognitive control dysfunction [20, 21, 22, 23] which is disturbed in PTSD.

CONCLUSION

The ERPs in a GO/NOGO task can be used in the assessment of the functional brain changes induced by chronic PTSD.

Acknowledgement
The study was funded from private sources.

REFERENCES

1. Nayak BB, Mohanty N, Patnaik AP, et al. Considerations for double-hand replantation in a resource-constrained healthcare facility. Indian J Plast Surg. 2016 Jan-Apr; 49(1): 81–85. doi: 10.4103/0970–0358.182233 [Pub-Med].
2. deBruijn HP, Dhar BK, Robinson PH:A bilateral hand replantation, Ned Tijdschr Geneeskd. 1990 Jan 27; 134(4): 177–9. [Pub-Med].
3. Marques M, Correia-Sá I, Festas MJ, Silva S, Silva Al, Silva A, Amarante J. Six years of follow-up after bilateral hand replantation. Chir Main. 2013 Sep; 32(4): 226–34. doi: 10.1016/j.main.2013.06.005 [Pub-Med].
4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, DSM-V Washington: American Psychiatric Publishing Group. 2013.
5. Brewin CR. Encoding and Retrieval of Traumatic Memories. In J.J. Vasterling & C.R. Brewin (Eds.) Neuropsychology of PTSD, Biological, Cognitive, and Clinical Perspectives (pp. 131- 152). New York: The Guilford Press Brewin. 2005.
6. Brevin CR, Gregory JD, Lipton M. & Burgess N. Intrusive images in psychological disorders: characteristics, neural mechanisms, and treatment implications. Psychol Rev. 2010; 117(1): 210–232. doi: 10.1037/a0018113 [PubMed]
7. Bryant RA, O'Donnell ML, Creamer M, McFarlane A.C, Silove D. Posttraumatic intrusive symptoms across psychiatric disorders. J Psychiatr Res. 2011; 45: 842–847. doi: 10.1016/j.jpsychires.2010.11.012 [PubMed]
8. Danker JF, Anderson JR. The ghosts of brain states past: remembering reactivates the brain Regions Engaged During Encoding. Psychol Bull. 2010;136(1): 87–102. doi: 10.1037/a0017937 [PubMed]
9. Trystuła M, Zielińska J, Półrola P, Góral-Półrola J, Kropotov JD, Pąchalska M. Neuromarkers of anxiety in a patient with suspected schizophrenia and TIA: the effect of individually-tailored neurofeedback. Acta Neuropsychol. 2015; 13(4): 395–403. doi: 10.5604/17307503.1103831.
10. Kropotov JD. Functional neuromarkers for psychiatry. Application for diagnosis and treatment. Amsterdam, London, Paris, Sydney, Tokyo: Elsevier Academic Press. 2016.
11. Kropotov JD, Ponomarev VA. Decomposing N2 NOGO wave of event-related potentials into independent components. Neuroreport. 2009; 20(18): 1592–1596.
12. Kropotov JD, Ponomarev V A, Hollup S, Mueller A. Dissociating action inhibition, conflict monitoring and sensory mismatch into independent components of event related potentials in GO/NOGO task. NeuroImage. 2011; 57, 565–575.
13. Kropotov JD. Quantitative EEG, event-related potentials and neurotherapy. Amsterdam, London: Elsevier Academic Press. 2009.
14. Vigário RN. Extraction of ocular artefacts from EEG using independent component analysis. Electroencephalogr Clin Neurophysiol. 1997; 103(3): 395–404.
15. Tereshchenko EP, Ponomarev VA, Kropotov I, Muller A. Comparative efficiencies of different methods for removing blink artifacts in analyzing quantitative electroencephalogram and event-related potentials]. Fiziol Cheloveka. 2009; 35(2): 124–131.
16. Vasterling JJ, Brailey K, Constans JI, Sutker PB. Attention and memory dysfunction in posttraumatic stress disorder. Neuropsychology. 1998; 12(1): 125–133.
17. Koso M, Hansen S. Executive function and memory in posttraumatic stress disorder: a study of Bosnian war veterans. Eur Psychiatry. 2006; 21(3): 167–173. doi.org/10.1016/j.eurpsy.2005.06.004 [PubMed]
18. Leskin LP, White PM. Attentional networks reveal executive function deficits in posttraumatic stress disorder. Neuropsychology. 2007; 21(3): 275–284. doi: 10.1037/0894-4105.21.3.275 [PubMed]
19. Johnson JD(1), Allana TN, Medlin MD, Harris EW, Karl A. Meta-analytic review of P3 components in posttraumatic stress disorder and their clinical utility. Clin EEG Neurosci. 2013; 44(2): 112–34. doi: 10.1177/1550059411428716 [PubMed]
20. Dębiec J, LeDoux JE. The amygdala and the neural pathways of fear. In: Shiromani PJ, Keane TM & LeDoux JE (Eds) Post-Traumatic Stress Disorder: Basic Science and Clinical Practice (pp. 23–38). New York: Humana Press. 2009.
21. Pąchalska M, Grochmal-Bach B, MacQueen BD, Frańczuk B. Post-traumatic stress disorder in Polish stroke patients who survived Nazi concentration camps. Med Sci Monit. 2006; 12(4): CR137–149[PubMed]
22. Dolcos F, LaBar KS, Cabeza R. Remembering one year later: Role of the amygdala and the medial temporal lobe memory system in retrieving emotional memories. PNAS 2005; 102(7): 2626–2631. doi: 10.1073/pnas.0409848102 [PubMed]
23. Hellawell SJ, Brewin CR. A comparison of flashbacks and ordinary autobiographical memories of trauma: cognitive resources and behavioural observations. Behav Res Ther. 2002; 40(10): 1143–1156. [PubMed]
24. St Jacques PL, Botzung A, Miles A. Rubin DC. Functional neuroimaging of emotionally intense autobiographical memories in posttraumatic stress disorder. J Psychiatr Res. 2010; 45(5): 630–637 [PubMed]