#### CASE STUDY

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- A Study Design
- B Data Collection
- C Statistical Analysis D – Data Interpretation
- E Manuscript Preparation
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# NEUROPSYCHOLOGICAL FUNCTIONING AFTER TRAUMATIC INJURY TO THE

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# ORBITO-FRONTAL AREA: A POLISH PHINEAS GAGE

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## **SUMMARY**

The patient (PG, age 27, owner/operator of a construction company) suffered a severe head injury after a fall from scaffolding at a construction site. Two wooden pegs were broken off and driven deep-into his cranium, from left to right. However, the patient not only survived the injury, he did not even lose consciousness after the accident. The damage to the brain affected primarily the frontal lobes. The upper peg penetrated the right frontal lobe. In the years that followed neurosurgery, we observed a pattern of behavioral disorders consistent with frontal lobe syndrome, similar to the famous 19<sup>th</sup>-century case of Phineas Gage. These symptoms make it impossible for the patient to adapt to social and cultural life. The purpose of our research was to describe the profile of negative and positive behavioral disturbances in PG, and to determine whether these behaviors developed over time into orbitofrontal syndrome. Microgenetic theory is used to interpret the formation of the symptom.

**Key words:** frontal syndrome, neurobehavioral problems, social adjustment, cultural adjustment

## INTRODUCTION

The frontal lobes constitute the youngest part of the brain, in both phylogenetic and ontogenetic terms. In the cat, they make up approximately 3.1% of the cortex; in the higher apes, 13%; and in the human, 24% (Langlois et al 2006; Hyder et al. 2007). Despite longstanding controversies over the details of how the frontal lobes function, it is difficult to avoid the rather obvious conclusions that can be inferred from the facts of evolution and the development of the human brain: namely, that the frontal lobes, and in particular the prefrontal area, are of fundamental importance for the formation of specifically human behaviors (Pąchalska, Kaczmarek Kropotov 2014).

Due to their position in the skull, the frontal lobes are often exposed to impact during an accident, as for example in a collision, a fall forward, etc. Damage to this part of the brain, according to many authors, is the cause of disturbances in behavior control, but it is no easy task to define the nature of these disturbances (Kolb & Whishaw 2003).

In the neuropsychological literature one can find two ways of explaining how the frontal lobes control behavior:

- 1. The frontal lobes are responsible for forming and initiating the programs of action that determine the person's behavior (Goldberg 2001), especially voluntary behaviors. In daily life we can constrain our behavior when the purpose or conditions of action warrant, and choose another variant that is more effective, simpler, or preferred.
- 2. The frontal lobes are responsible for inhibiting undesirable behaviors by rejecting, from among numerous possible responses to a given situation, those options which would be contrary to moral, religious, cultural, or personal values (Brown 2010).

These approaches are not necessarily mutually exclusive, since, due to the complexity of both the neuroanatomical structures involved and the behaviors being regulated, it seems unlikely that one, relatively simple mechanism can be responsible (Fuster 2000, Pąchalska 2007).

Clinical observation and scientific research suggest that patients with damage to the frontal lobes exhibit more or less predictable disturbances of behavior control, forming what is now generally called "frontal syndrome" (Kaczmarek 2012; Pąchalska 2007, 2008; Pąchalska, Kaczmarek & Kropotov 2014). The concept of frontal syndrome was developed primarily, however, in the context of neurode-generative diseases, which causes the literature to be dominated by research on patients with dementia and mental illnesses resulting from degeneration of the frontal lobes (Kertesz et al. 2000). Relatively little attention has been paid to frontal syndrome in patients with traumatic brain injury (TBI), even though the whole history of this concept really begins with a TBI case: the famous case of Phineas Gage (Pąchalska 2007), an American railroad engineer who in 1848 underwent a catastrophic brain injury when a tamping iron was accidentally shot through his left frontal lobes. Gage miraculously survived this massive injury, but

his behavior and character subsequently underwent such radical changes that he could no longer function in social life, at work, or at home. The changes that took place in this patient caught the attention of specialists at the time, so that the case of Phinease Gage (or more precisely, the wide attention it received among physicians at the time) is now widely recognized as the beginning of neuropsychology (Kolb & Whishaw 2007). Moreover, the frequency of citations in the literature indicates how often researchers return to this case in order to illuminate some essential issues associated with the functions of the frontal lobes and the results of frontal damage.

