

Evaluation of the effectiveness of neurofeedback in the reduction of Posttraumatic stress disorder (PTSD) in a patient following high-voltage electric shock with the use of ERPs

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Abstract

Background. The aim of our research was an evaluation of the effectiveness of neurofeedback in reducing the symptoms of Post-trauma stress disorder (PTSD), which had developed as a result of a high-voltage electric burn to the head. Quantitative EEG (QEEG) and Event related potentials (ERPs) were utilised in the evaluation.

Case study. A 21-year-old patient, experienced 4th degree burns to his head as a result of a high-voltage electric burn. The patient was repeatedly operated on and despite the severity of the injuries was to recover. However the patient complained of flashbacks, difficulties with sleeping as well as an inability to continue work in his given profession. Specialist tests were to show the presence within him of PTSD. As a result of which the patient was provided with neurofeedback therapy. The effectiveness of this therapy in the reduction (eradication) of the symptoms of PTSD were evaluated through the utilisation of **quantitative EEG (QEEG) and Event related potentials** (ERPs).

Results. It was found that in the first examination that ERPs display the most significant deviations from the reference in the two components: (1) the one component is generated within the cingulate cortex. The pattern of its deviation from the norms is similar to that found in a group of OCD patients. In contrast to healthy subjects the component repeats itself twice; (2) the second component is generated in the medial prefrontal cortex. Its pattern (neuromarker) is similar to that found in PTSD patients. There is a delay in the late part of the component, which probably reflects the flashbacks. In the second examination, after neurofeedback training, the ERPs were similar to the norm. The patient returned to work.

Conclusions. Chronic PTSD developed within the patient as a result of a high-voltage electric burn. The application of a method of therapy (neurofeedback) resulted in the withdrawal of the syndrome symptoms. ERPs in a GO/NOGO task can be used to plan neurofeedback and in the assessment of functional brain changes induced by neurotherapeutic programmes.

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Key words

electric burns, dyssomnia, Chronic Post-Traumatic Stress Disorder, executive functions

INTRODUCTION

There is an absence within the world subject literature of research devoted to a reliable evaluation of the effectiveness of neurofeedback in the therapy of those who have survived high voltage electric burns to the head, of a voltage of more than 1000 V,¹ within whom PTSD symptoms have developed [1, 2]. Such an evaluation as may be concluded on the basis of the results obtained in other pieces of research [3, 4, 5], may be obtained thanks to a testing of the event related potentials (ERPs). The absence of such tests is the result of the extremely high mortality rate for those involved in

high-voltage electric burns to the head. Those who have survived such an injury display various stress disturbances as well as disturbances to the cognitive processes and to their behaviour. The profile of these disturbances as equally the mechanism of their causes is not totally known. Depending on the genetic susceptibility [6] within the scope of emotional reactions PTSD may develop.

In our work we adopted a definition of PTSD based on the DSM-5 classification. We define PTDS as a disorder with

1. The basic physics of electrical current are represented by the formula $V=I \times R$. This identifies that voltage (V) is a product of current (I) and resistance (R). The terms AC and DC describe the flow of electrical current, with direct current (DC) traveling in one direction and alternating current (AC) resulting from the changing direction of the electrical flow. The number of field directional changes is referred to as a cycle, and 1 cycle per second represents 1 Hz. Standard North American household power is 60 cycles per second, or 60 Hz.

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symptoms that last for more than one month. There are various forms of post-traumatic stress disorder, depending on the time of onset and the duration of these stress symptoms. In an acute form, the duration of the symptoms is between 1 to 3 months. In a chronic form, symptoms last for more than 3 months. With delayed onset, symptoms develop more than 6 months after the traumatic event. Flashback is one of the axial symptoms of post-injury stress [7], involving a recurrence of recollections similar to hallucinations with a simultaneous absence of processing the actual stimuli. Most frequently the recollections are visual-pictorial in nature although there also occur symptoms containing aural or sound recollections. These concern an incident which resulted in brain damage (in patients with other forms of damage there is obviously an analogical flashback concerning injuries to other parts of the body). A repeat experience of a traumatic event leads to the appearance of stress as well as other emotions accompanying the accident such as fear or anxiety. These emotions may equal or even exceed the level that occurred during the accident. The frequency of the flashback symptom is extremely varied, it may be recollections occurring once every several years, while some patients experience flashback type recollections multiple times in the course of a single day. Most often this symptom is brought about by a definite stimulus (for example blood, or the sound of a car engine) which is associated with the accident [8].

