CASE REPORT

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Neurofeedback training for peak performance

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Abstract

Aim. One of the applications of the Neurofeedback methodology is peak performance in sport. The protocols of the neurofeedback are usually based on an assessment of the spectral parameters of spontaneous EEG in resting state conditions. The aim of the paper was to study whether the intensive neurofeedback training of a well-functioning Olympic athlete who has lost his performance confidence after injury in sport, could change the brain functioning reflected in changes in spontaneous EEG and event related potentials (ERPs).

Case study. The case is presented of an Olympic athlete who has lost his performance confidence after injury in sport. He wanted to resume his activities by means of neurofeedback training. His QEEG/ERP parameters were assessed before and after 4 intensive sessions of neurotherapy. Dramatic and statistically significant changes that could not be explained by error measurement were observed in the patient.

Conclusion. Neurofeedback training in the subject under study increased the amplitude of the monitoring component of ERPs generated in the anterior cingulate cortex, accompanied by an increase in beta activity over the medial prefrontal cortex. Taking these changes together, it can be concluded that that even a few sessions of neurofeedback in a high performance brain can significantly activate the prefrontal cortical areas associated with increasing confidence in sport performance.

Key worda
neurofeedback, cognitive control, anxiety, ERPs

INTRODUCTION

One of the applications of the neurofeedback methodology is peak performance in sport. Neurofeedback (EEG biofeedback) holds potential for retraining brainwave activity to enhance optimal performance in athletes in various sports [1]. Neurofeedback has been shown to have the potential for quieting the mind to improve performance in archery, for example. It can also be used to improve concentration and focus, cognitive function and emotional control following concussions and mild head injuries, and it has untapped potential to increase physical balance in gymnastics, ice skating, skiing, and other areas of performance [2, 3, 4, 5].

Clinical examples are provided on the use of neurofeedback to improve physical balance, while controlled research is called for [2, 3]. The protocols of the neurofeedback are usually based on an assessment of the spectral parameters of spontaneous EEG in resting state conditions. The case is presented of a sportsman who had lost performance confidence and wanted to resume his activities by means of neurofeedback training [6].

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OBJECTIVE

The aim of the paper was to study whether the intensive neurofeedback training of a well functioning sportsmen who has lost his performance confidence after injury in sport, could change the brain functioning reflected in changes in the spontaneous EEG and event related potentials (ERPs).

CASE STUDY

The case study is presented of an Olympic athlete, 25 years of age, a member of the Polish javelin team at the 2012 Olympic Games in London. The patient had achieved a personal best of 84.99 m, which would have been sufficient for a gold medal at the London Olympics. Following obtaining this personal best, he was subjected to strong psychological pressure from the media and sporting circles resulting from medal expectations, something that was to cause significant stress for the Polish and international sportsman.

During the period of direct preparation for the Olympics, he suffered an injury to the ankle joint and damage to the Achilles tendon. However, despite severe pain, the sportsman continued his preparations for the Olympics, using only permitted anaesthetics, as well as taking part in physiotherapy treatment. A standard treatment programme for this type of case was applied, with the aim of immobilization tapping was also used. The sportsman attended the Olympics where, unfortunately, he achieved only 22nd place, which he explained both on the basis of the injury as well as the pressure exerted on him from his immediate sporting circles, which resulted

in a reduction in his confidence and belief in being able to finally achieve victory.

Following his return to Poland, the chronic pain at the end of August 2012 intensified, a pain that appeared not only during intensive exertion, but increasingly so during warm-ups, walking, and even when at rest. He underwent arthroscopy and was clinically diagnosed as having 'posterior ankle impingement syndrome'. This syndrome, also known as *os trigonum* syndrome and posterior tibiotalar compression syndrome, is a clinical disorder characterized by acute or chronic posterior ankle pain triggered by forced plantar flexion, which causes chronic repetitive microtrauma [7].

The results of standard psychological and neuropsychological tests confirmed lost of cognitive control, as well as the appearance of emotional disturbances. He decided to resume his activities by means of neurofeedback training.