Lebrun (1995) states that the most characteristic clinical signs of frontal syndrome are the following:

- cognitive disturbances, including perseveration, distractibility, unsystematic scanning of images (with a tendency to concentrate attention on a relatively small area), difficulties in recalling previously learned information when other information interferes, and impaired access to information stored in memory;
- various specific disturbances of speech, depending on whether the damage is located in the right or left hemisphere: when the right frontal lobe has been damaged, the dominant features are aprosody, frequent use of verbal stereotypes, literal interpretation of metaphors, proverbs, etc., inability to identify or interpret similarities and differences, and inability to generalize by induction; when the left hemisphere has been damaged, there is nonfluent aphasia and apraxia of speech (sometimes also motor apraxia);
- *disturbances of affect and mood*, including apathy, indifference, emotional lability, excessive joviality, flat affect, mood swings, occasionally catastrophic reactions, irritability, quarrelsomeness, and a tendency to be easily insulted;
- neurobehavioral disturbances, including irresponsibility, a tendency to inappropriate joking, possibly exhibitionist tendencies, impulsiveness, excessive risk taking, and lack of common sense.

It should be emphasized, however, that this complex configuration of behavioral symptoms manifests itself differently in different people. This results from the type and localization of the injury, the interruption of synaptic connections, neurochemical disturbances, etc., not to mention individual differences.

Three main types of frontal syndrome are distingushed in the neuropsychiatric literature (Miller & Cummings 2006; Pąchalska 2007):

- frontal convexity syndrome, which is characterized by many symptoms, including disturbances of psychomotor drive, loss of initiative and spontaneity, apathy, abulia, depressed mood, lability, reduced feelings of responsibility or ambition, attention deficits, recall deficits, and loss of the capacity for abstract or cause-and-effect thinking (Kaczmarek 2005; 2012).
- orbitofrontal syndrome, which involves reduced or absent sociability, disinhibition, inappropriate affect, weakened criticism, loss of insight, emotional lability (fluctuating between insouciance and rage), and a tendency to tell silly jokes and frequently off-color stories (moria);

 medial frontal syndrome, in which the damage affects the anterior portion of the cingulate gyrus (Miller & Cummings 2006); the main symptoms are apathy (loss of motivation and reduced purposeful activity), weakness in the lower limbs, loss of sphincter control, and logopenia.

Kertesz et al. (1997) developed the Frontal Behavioral Inventory (FBInv), which can be used to evaluate the occurance of these symptoms. The items in the questionnaire are grouped in two general categories, negative (resulting from the disappearance of behaviors exhibited premorbidly) and positive (resulting from the appearance of behaviors that were not exhibited premorbidly; see Pachalska 2007, 2008; Pachalska, Kaczmarek & Kropotov 2014):

- the negative symptoms include apathy, loss of spontaneity, indifference, inflexibility, concrete thinking, personal neglect, distractibility, inability to focus attention, loss of insight, logopenia, verbal apraxia, and "alien hand" syndrome (these last three items were included in the FBInv in order to evaluate specific motor and linguistic behaviors that can occur in fronto-temporal dementia);
- the *positive* symptoms resulting from disinhibition, such as perseveration, irritability, joviality, irresponsibility, inappropriate behavior, impulsivity, euphoria, aggression, hyperorality, hypersexuality, utilization behavior, and urinary incontinence.