The mechanism for the development of these disturbances is not only connected with the memory of the event itself, resulting in the burn, but also with temporary or permanent neurone organic changes within the brain. However, it is known that the current passing through the cells results in a series of instantaneous damages resulting from depolarisation and disturbances to the permeability of cellular membranes. There occurs damage to the cells, the clinical effect of which is extended in time. Additionally the differences in the tissue resistance are the cause for the various damage risk degrees at the moment the current passes through the tissue. Another damaging element is temperature increase as a result of the arising of thermal energy in the course of the through flow of current through tissues of a greater conduction resistance, an example of which being the skull. As a result of this there occurs thermal damage to those tissues adjoining the bone [9].

A characteristic feature of high-voltage burns is the noticeably reduced scope of damage on the surface of the body in the place of contact with the current when compared to the damage that occurs deep within the tissues. Varied resistance and conduction of tendons, muscles, blood vessels and nerves is the cause of the various levels of tissue damage that occur as a result of the flow of current. An additional and significant element resulting from the laws of physics is the dependence of the period of damage on the transverse section of the environs through which the current flows. For example, the passing of the same amount of current through the wrist and arm results in a greater level of damage in the wrist where the density of the current is the greater [9].

In the case of the passing of current through the skull this results in a lesser or greater neurone destabilisation and the subsequent disturbances in the emotional cognitive processes and ways of behaviour connected with it. If the patient remembers the injury, for he had not lost consciousness, there may result Post Trauma Stress Disorder (PTSD), and its consequent lowering of the quality of life.

The mechanism of this process can be linked not only to the psychic experiences which the injury itself leaves behind, with the awareness of tissue injury, but first and foremost with stress during the course of treatment as well as anxiety over one's own life. In the case wherein the wound refers to the head and the patient is aware of possessing a damaged, exposed and burnt skull as well as the risk of dying there appears a strong fear of death. When such a situation lasts several weeks as is the case in patients who have suffered high-voltage head injuries, then regardless of surgical success and treatment there remains enormous damage to the patient's psyche.

It is worth adding that if a patient did not lose consciousness and remembers the moment of injury and this fact is constantly stressed during the course of hospital treatment then PTSD may form with associated unpleasant symptoms for the patient. The care of medical personnel and the constant maintaining of hope as to the beneficial effect of treatment does not always provide sufficient psychological protection, for during the course of hospitalisation the patient is wearied by the illness, something which can also accentuate the risk of other symptoms developing.

The aim of our research was the evaluation of the effectiveness of neurofeedback in the reduction of the symptoms of chronic Post Trauma Stress Disorder (PTSD), which developed as a consequence of a high-voltage electric burn to the head. The evaluation made use of the event related potentials (ERPs).

CASE REPORT

A 21-year-old patient, an office employee, experienced a high-voltage electric burn as a result of the severing of a high-voltage cable of a voltage of 15 000 V during the course of work on the building's roof. The severed cable which was under voltage struck the patient in the head. The current entered the calvaria, and left through the area around the right wrist. As a result of the shock the patient fell from the roof. As a consequence of the accident the patient experienced 4th degree burns to the head. The burns covered an area of over 200 cm² in the vicinity of the calvaria (Fig. 1A). In addition the burns covered the right upper limb with deep 4th degree burns of the area around the right wrist. As a result of the fall the rib cage was bruised with right-sided pneumothorax. The patient was operated on a number of times, was treated in intensive care, was isolated from other patients in order to minimise the risk of wound infection. The medical and rehabilitation personnel conducted a strict sanitary regime whenever they entered the patient's room. The period of the first three weeks from the injury saw an intensification of the necrotic areas characteristic for high-voltage injuries. The bone areas of doubtful blood flow were drilled to the bone marrow and subsequently as a result of the occurrence of necrotic features the outer lamina of the skull was removed from the entire area as presented in Fig. 1B.

Surgical treatment and the application of the negative pressure therapy was conducted. During the course of treatment there was also carried out intensive rehabilitation, nutritional treatment, antibiotic treatment as well as treatment in a hyperbaric chamber (HBO). Hospitalisation at the Małopolska Burns and Plastic Surgery Centre, at The Rydygier Memorial Hospital, Krakow lasted two months