Peak performance training with neurofeedback. The Olympic athlete took part in 4 peak performance training sessions with neurofeedback at the beginning of September 2012. HRV biofeedback training was conducted for a period of 10 minute, as well as EEG feedback (neurofeedback) for 20 minutes on a bipolar montage with electrodes at points C3 – C4 on the 8 canal PROCOM Infiniti BIOMED Neurotechnology apparatus. The training sessions were conducted by the psychologist Robert Kozłowski at the National Research-Implementation Centre for Sport Psychology at the University of Physical Education and Sport in Gdańsk, Poland. The training protocol was developed on the basis of results obtained by means of the QEEG/ERPs method. Electrodes were placed in accordance with the international system for the localisation of electrodes 10–20. The patient was prepared for tests in a standard manner, keeping the impedance of the electrodes below 5 kilo Ohm. The frequency 9–13 Hz was amplified during training.

The patient was placed in a NEEDO company chair with a footrest ensuring a comfortable body position with particular attention being placed on the foot under treatment. The head was placed on the headrest, while the arms were comfortable placed on the armrests of the chair. The monitor displaying the stimuli was located out of sight on a separate small table. The implementation of such a model of intensive Peak performance training with neurofeedback was the result of the sportsman's request for rapid help for the difficult psychological situation in which he found himself following unsatisfactory results in the competition, as well as being conditioned by the absence of a strategic goal-directed programme within the process of neuromodulation, and a repeated reintegration of cognitive control for competitors at the very highest sporting levels. During the course of the tests and training sessions, the patient took medication [framin 5000, ciprinol 500, rantudil forte, cyclo3Fort], which did not have an effect on the monitoring abilities of the frontal lobes [5, 8, 9, 10].

Permission to conduct the experiment was obtained from both the Olympic athlete himself and the Bioethics Commission.

MATERIALS AND METHOD

The following methods were used to ascertain the Olympic athlete's state of health:

1. Analysis of the patient's relevant documentation (illness case history, test results, including the results of arthroscopy)
2. A clinical interview, during which emphasis was placed particularly on psychic experiences in connection with MEDIA pressure and patient expectations, as well as the means of coping with the limitations resulting from the threat of illness connected with dysfunction of the ankle joint.
3. QEEG/ERPs directed for evaluation of performance in GO/NOGO task.

Neuropsychological testing. Neuropsychological testing at baseline (Exam 1) showed mild multiple deficits (Tab. 1). At follow-up, after conclusion of the neurotherapy programme (Exam 2), the Olympic athlete showed improvements in neuropsychological functioning. His cognitive and executive functions increased significantly and reached norm. This general pattern was repeated in nearly all the neuropsychological parameters (Tab. 1).

Table 1. Neuropsychological testing of the Olympic athelets

Measure	Exam. 1	Exam. 2
Attention		
WMS-III Spatial Span	12 (75th%ile)	100th percentile
Visuospatial Ability		
WAIS-III Block Design	3 (1st%ile)	100th percentile
Verbal memory		
CVLT Short Delay Free Recall	2/9 (<1st%ile)	100th percentile
CVLT Long Free Recall	2/9 (<1st%ile)	100th percentile
CVLT Long Delay Cue Recall	2/9 (<1st%ile)	100th percentile
Executive Functions		
TMT– Number Sequencing	54s. (10th%ile)	100th percentile
TMT– Number Letter Sequencing	150s. (<1st%ile)	100th percentile
Stroop		
Colour	41 s. (16th%ile)	100th percentile
Word	42 s. (63rd%ile)	100th percentile
Interferences	128 s. (<1th%ile)	100th percentile
WCST		
Categories	2 (>16th%ile)	100th percentile
Perseverative Errors	19 (37th percentile)	100th percentile
Conceptual Level Responses	48 (45th%ile)	100th percentile
Fail to Maintain Sets	4 (2–5th%ile)	100th percentile

Neurophysiological testing – EEG recording. The electroencephalogram (EEG) was recorded with the Mitsar 21-channel EEG system, manufactured by Mitsar, Ltd. (http://www.mitsarmedical. com), with a 19-channel electrode cap with tin electrodes that included Fz, Cz, Pz, Fp1/2, F3/4, F7/8, T3/4, T5/6, C3/4, P3/4, O1/2. The cap (Electro-cap) was placed on the scalp according to the standard 10–20 system. Electrodes were referenced to linked earlobes (off-line) and the input signals sampled at a rate of 250 Hz (bandpass 0.5–30 Hz). The ground electrode was placed on the forehead. Impedance was kept below 5 kΩ. The participant sat upright in a comfortable chair, looking at a computer screen (17 inch screen), 1.5 meter in front of him. All recordings were performer by the author of this article. ERP waveforms were

averaged and computed off-line and trials with omission and commission errors were automatically excluded.