The purpose of our research was to evaluate the occurrence of negative and positive disturbances of behavior in a patient who experienced a major injury to the anterior frontal lobes of both hemispheres, and to determine whether the pattern of these disturbances changed over time to take on the characteristics of orbitofrontal syndrome.

## CASE STUDY

The patient (identified here by the initials PG) was 27 years old, a businessman running his own construction company, when he suffered a head trauma resulting from a fall from high scaffolding at the building site. Two wooden pegs broke off and penetrated deep into the cranium, from left to right, producing the kinds of wounds that might have been suffered on a medieval battlefield (Fig. 1A). However, not only did PG survive this horrific accident: he did not even lose consciousness. The damage to the brain affected chiefly the frontal lobes. The upper peg penetrated the right frontal lobe of the brain (Fig. 1B).

A CT scan of PG's head was conducted prior to neurosurgery. The arrow in Fig. 2A indicates a hole (appearing as a black space, since the peg does not cast a radiological shadow), and also visible fragments of the bone driven inside the right frontal lobe. In the CT scan made post-surgery, the arrow indicates a pocket of air left in the brain after the peg had been extracted (Fig. 2B).

After eight weeks, the injury had nearly healed. Follow-up neuro-imaging examinations showed that the frontal cortex of both hemispheres had been damaged, and that the damage also affected the medial orbital (supraorbital) region above, behind, and to the right of the pre-central region (cf. Pąchalska 2007).

#### Pąchalska et al., A Polish Phineas Gage

In neuropsychological examinations conducted after surgery, no disturbances of cognitive processes (attention, memory, speech and intellect) were detected. The only deficit discovered at this point was slow reaction time in the tapping test from the Mindstreams<sup>™</sup> Computer Interactive Test Battery (cf. Pachalska 2008). What attracted our attention, however, was PG's behavior when he was tired. The mistakes he made while moving a rectangle to catch an object falling from the upper edge of the screen provoked him to more and more abrupt and impulsive reactions, as he tried to solve problems with less and less success. When he was not able to catch the object, he commented on the task, using rapid, impulsive utterances. He was incapable of inhibiting this behavior or subjecting it to analysis. Iimpulsive and abrupt reactions are frequently connected with damage to the frontal lobes (cf. Pachalska 2007).

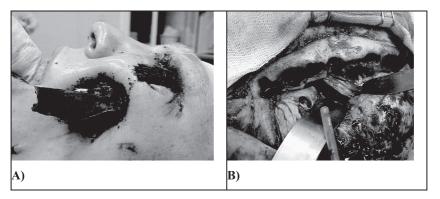


Fig. 1. Patient PG, age 27, after a neurosurgical operation necessitated by damage to the brain after a fall from high scaffolding at a construction site. A) two wooden pegs broke off and penetrated the cranium from left to right. B) the upper peg penetrated the right frontal lobe. *From: Pachalska & Moskała (2010)* 

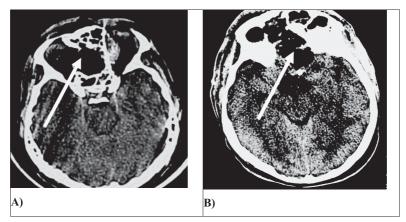


Fig. 2. CT scans of PG's head. A) prior to surgery: the arrow points to a black hole in black (the peg does not cast a radiological shadow), and also visible fragments of the skull driven inside the right frontal lobe. B) after surgery: the arrow indicates an air space left after extraction of the peg *From: Pachalska & Moskała (2010)* 

During the first year after the accident, PG developed increasing signs of behavioral disorders, aggressiveness, and impulsiveness. PG's results in the FBInv (Polish version by Pachalska & MacQueen 2000), especially the second 12 items (which measure the positive behaviors caused by disinhibition), indicate that in his case the positive symptoms were markedly worse, so the extent that a diagnosis of frontal syndrome was justified (see Fig. 3). The highest scores, which had reached the maximum, were observed in aggressiveness, anxiety, impulsiveness, inappropriateness and lack of good manners, unreasonable behaviour, hypersensitivity, and irritability.