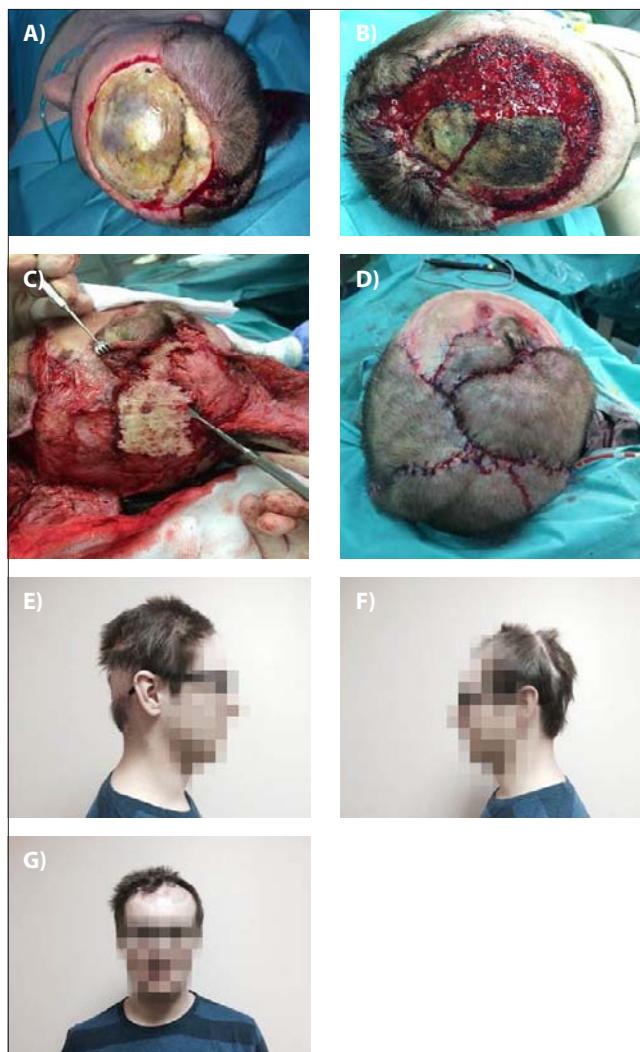


Figure 1. The effects of surgical treatment of the patient after experiencing of a high-voltage electric burn.

- A) 4th degree burns to the head: these covered over 200 cm² of the area of the cranial vault (Fig. 1A).
- B) The area not covered by granulation tissue represents the dead lamina of the internal skull (the post-burn necrosis to the total thickness of the skull).
- C) A dead fragment of the skull's internal lamina during removal.
- D) The second extensive plastic surgery of the integuments of the skull performed by a modified Orticochea banana-peel technique by the first author of this article
- E, F, G) The patient four months after the last procedure (May 2014)

from the moment of patient admittance during the first twenty-four hours following the high-voltage injury to the discharge of the patient home with residue scars in the area of the reconstructed scalp.

In the subsequent weeks of rehabilitation and the second cycle of oxygen treatment in the hyperbaric chamber there occurred the idiopathic debridement of the dead internal lamina, the fragments of which had punctured the skin in the fronto-parietal region through the post-burn scars. The patient had been admitted to the hospital and accepted for the meticulous planned surgical treatment. The necrotic part of the internal lamina of the skull had been removed. (Fig. 1C). During the course of this operation a second stage of reconstructive surgery of the scalp using a modified method of the Orticochea banana-peel technique was carried out [10] (Fig. 1 D).

The patient's state and condition four months following the procedure is presented in Fig. 1 E, F, G. One may note that

the post-burn scar has been moved to the occipital region, so that the initially burnt frontal and parietal region was covered by hairy skin without the need for secondary plastic surgical procedures. These actions are aimed to increase the patient's quality of life. Additionally the covering of the skull damaged as a result of the electric current by a complete scalp increased the patient's life safety.

THE EFFECT OF ACUTE ELECTRIC SHOCK TRAUMA ON THE NEUROPSYCHOLOGICAL FUNCTIONING

The injury was not accompanied by a loss of consciousness with the patient remembering the whole event; something which resulted in him suffering from strong fears and anxieties and recurring nightmares during sleep.

The brain MRI studies (Fig.2) showed changes at levels of the semioval centre (A), corona radiata (B), and the posterior limb of the internal capsule (C) demonstrated was an increased T2 signal intensity within the left corticospinal tract 5 months after the electrocution injury.

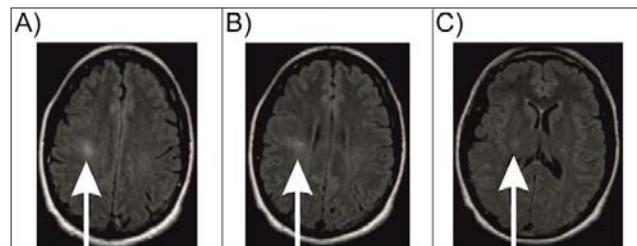


Figure 2. Brain MRI: Axial FLAIR images (TE = 140 ms; TR = 11,002; inversion time = 140 ms) obtained at the levels of the semioval centre centrum semiovale (A), corona radiata (B), and posterior limb of the internal capsule (C) demonstrate increased T2 signal intensity within the left corticospinal tract 5 months after the electrocution injury

The protocol of this study has been approved by the Bioethical Committee. The patient agreed to participate in this study. The study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

RESULTS OF THE NEUROPSYCHOLOGICAL TESTS

In May 2014: (1) fatigue, (2) arrhythmia, (3) fast breathing, (4) muscle pain, (5) fear/anxiety, (6) working memory loss memory problem (7) calculation impairment.