Behavioural task. The task consisted of 400 trials sequentially presented to the subject every 3 seconds. Three categories of visual stimuli were used:
1) 20 different images of animals – referred to later as ***A;***
2) 20 different images of plants – ***P;***
3) 20 different images of people of different professions (presented together with an artificial 'novel' sound) referred to as ***H***.

The trials consisted of presentations of pairs of stimuli with inter-stimulus intervals of 1 s. Duration of stimuli presentation was 100 ms. Four categories of trials were used: ***A-A***, ***A-P***, ***P-P***, and ***P-H*** (Fig. 1). In the trails with A-A and P-P pairs, the first and the second stimuli were identical (physically the same). The trials were grouped into 4 sessions with 100 trials in each. In each session, a unique set of 5 ***A*** stimuli, 5 ***P*** and 5 ***H*** stimuli was selected. Each session consisted of a pseudo-random presentation of 100 pairs of stimuli, with equal probability for each category and each trial category.

The task was to press a button with the right hand for all **A-A** pairs as fast as possible, and to withhold from pressing in response to other pairs. The participant performed 10 trials without recording to see if they understood the instruction. He rested for a few minutes after completing 100 trials. Stimuli occupied about 3.8° of the visual field around the centre of the screen. Visual stimuli (were selected to have) had similar 2D sizes and luminosities.

Artifact correction procedures. 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 in [9,10]. 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 follows:
1) 100 μV for non-filtered EEG;
2) 50 μV for slow waves in 0–1 Hz band;
3) 35 μV for fast waves filtered in the band 20–35 Hz.

In addition, the recordings were visually inspected and excluded remaining artifacts.

EEG spectra. EEG spectra were computed for Eyes Open, Eyes Closed, and the GO/NOGO task conditions separately. The artifact free fragments of EEG were divided into 4 sec episodes with 50% overlap. The Hanning time window was used [2]. EEG spectra were computed for each episode and averaged. Mean value and standard deviations for each 0.25 Hz bin were computed. For comparison of EEG spectra pre- and post-intervention, the t-test was used.

Decomposition of collection of ERPs into independent components. To obtain valid independent components, the number of training points is essential (Onton and Makeig 2006). In this study, ERP's from 215 healthy subjects recorded under the HBIdb project were used [11].

ICA was performed on the full 'ERP scalp location' x 'Time series' matrix P. ERPs were constructed in response to the second (S2) stimuli in the time interval of 700 ms after the second (S2) stimulus presentation for GO and NOGO cues. Assumptions that underlie the application of ICA to individual ERPs are as follows:
1) summation of the electric currents induced by separate generators is linear at the scalp electrodes;
2) spatial distribution of components' generators remains fixed across time [12, 13].

The ICA method was implemented in the analysis software described in [14]. The topographies of the independent components are presented as topographic maps, while time courses of the components (also called 'activation time courses') are presented as graphics with time corresponding to the *X*-axis.

Spatial filters were obtained and applied to individual ERPs in order to estimate the corresponding components in a single individual [15]. The ERP independent components of the subject who participated in the presented study were compared with the grand average ERPs of the healthy controls aged 24–25 (N= 46). The ERP independent components of the subject were also compared between pre and post-intervention conditions.

RESULTS

Behavioural data. The behavioural data, such as omission and commission errors, reaction time and variance of the reaction time, are presented in Table 2. When the parameters of the first recording were compared with the averaged parameters of the healthy control group of the corresponding age, no statistically significant at $p<0.05$ deviations from the norms were found. It should be stressed, however, that the subject is 100 ms faster than the average norm, which is almost twice more consistent in response than the average. However in the second recording, the subject performed so consistently that the variance of reaction time became statistically ($p<0.05$) smaller than the average norm.