In the years that followed, neurobehavioral disorders consistent with "Phineas Gage syndrome," or in other words, personality disorders resulting from damage to the frontal regions, continued to increase (Pachalska 2007). These symptoms included:

- · increased impulsiveness and aggressiveness;
- rudeness;
- hoarding behavior;
- a tendency to confabulation;
- the loss of the ability to manage his affairs rationally.

PG's mother emphasizes that the most difficult thing to cope with is his impulsiveness and aggressiveness, as well as rudeness. His hoarding is less of a nuisance; for example, after he read the novel *The Bone Collector* by Jeffrey Deaver, PG started collecting chicken bones and hiding them deep inside the

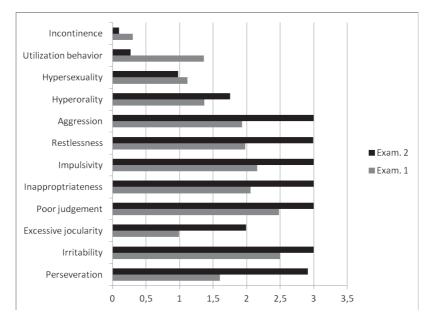


Fig. 3. PG's results on the FBInv, from the second 12 items, connected with disinhibition) at baseline (one year after the accident) and follow-up (three years after the accident). The scores as assigned on a scale from 0 to 3 points, where 0 means no disorder, and 3 means a serious and continuous problem.

couch. The financial situation of the family deteriorated dramatically because of his loss of the ability to manage his own affairs rationally. By no means could he manage his own finances, because of his extravagance. His wife, who is a professional accountant, claims that her husband has changed utterly:

Before the accident, he was a nice, thoughtful, responsible man, and a good worker. After the accident, he changed beyond all recognition. He has become aggressive, rude, insensitive to his own family and friends, and he shows a lack of interest in work, or learning anything new. He is terribly impulsive, irritable and rude, and his speech is full of the foulest language. He does not have much in the way of good manners, does not know how to behave in the company of other people, hates any restrictions and cannot stand good advice if it contradicts his own opinions. When he hears it, he becomes incredibly furious. He is very, very stubborn and moody. He can neither make plans nor execute them. Actually, he does make a lot of plans, and afterwards he drops them and comes up with new ones which seem to him more viable. He has started drinking and wanders aimlessly. His friends and family keep saying that 'this is not the same fellow anymore'. His home and his company are in a terrible mess. It was necessary to close the company. In his behavior, he resembles, on the one hand, an impatient and spoiled brat, while on the other hand, he displays the full, animal passions of a strong man, which he uses to dominate me and everybody in his environment. This is quite a strange man, and an evil one, now, little more than a bum, and I have nothing in common with him. Nor do I want to. I was afraid that he might kill me one day, and that's why I divorced him.

## DISCUSSION

An analysis of PG's symptoms subsequent to the destruction of a large part of the anterior frontal lobes in both hemispheres shows diverse behavioral disturbances characteristic of frontal syndrome, towards which the patient exhibits no insight. For his family, the most troublesome symptoms have been those resulting from disinhibition.

The opinion can be found in the literature that the primary mechanism of disinhibition in patients with frontal syndrome involves the lack of criticism (Zola & Squire 2000; Gainotti 2003; Kolb & Whishaw 2007). Careful clinical observation, however, suggests a somewhat different interpretation of the characteristic disinhibition in these patients. Many of them, when asked later for an analysis of their own behavior in particular situations, can evaluate it more or less reasonably. The patients quite often come to the conclusion that their behavior was essentially inappropriate. In this respect, the behavioral disturbances occurring in patients with frontal syndrome differ significantly from those of patients with dementia or psychosis, although from the point of view of the family, the effects are quite the same. The patient's inability to initiate appropriate behavior or inhibit inappropriate behavior at the proper time is perceived as a defect of either reason or good will.