He was examined two times: the first examination was conducted before neurotherapy and the second after neurotherapy. The standard Polish version of the *Mindstreams™* Interactive Computer Tests called Neurotrax TR [11] was employed. The effect of acute electric shock trauma on the neuropsychological functioning is presented in fig. 3.

In the first test disturbances for all the tested cognitive functions occurred. The greatest changes were to occur however in the areas of attention, memory, visual-spatial functions and executive functions. In the second test a return to the norm was achieved for the disturbed cognitive functions, though for attention, sight-spatial functions and executive functions the patient obtained better results than the norm.

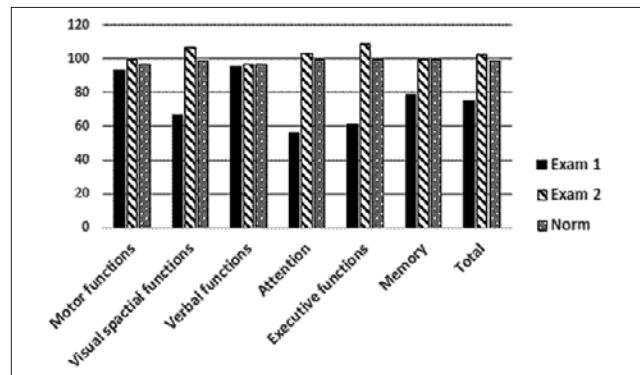


Figure 3. The effect of acute electric shock trauma on the neuropsychological functioning as measured by Neurotrax TR

THE EFFECT OF ACUTE ELECTRIC SHOCK TRAUMA ON BRAIN FUNCTIONING

EEG recording

The electroencephalogram (EEG) was recorded with the help of 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 were sampled at a rate of 250 Hz (bandpass 0.5–30 Hz). The ground electrode was on the forehead. Impedance was kept below 5 kΩ. The participants sat upright in a comfortable chair looking at a computer screen (17 inches) 1.5 metres in front of them. All recordings were done by the author of this article. ERP waveforms were averaged and computed off line and trials with omission and commission errors were automatically excluded [1].

The 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 the presentation 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** (see Fig. 1). In the trials with **A-A** and **P-P** pairs the first and the second stimuli were identical (physically the same). The trials were grouped into four sessions with one hundred trials in each. In each session a unique set of five **A** stimuli, five **P** and five **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 [1,4].

The task was to press a button with the right hand to all **A-A** pairs as fast as possible and to withhold from pressing in response to other pairs. The participants performed 10 trials without recording to see if they understood the instruction. They 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.

Artefact correction procedures

Eye blink artefacts were corrected by zeroing the activation curves of individual independent components corresponding to eye blinks. These components were obtained by the application of Independent Component Analysis (ICA) to the raw EEG fragments [1]. 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; and (3) 35 µV for fast waves filtered in the band 20–35 Hz. In addition we visually inspected the recordings and excluded the remaining artefacts.

EEG spectra

EEG spectra were computed for Eyes Open, Eyes Closed and the GO/Nogo task conditions separately. The artefact free fragments of EEG were divided into 4 sec epochs with 50% overlap. The Hanning time window was used. The EEG spectra were computed for each epoch and averaged. The Mean value and standard deviations for each 0.25 Hz bin were computed. For comparison EEG spectra pre and before intervention the t-test were used.

Decomposition of the collection of ERPs into independent components

To obtain valid independent components the number of training points is essential [1]. We used ERPs from 215 healthy subjects recorded under the HBIdb project [12].

ICA was performed on the full “ERP scalp location” x “Time series” matrix P. ERPs were constructed in response to the second (S2) stimuli at a time interval of 700 ms after the second (S2) stimulus presentation for the GO and Nogo cues. The 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 component generators remains fixed across time [1].

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 [1]. The ERP independent components of the subject participated in the present study were compared with the grand average ERPs of the healthy controls of the age 17–33 (N=?). The ERP independent components of the subject were also compared between the pre- and post-intervention conditions.

RESULTS

First recording (before neurofeedback intervention).

Behavioural data.