Table 2. Parameters of the subject's performance in the cued GO/NOGO task in the first and second recording, compared with the averaged data of the healthy controls group

	Omission errors	Commission errors	Reaction time (RT)	Variance of RT in ms
1 recording	0	0	273	39
2 recording	0	0	276	25
Healthy controls	4.4.%	0.6%	378	83
p-value of the difference from the normal average	0.58	0.54	0.22	0.21

Spectra. In the first recording, no statistically significant deviations from the reference were found in EEG spectra for Eyes Open, Eyes Closed, and GO/NOGO task conditions. In the second recording, compared with the first recording, a statistically significant increase in high beta activity was found in central-frontal locations (Fig. 1A). The decomposition of the background EEG into independent components revealed 3 independent components associated with this beta activity. The topographies and sLORETA images of these components are presented in Figure 1 B, C.

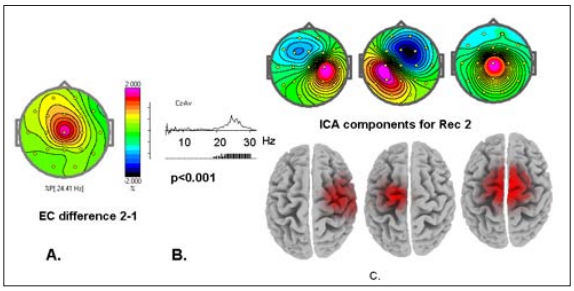

Figure 1. Relative EEG spectra differences between the first and second recordings
A. Map of spectra difference (2 rec – 1 rec) at 25 Hz.
B. Relative spectra difference (2 rec – 1 re). Below the curve p-values of the spectra difference. Large vertical bars – p<0.001, small vertical bars – p<0.05).
C. Maps and sLORETA images of independent components associated with increase in beta activity.

Event-related potentials. The largest changes in ERPs induced by the intervention were observed for the NOGO condition. Fig. 2A depicts ERPs computed for NOGO condition in the first (red line) and the second (green line) conditions. At the bottom, topographies at the peak amplitude at the first and second recordings are presented. Fig 2B depicts the two P3 NOGO independent components into which the P3 NOGO is decomposed. They are: 1) early P3 NOGO component, and 2) the late P3 NOGO components. Time courses and topographies of the components are presented at the bottom. As can be seen, only the P3 NOGO late component changes after intervention.

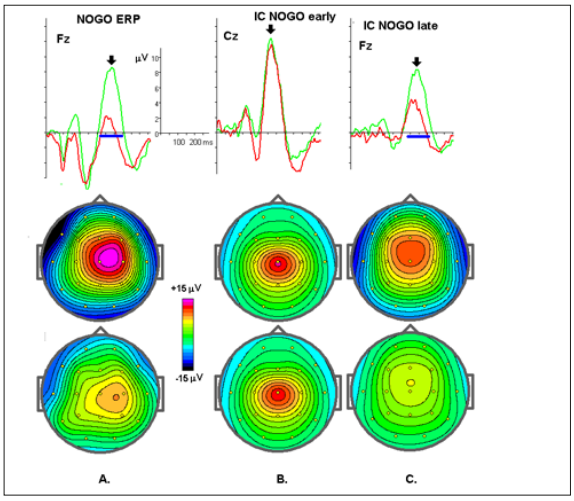

Figure 2. ERP changes induced by intervention

Raw ERP data for the NOGO condition in the first (red line) and the second (green line) conditions. Below are topographies computed at the peaks of the NOGO P3 waves (indicated by an arrow). The horizontal blue line indicates the time interval with significant pre-post changes at p<0.01.

Independent component P3 NOGO early. Above – activation time courses for the first and second recordings. The horizontal blue line indicates the time interval with significant pre-post changes at p<0.01. Below – topographies at the peaks (indicated by an arrow).

Independent component P3 NOGO late. Above – activation time courses for the first and second recordings. The horizontal blue line indicates the time interval with significant pre-post changes at p<0.01. Below – topographies at the peaks (indicated by an arrow).