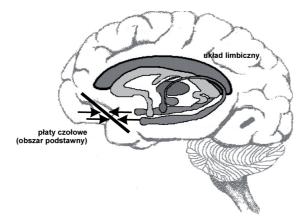
According to Fuster (2000), impairment in the functioning of the frontal lobes does not lead directly to the loss of basic brain functions, but rather to the loss of "metafunctions," that is, functions that control the course of the remaining brain functions. As a result, for a patient with frontal syndrome:

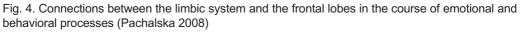
- a given behavior can still be performed the same as before the accident;
- a critical analysis of this behavior is also still possible;
- the control of this behavior in real time becomes difficult or even impossible.
  Fuster's views are confirmed by the clinical symptoms observed in our patient.

However, long term observation has shown that over time the changes in emotional functioning, personality, and behavior have intensified (cf. Damasio 1999; Zieniewicz 2011). Orbitofrontal syndrome began to appear, and in the description given here, it seems clear that the pattern of behavioral symptoms was very similar to that presented by Phineas Gage. Some authors (cf. Pachalska 2007; Pachalska, Kaczmarek & Kropotov 2014) have suggested an analogy between the symptoms occurring in patients with frontal syndrome and the behavior of the protagonist of Robert Stevenson's 1886 novella, The Strange Case of Dr. Jekyll and Mr. Hyde. The fictional doctor Jekyll is a noble and brilliant scientist, a student of the relationship between the brain and character (in our terms, he would be a neuropsychologist). He discovers a drug which he believes will be able to destroy all the evil that lurks in the human soul, and experiments on himself. However, the effect of the drug was not what he had expected: his personality was completely separated into two different personalities, Jekyll and Hyde, containing respectively all the good and evil aspects of his character. Under the influence of the drug the aggressive and thoroughly wicked Edward Hyde emerged from the good doctor's psyche, and began to live his own life, in complete contradiction to all the values held by Jekyll.

While the fictional Dr. Jekyll remained all the time one and the same person, in whom the drug evoked radical changes of personality, our patient, who before the accident was rather similar in his behavior to Dr. Jekyll, became Mr. Hyde after the accident, and for many years. As a result of his brain injury, the balance between human feelings and animal drives was upset. Like Phineas Gage, he was childishly insouciant, and did not respect social norms. He made ill-thought-out decisions; he was unable to establish strong bonds with other people, and did not respect any social or cultural values. He lost the ability to control his own life.

An analysis of the neurobiological mechanisms underlying this behavior suggests that the injury may have severed the connections between the orbitofrontal regions, the limbic system, and other brain structures (see Fig. 4). The normal flow of activation in the brain was disrupted, and because of the loss of limbiccortical connections, the patient after injury to the orbital regions has difficulties in maintaining "adult" or "civilized" behavior (Pachalska 2008; Pachalska & Mac-Queen 2008).





Under these circumstances, behavioral disturbances can be observed as the first visible signs of dysfunction, which, with appropriate diagnostic preparation, can be the basis for proper neurorehabilitation (Pachalska 2008).

The cortex (especially the frontal lobes) does not so much cause behavior as constrain it, shape it, sculpt it to fit more precisely the situation into which the behavior must fit. Anatomically, this is reflected in the extensive connections (Fig. 5) between the frontal lobes and the limbic system (Pachalska 2007).