In Table 1 are presented the behavioural data in the cued GO/Nogo task during the first examination. No statistically significant deviations from the reference data were obtained. It should be stressed however that the subject was 100 ms faster than the average norm.

Table 1. Behavioural data in the cued GO/NOGO task

	Omission errors	Commission errors	Reaction time (RT) in ms	Error of the RT Variance in ms
Patient	2%	1%	319	7.9
Healthy controls	1.8%	0.5%	414	9.1
p-value	0.91	0.68	0.31	0.68

Spectra.

In EEG spectra in all three conditions (EO, EC, GO/NOGO task) the two characteristics were deviant from the normative average data. First, an increase in theta activity frontally (with the maximum at Fz) was observed (Fig. 3 a). In Fig. 3a the difference spectra (patient – healthy controls of the same age), the topography at 5.6 Hz and a fragment of EEG at Fz are presented. Second, an increase in alpha activity at the right parietal area was observed (Fig 3b). In Fig. 3b the difference spectra, the topography at 11.2 Hz and a fragment of EEG at P4 are presented. The EEG in EO condition was decomposed into independent components by means of Infomax algorithm. Extracted were two components that corresponded to the deviations in the spectra. The topographies and sLORETA images of these two components are presented in Fig. 4 a and b bottom rows.

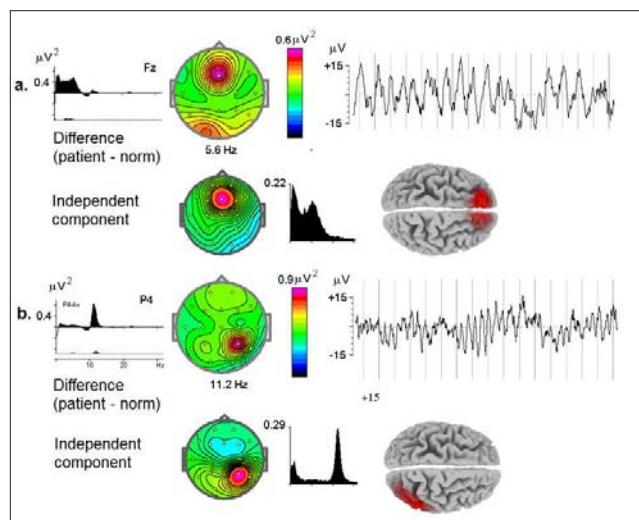
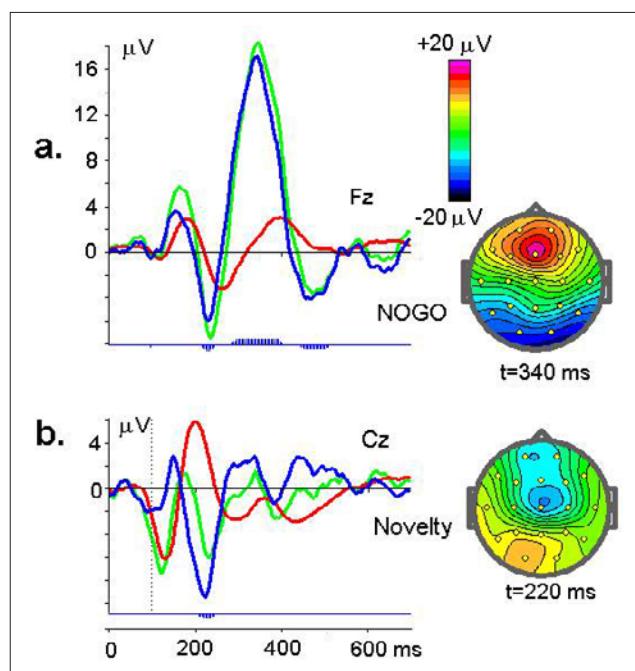


Figure 4. First recording – EEG spectra deviations from the norms
a. The frontal cortex deviation (Fz electrode). Top row – from left to right: 1) the difference EEG spectra (patient – grand average of a group of healthy subjects of the same age). At the bottom p-values of the confidence of the difference (small bars – $p < 0.05$, medium bars – $p < 0.01$, large bars – $p < 0.001$). 2) the map of the difference at 5.6 Hz, 3) an EEG fragment recorded at Fz. Bottom row – from left to right: 1) map of independent component corresponding to the deviation from the norms, 2) EEG spectra of independent component, 3) sLORETA image of independent component.
b. The right parietal cortex deviation (P4 electrode). Top and bottom rows as in a).

Event related potentials in the cued GO/NOGO task.

The results in comparison of the patient's ERPs in the two conditions (NOGO, and Novelty) are presented in Fig 5. The greatest differences were found in the NOGO condition (Fig 5a).