DISCUSSION

An Olympic athlete took part in 4 peak performance training sessions with neurofeedback. The training protocol was developed on the basis of results obtained by means of the QEEG/ERPs method.

Spectra changes after relative beta training. The results of the presented study show that even short-term but intensive training sessions in the peak performing subject changed the beta activity over the trained electrodes. This beta activity was decomposed into 3 independent components localized in the somato-sensory strip. Taking into account the positive relationship between the beta EEG activity and underlying cortical metabolic activity [16], and the results of decomposition of the increased beta activity into 3 independent components, it can be concluded that the neurofeedback intervention in this subject induced elevation of metabolic activity in the areas located near the Rolandic fissure.

Post- pre-changes of event-related potentials. Only the P3 NOGO wave was changed in the course of training. As shown in our previous paper [15], the P3 NOGO wave is decomposed into 2 independent components: 1) the P3 NOGO early component with latency of 340 ms and central distribution, and 2) the P3 NOGO late component with latency of 400 ms and more frontal distribution. In this study [17], these components were shown to be rather stable and did not change within the time interval of up to several months. In the other studies in which the task setting was manipulated [14] and the components were correlated with neuropsychological parameters [18], these 2 components were shown to have a quite different functional meaning. The numerous results of lesion studies enabled separation into 3 quite independent domains of the prefrontal lobe functioning, such as energization, monitoring and task setting [19, 20].

In our previous studies, the P3 NOGO early component disappeared when the subjects had to respond to GO and NOGO cues with different hands [14], and strongly correlated in amplitude with the parameters of energization neuropsychological domain [18]. These results enabled association of the P3 NOGO early component with the subject's ability to sustain attention, to respond as fast as possible, and to suppress the prepared action, i.e. with energization domain.

In contrast, the amplitude of the P3 NOGO late components strongly correlated with the other neuropsychological domain – the monitoring domain [19, 20], i.e. the ability to keep the balance between speed and accuracy in task performance. As the results of the presented study show, the neurofeedback training resulted in a selective increase in the energization component of the ERPs of the Olympic athlete under study. Therefore, it is a valuable technique to change the brain and life of individuals [21, 22, 23, 24], and therefore it can help to overcome or more effectively manage a variety of conditions in sportsmen who have lost the performance confidence after injury in sport.

CONCLUSIONS

The results of the presented study show that peak performance neurofeedback training in the highly-performing sportsman changed both the spontaneous EEG pattern and ERPs in the cued visual GO/NOGO task. The peak performance training resulted in an increase in high beta activity recorded centrally. Taking into account the positive relationship between beta EEG activity and underlying cortical metabolic activity, and the results of decomposition of the increased beta activity into 3 independent components, it can be concluded that the training induced elevation of metabolic activity in the areas located near the Rolandic fissure. It can also be concluded that Event-Related Potentials (ERPs) in the GO/NOGO task can be used as valuable neuromarkers to assess functional brain changes induced by urotherapeutical programmes.