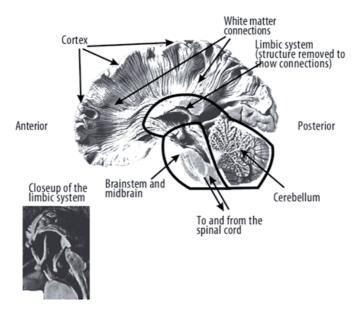


Fig. 5. Medial cross-section of the human brain and white matter connections (From: Pachalska & MacQueen,2008)

The frontal lobes retard, suspend, or even interrupt the algorithmic stimulusresponse mechanisms, to allow parcellated perception and action to develop. This mechanism surely accounts, at least in part, for the intuitive conviction that "good" behavior is characterized by restraint (read: self-imposed constraints), and for psychological theories from Socrates to Freud, according to whom there is a constant struggle within the psyche between impulse and inhibition. The same thing goes on within each cell of the organism, as changes in the chemical and physical environment cause various genes in the DNA strands to be activated or deactivated, and specific receptors in the cell walls are opened by agonists or closed by antagonists. Nature displays a great conservatism of principle, constantly repeating the same themes at different levels of organization, from atoms and molecules through cells and tissues up to human societies and the cosmos.

In microgenetic theory, the flow of mental process is from the archaic to the recent in forebrain evolution, from the continually re-activated past to the present moment, which is born and then immediately dies and decays. Momentary cognitions are extrapolated from memory to perception, and from the intrapsychic to the extrapersonal in spatial and temporal representation. Mental process is uni-directional, obligatory and recurrent, like the flow of a fountain, whose water continually circulates in such a way that the stream, though in reality it is constantly changing, seems to be constantly the same. What is fixed in a given state of mind is the cumulative series of phases traversed in a given actualization as it rises and falls in this "fountain" structure. Continuity, the ability to sustain a thought, an argument, a train of associations, even a mood, results from the constant repetition of these actualizations, which to the last moment traverse the same routes, and from the fact that a new actualization always appears before the last has faded. This overlapping of past and present gives the psychological present a certain duration, which for William James (1890) was one of the central guestions of psychology (cf. Brown 2004, 2005).

Each present moment, then, is derived out of the memory and past experience of the antecedent state. It appears, perishes and is replaced, like the overlapping pulses or drops of experience described by James, or the succession of *ksana* ("point-instants") in Buddhist metaphysics. External and internal constraints on this process, such as sensation and habit, determine the degree of novelty in the mental contents. Concepts and categories may control the process in an algorithmic manner, virtually a reflex, and be derived all the way down to objects perceived in the world without pausing to consider alternatives, or the cognitive process may undergo neoteny (the prolongation of an earlier stage of development or maturation) at an earlier, imaginal or conceptual phase, to serve as a springboard for the propagation of novel content. This is, once again, the evolutionary principle of branching: novelty does not appear from a relentless forward movement, but from a branching that occurs at some point before the main process reaches its natural endpoint. The content of the neotenous image may be reproductive, as in the case of a memory image, or productive, as in imagination (or hallucination). There is, to be sure, some novelty in every act of cognition (no two moments are ever exactly alike), but what is productive should be interpreted as a deviation from a more general trend to repetition. Without the stability of this repetition the mind/brain would jump from one world to the next in each blink of the eye, which is very nearly what seems to be occurring in frontal lobe syndrome.

This interpretation brings us closer to an evolutionary, process-based model of brain work. In the case of damage to the frontal lobes we should remember that it is not so much specific cognitive functions that undergo disruption, as occurs in focal lesions to the brain, but rather entire brain systems. The work of these systems is first disorganized in various segments, and then undergoes a global disturbance, when the level of destabilization reaches the point of disintegration.

At least since the time of A. R. Luria (1970; 1976), it has been understood that damage to the brain does not add anything new to behavior, but rather removes significant components of behavior. Every brain injury results in greater or lesser disturbances to the electrical and chemical language of the central nervous system, changes in the area of existing connections, as well as in the action and activity of neurotransmitters, which results in a destabilization of brain systems (Fig. 6). This leads to a reduction in the effectiveness of the action of specific constitutive activities (which vary, depending on the location and depth of the injury), and therefore in disturbances in behavior; in normal circumstances, these activities occur without any noticeable effort (Pąchalska & Kaczmarek 2012; Pąchalska, Kaczmarek, Kropotov 2014).