**Figure 5.** First recording. ERP deviations from the norms.

- a. From left to right – the NOGO ERPs recorded at Fz for the patient (green), the averaged group of healthy subjects (red) and the difference wave (patient-healthy) with p-values of the confidence of the difference; the map of the difference wave at the maximum ($t=340$ ms).
b. From left to right – the Novelty ERPs recorded at Cz for the patient (green), the averaged group of healthy subjects (red) and the difference wave (patient-healthy) with p-values of the confidence of the difference; the map of the difference wave at the maximum ($t=220$ ms)

Changes in brain activity after neurofeedback**Behavioural data in the cued GO/NOGO task.**

Behavioural parameters of the subject in the task were quite normal at the beginning and did not change during the training (Table 2).

Table 2. Behavioural parameters of the patient MG

	Omission	Commission	RT1	var(RT1)
Exam 1	2%	1%	319	7.9
Exam 2	3%	2%	346	8.5
Norms	1.8%	0.5%	414	9.1

Spectra.

The neurofeedback intervention produced dramatical changes in EEG spectra: the excessive alpha activity at the right parietal area completely disappeared. This is illustrated in Fig. 6.

ERPs.

As was shown above the response to novel stimuli was significantly suppressed in the first recording. The neurofeedback intervention led to normalization of the novelty response. This is illustrated in Fig. 7.

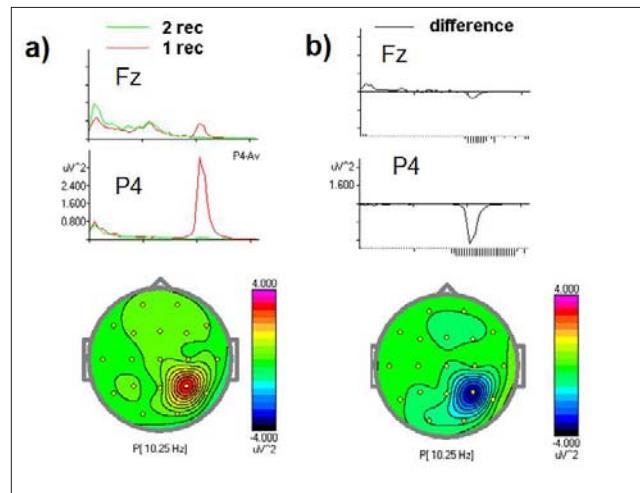


Figure 6. Changes in EEG spectra after neurofeedback treatment.
a. EEG spectra in EO condition in the first (red) and second (green) recordings at Fz and Pz. Note the peak at Fz (frontal midline theta rhythm) at around 6 Hz, which does not change after training. Note also the peak at 10 Hz at P4 in the first recording, which disappears in the second recording. The map below is taken for the first recording at 10 Hz.
b. EEG spectra differences 2rec-1 rec. Spectra difference waves are computed at Fz and P4. The levels of statistical significance are shown below the difference wave (small vertical bar – $p < 0.05$, medium vertical bar – $p < 0.01$ and large vertical bar – $p < 0.001$). The map below is taken at 10 Hz in the spectra difference

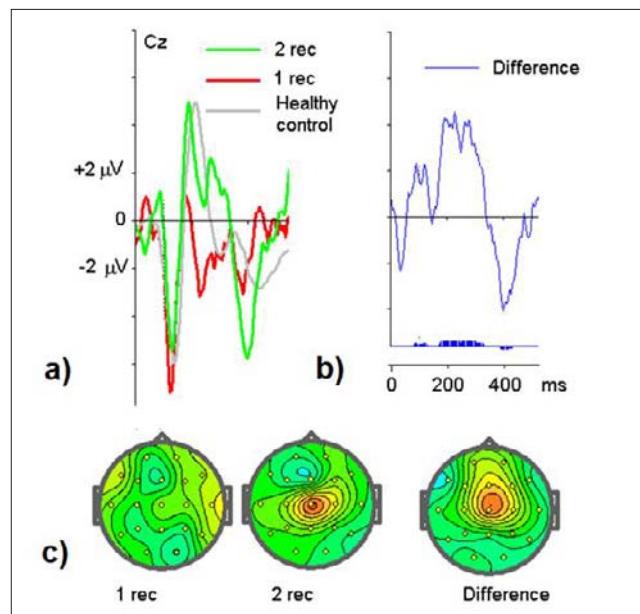


Figure 7. Changes in ERPs after neurofeedback training.
a. ERP to novel stimuli at Cz for the 1 recording (red), the second recording (green) and the grand average for the healthy control group (grey) taken at Cz.
b. ERP difference wave (2 rec – 1 rec). Below the curve – the levels of statistical significance are shown below the difference wave (small vertical bar – $p < 0.05$, medium vertical bar – $p < 0.01$ and large vertical bar – $p < 0.001$).
c. maps at 200 ms taken for ERPs at the first, second recording and the difference wave (2 rec-1 rec).