REFERENCES

1. Hammond DC. Neurofeedback for the Enhancement of Athletic Performance and Physical Balance. The Journal of the American Board of Sport Psychology 2007; 1: 1.
2. Kropotov JD. Quantitative EEG, event related potentials and neurotherapy. San Diego: Academic Press, Elsevier, 2009.
3. Ziółkowski A, Graczyk M, Strzałkowska A, Wilczyńska D, Włodarczyk P, Zarańska B. Neuronal, cognitive and social indicators for the control of aggressive behaviors in sport. Acta Neuropsychologica 2012; 10(4): 537–546.
4. Kropotov JD, Ponomarev VA, 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(2): 565–575.
5. Kropotov JD, Ponomarev VA. Decomposing N2 NOGO wave of event-related potentials into independent components. Neuroreport. 2009; 20(18): 1592–1596.
6. Pachalska M. Rehabilitacja neuropsychologiczna. Lublin: Wydawnictwo UMCS, 2008 (in Polish).
7. Chiereghin A, Martins MR, Mori FGC, Ferreira Rosa R, Alvarenga Anti Loduca SM, Chahade WH. Posterior ankle impingement syndrome: a diagnosis rheumatologists should not forget. Two case reports. Rev Bras Reumatol. 2011; 51(3): 283–288.
8. Pachalska M, Kaczmarek BLJ, Kropotov JD. Neuropsychologia kliniczna: od teorii do praktyki. Warszawa: Wydawnictwo Naukowe PWN, 2009 (in Polish).
9. Vigário R, Särelä J, Jousmäki V, Hämäläinen M, Oja E. Independent Component Approach to the Analysis of EEG and MEG Recordings, IEEE Transactions on Biomedical Engineering, 2000; 47(5): 589–593.
10. Jung T-P, Makeiga S, Westerfield M, Townsend J, Courchesne E, Sejnowski TJ. Removal of eye activity artifacts from visual event-related potentials in normal and clinical subjects. Clinical Neurophysiology 2000; 111: 1745–1758.
11. Kropotov JD, Mueller A. What can Event Related Potentials contribute to neuropsychology? Acta Neuropsychologica 2009; 7(3): 169–181.
12. Makeig S, Bell AJ, Jung T-P, and Sejnowski T. Independent component analysis of electroencephalographic data. Advances in Neural Information Processing Systems 1996; 8: 145–151.
13. Onton J, Makeig S. Information-based modeling of event-related brain dynamics. Prog Brain Res. 2006; 159: 99–120.
14. Kropotov JD, Ponomarev VA, 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.
15. Kropotov JD, Ponomarev VA. Decomposing N2 NOGO wave of event-related potentials into independent components. Neuroreport 2009; 20: 1592–1596.
16. Cook IA, O'Hara R, Uijtdehaage SH, et al. Assessing the accuracy of topographic EEG mapping for determining local brain function. Electroencephalogr Clin Neurophysiol. 1998; 107: 408–414.
17. Brunner JF, Hansen TI, Olsen A, Skandsen T, Håberg A, Kropotov J. Long-term test-retest reliability of the P3 No Go wave and two independent components decomposed from the P3 No Go wave in a visual Go/NoGo task. International Journal of Psychophysiology 2013; 89: 1.
18. Brunner JF, Olsen A, Aasen I, Løhaugen G, Håberg A, Kropotov ID. Mapping neuropsychological domains of attentional control to Independent Components of Event Related Potentials [in press].
19. Stuss DT, Levine B, Alexander MP, Hong J, Palumbo C, Hamer L, Murphy KJ, Izukawa D. Wisconsin card sorting test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes. Neuropsychologia 38: 388– 402.
20. Pąchalska M, Kropotov ID, Mańko G, Lipowska M, Rasmus A, Łukaszewska B, Bogdanowicz M, Mirski A: Evaluation of a neurotherapy program for a child with ADHD with Benign Partial Epilepsy with Rolandic Spikes (BPERS) using event-related potentials. Medical Science Monitor 2012; 18:(11): 94–104.
21. Tomaszewski W, Mańko G, Ziółkowski A, Pąchalska M. An evaluation of health-related quality of life of patients aroused from prolonged coma when treated by physiotherapists with or without training in the 'Academy of Life' programme. Ann Agric Environ Med. 2013; 20(2): 319–323.
22. Pąchalska M, Kropotov ID, Mańko G, Lipowska M, Rasmus A, Łukaszewska B, Bogdanowicz M, Mirski A. Evaluation of a neurotherapy program for a child with ADHD with Benign Partial Epilepsy with Rolandic Spikes (BPERS) using event-related potentials. Medical Science Monitor 2012; 18(11): 94–104.
23. Pachalska M, Mańko G, Kropotov ID, Mirski A, Łukowicz M, Jedwabińska A, Talar J. Evaluation of neurotherapy for a patient with chronic impaired self-awareness and secondary ADHD after severe TBI and long term coma using event-related potentials. Acta Neuropsychologica 2012; 10(3): 399–417.
24. Kropotov JD, Pronina MV, Ponomarev VA, Murashev PV. In search of new protocols of neurofeedback: Independent components of event-related potentials. Journal of Neurotherapy 2011; 15:151–159.