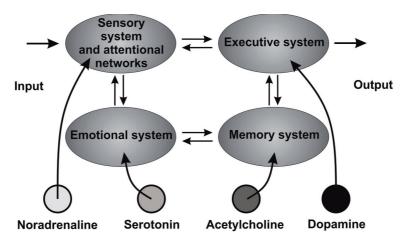


Fig. 6. Schematic representation of the conditions of a given system's responses depending on their arousal on entry and the activating neuromodulators in the brain. The reaction is defined as the response of the system to a small and elementary growth in arousal on entry (cf. Pachalska, Kaczmarek, Kropotov 2014)

The efficiency of every system (emotional, sensory, memory and executive) is defined as the ability of that system to react to small changes of input. Mathematically, the efficiency of a system is defined as the ratio: dO/dI, that is, the ratio of the differential output to the differential input.

It will be worthy to disscuss, that new discoveries in the neurosciences have made it possible to develop a program of therapy for our patient, in this case rTMS neurotherapy. In a previous study (Pąchalska et al 2011), we described a severe TBI patient suffering from anosognosia, executive dysfunction, and behavioral changes, who took part in 20 sessions of relative beta training, and later in 20 sessions of rTMS; both programs were combined with behavioral training. We used standardized neuropsychological testing, as well as ERPs before the experiment, after the completion of the neurofeedback program, and again after the completion of the rTMS program.

The patient showed small improvements in executive dysfunction after the conclusion of the relative beta neurofeedbak training program, and major improvement after the rTMS sessions. To monitor the physiological mechanisms of cognitive control in this patient, the ERPs in the cued GO/NOGO task were measured before the therapy, after the neurofeedback sessions, and after the rTMS sessions. Similarly to the behavioral parameters, the P3 NOGO component, as an index of cognitive control, showed small changes after relative beta training, but significant improvement after the rTMS program (Fig. 7).

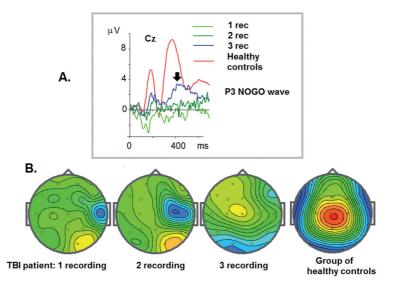


Fig. 7. Increase of the P3 NOGO wave in a TBI patient during sessions of neurotherapy (adapted from Pachalska et al., 2011). Event-related potentials (ERPs) were recorded at Cz in the TBI patient before treatment (1 rec), after neurofeedback sessions (2 rec) and after rTMS sessions (3 rec), and compared to ERPs in a group of healthy subjects. X-axis – time in ms after NOGO stimulus presentation. Y axis – potential in  $\mu$ V. In the map red corresponds to positive values, blue to negative values

This study reveals that the rTMS program produced larger physiological and behavioral changes than did relative beta training. In other works, we proved that a combination of different neurotherapeutical approaches (such as neurofeedback, rTMS, tDCS) can be suggested for similar severe cases of TBI. ERPs can be used to assess functional brain changes induced by neurotherapeutic programs (Kropotov 2009).

The patient is not entirely convinced that he needs any treatment, since he does not think that he is impaired in any way. It would be reasonable to assume, then, that he displays certain characteristics of anosognosia. Nevertheless, he has gradually become aware that he needs to work on himself, since he sees other people's negative reactions to some of his behaviors.

To sum up, it should be emphasized that the results described here, which make it possible to develop specific behavioral profiles of positive and negative symptoms, can be important for further therapy. They also make it possible to suggest the proper forms of neurotherapy. They can contribute, then, to improving the quality of life of persons with damage to the orbitofrontal cortex.

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