DISCUSSION

Remarkably, the patient survived the trauma and at the date of recording he was able to perform the cued GO/NOGO task quite well without any significant deviation from the reference. However, his EEG spectra and ERPs taken at the first recording before neurofeedback intervention were quite deviant from the average.

EEG spectra.

The most striking feature of EEG spectra was the increase of theta activity in the frontal leads. This theta activity runs in long bursts and is different from the frontal midline theta rhythm found in 20–40% of healthy subjects [1, 2]. According to the sLORETA imaging of the independent component related to the theta activity, this rhythmic activity is generated in the medial prefrontal cortex. However the neurofeedback treatment did not change this form of oscillations in the brain.

The other deviant variant of brain oscillations was found in the right parietal cortex. It was associated with excessive activity in the alpha band in the right parietal cortex. From neuroscience we know that alpha rhythms indicate inhibition (or idling) of the corresponding part of the cortex [1, 2]. This cortical inhibition might be caused by traumatic brain damage. The dysfunction occurred to be functional and transient because disappeared after the neurofeedback intervention.

Event related potentials.

In event related potentials the suppression of information flow was associated with the network involved in reaction to novelty. The P3 novelty was significantly reduced in the first recording. This pattern of deviation from normality was similar to that observed in a group of patients with TBI and PTSD [16]. And again this dysfunctional network remarkably recovered after the neurofeedback intervention: the novelty P3 was significantly enhanced after treatment.

So what happened in the patient's brain?

Following the high-voltage electric shock we confirmed in the patient several temporally distributed possible mechanisms for the destabilisation of the neuronal networks connected with:

1. Limited brain damage confirmed by MRI, which occurred as a result of the passage of the high-voltage current and the accompanying rise in temperature dependant on the skull resistance.
2. The stress associated with numerous operations and the change of dressings with general anaesthetic.
3. The awareness of the seriousness of the condition health wise and with the threat to life or the risk of death
4. Discharge from the hospital with still unhealed residual wounds and with the fear as to the correct care of the wounds while at home.
5. The personal awareness and intrusive worry as to the changed appearance from that prior to the accident brought about by the burn scars and the as yet unhealed wounds; something that results in a fixation on the accident itself and its aftermath.
6. The wait for another planned operation to reconstruct the hirsute scalp

This long-term process resulted initially in an accentuation of fears and stress reactions as well as a multiple reliving of the accident itself and its consequences. As a result of this there occurred a pushing out from the consciousness of the accident situation itself and its consequences. However, the patient was to experience flashbacks which caused a return in recollections and an increase in the level of fear. This complex mechanism resulted in chronic PTSD, in a similar way to that presented in individuals with brain injuries who remember the accident [8].

That said even though the application of modified techniques in reconstructive surgery resulted in the post-operative scalp obtaining a pleasing aesthetic and appearance the patient continued to suffer from post-injury stress syndrome. This points to the absence of conscious patient control over the neuronal activities of the brain. Particularly unpleasant for the patient were symptoms of:

1. flashback,
2. intrusive thoughts about stressful events,
3. sleep disturbance initially connected with the treatment conditions at the intensive care unit (the need for nursing care, the noise of the monitoring devices to which the patient was wired up, the lighting), and subsequently with the recurrent nightmares.

It is worth adding that burns treatment is concentrated on the saving of life and the closing of the burn wounds [17]. Despite the fact that a patient received constant psychological care while in hospital, following their discharge PTSD may appear together with a whole array of accompanying symptoms, as was the case in our patient.

Even though the patient's life was saved and he returned to full physical condition he was unable to return to his office job as a result of the impairment to certain cognitive functions (working memory and counting). The patient was able to carry out arithmetical operations only with much effort and was unable to concentrate.

Such disturbances may be explained by the fact that even minor damage to the structure of the brain, or a destabilisation of the neuronal connectors leads to its own stratification in behaviour [18, 19]. This happens because the complex neuronal networks create in their essence a dynamic system, one which is relatively easy to destabilise. Such a destabilisation of brain systems we can observe, for example, in states of extreme fatigue, on falling asleep and equally self-induced after the use of psychoactive substances, of which the most common is alcohol.

In analysing the results of the tests with regard to our patient we may equally imply the occurrence of just such a destabilisation (cf. Fig 8). Wherein brain damage, even the slightest, results in not only changes to the existing connections but forces new principles in their functioning. In the biological aspect this manifests itself in the form of neurological disturbances and/or cognitive ones, in the social aspect this results in disturbances in a patient's functioning, while in the personal aspect emotional changes arise (e.g., strong fear/anxiety, depression or symptoms of PTSD).

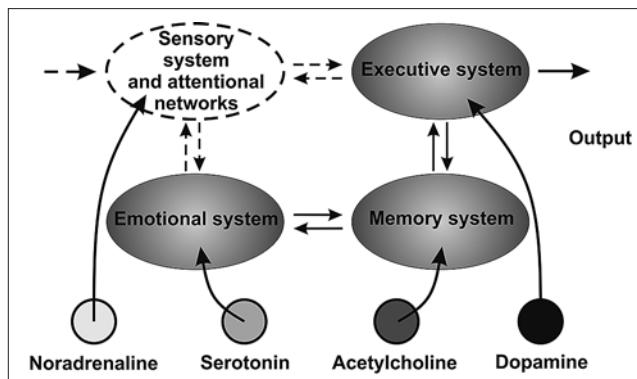


Figure 8. Destabilisation of the neurological networks connected with attention
Source: Pąchalska, Kaczmarek and Kropotov [20]

Destabilisation which initially covered the attention networks, was to shortly take over the memory system. On the one hand the patient wanted to forget the accident and all the circumstances connected with it, while on the other these thoughts intrusively returned both in the form of flashbacks as in dreams. The swamping of memory through stress generating events excluded the possibility for real thought on current situations strengthening the symptoms of PTSD.

Why did an improvement occur as a result of neurotherapy? The dynamic character of brain systems is taken into consideration in a patient's neurotherapy. This was based on the premise that it creates the possibility for a 'rebuilding' of the lost functions [1]. This was to find confirmation in the presently widely described phenomenon of brain plasticity. In cases of brain damage two phenomena take place: first and foremost there often occurs the spontaneous creation of new connections. These connections are, however, as a rule too weak, something presented in Fig. 2 graphically by the means of a broken line. They therefore disappear with time although quite often there is observed the phenomenon of a spontaneous return of the lost functions. For all this depends on the fact as to whether they have been appropriately strengthened. It happens that this occurs in the case of a spontaneous activity on the part of a given patient, however in the majority of cases there is a need for external reinforcement and stimulation. Effective therapeutic interaction requires, however, the appropriate knowledge and is linked to the making of a correct diagnosis. For in any other instance regardless of the effort and hard work put into the process by the therapist and patient the results will be minimal. Diagnosis of chronic PTSD confirmed in the ERPs tests was to prove a breakthrough in patient treatment. As a consequence an appropriate therapeutic protocol was modified to the modelled brain waves and neurofeedback was provided. After 20 sessions of neurofeedback the patient's state underwent noticeable improvement, something borne out by both the results obtained in the neuropsychological tests as the neurophysiological ones.

The use of games in the neurofeedback resulted in an improvement in the attention processes as well as those of memory enabling a change in the scheme of connections between the ganglia structures at the base of the brain. Obtained was a regulation of the amygdaloid nucleus as well as the hippocampus, and as an effect the deactivation of pathological connections and the conditions for the creation of new neuronal networks enabling the correct working of the brain cortex.

Even though certain authors suggest that in the case of electrical injuries there can appear and escalate cortical atrophy [21, 22, 23], the patient's psychic state stabilised, which allowed for its correct functioning in the environment. The abating of problems with cognitive functions (working memory and the ability to count) allowed the patient to return to work to his previously held management position.

The results obtained in the evaluation of the effectiveness of neurofeedback for the patient presented can act as proof as to the effectiveness of this method. They may be worthy of recommendation to other patients who have been subject to similar stress and neuronal destabilisation as equally those treated for high voltage electric burns. This method could have a significantly broader application and valuable

would be an analogical evaluation of the effectiveness of neurofeedback with the aid of ERPs on larger groups of patients suffering from PTSD connected with a stressful event exceeding the possibility for self regulation.

CONCLUSION

1. In the patient as a result of a high-voltage electric burn there developed chronic PTSD syndrome. The method of therapy applied (neurofeedback) resulted in the removal of the syndrome symptoms.
2. The ERPs show the most significant deviations from the reference in the novelty response. The response is associated with the frontal P300 wave and is generated in the medial prefrontal cortex. Its pattern is similar to that found in PTSD patients.
3. ERPs in a GO/NOGO task can be used to plan neurofeedback and assess the functional brain changes induced by neurotherapeutic programmes.

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