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Case Study

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Evaluation of a neurotherapy program for a child with ADHD with Benign Partial Epilepsy with Rolandic Spikes (BPERS) using event-related potentials

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

Background: We hypothesized that there would be a good response to relative beta training, applied to regulate the dynamics of brain function in a patient with benign partial epilepsy with Rolandic Spikes (BPERS), associated with neuropsychiatric deficits resembling the symptoms of attention deficit-hyperactivity disorder (ADHD).

Case Report: The patient, E.Z., age 9.3, was suffering from neuropsychiatric symptoms, cognitive dysfunction, especially attention deficits, and behavioral changes, rendering him unable to function independently in school and in many situations of everyday life. He was treated for epilepsy, but only slight progress was made. The patient took part in 20 sessions of relative beta training combined with behavioral training. We used standardized neuropsychological testing, as well as ERPs before the experiment and after the completion of the neurotherapy program. Neuropsychological testing at baseline showed multiple cognitive deficits. Over the course of neurotherapy, E.Z.'s verbal and non-verbal IQ increased significantly. His cognitive functions also improved, including immediate and delayed logical and visual recall on the WMS-III, maintaining attention on the WMS-III, and executive functions, but remained below norms. Physiologically, the patient showed substantial changes after neurotherapy, including fewer spikes and an increased P300 NOGO component.

Conclusions: The cognitive deficits characteristic for ADHD in a child with BPERS may be unresponsive to antiepileptic treatment, but are reversible after a carefully selected neurotherapy program, combined with antiepileptic treatment. Event Related Potentials (ERPs) in the GO/NOGO task can be used to assess functional brain changes induced by neurotherapeutical programs.

key words: **interictal spikes in EEG • cognitive dysfunctions • neuropsychiatric changes**

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Background

Rolandic epilepsy (RE) is the most common epilepsy syndrome affecting children [1]. It is a developmental epilepsy with a complex genetic inheritance that has yet to be elucidated [2]. Centrotemporal spikes (CTS) are the electroencephalographic hallmark [1].

RE is also known as benign rolandic epilepsy of childhood (BREC) or benign epilepsy with centro-temporal spikes (BECTS) [3]. 'Rolandic' means that the seizures begin in the part of the brain called the Rolandic area. The seizures are classified as a partial seizure because only this one part of the brain is involved [4–6].

The syndrome is called 'benign' because it has a good outcome: nearly all children with RE will outgrow it during puberty. It affects almost one in five of all children who have epilepsy. which makes it is one of the most common types of epilepsy in children. It affects boys and girls equally. RE usually begins between the ages of 3 and 10 years, and often stops around puberty (age 14–18) [6,7].

Some children who have this type of epilepsy are usually well otherwise and do not have learning difficulties, although some may have specific difficulties with reading and language or with drawing and visuo-spatial skills [1,4,8,9], and some have associated neuropsychiatric deficits resembling the symptoms of attention deficit-hyperactivity disorder (ADHD), the most common neurobehavioral disorder of childhood [7,9].

It was gradually realized that there was a close relationship between benign partial epilepsy with Rolandic Spikes (BPERS) and acquired epileptic aphasia (Landau-Kleffner syndrome), which was the first example of a mainly "cognitive" epilepsy in children [9]. Prolonged reversible oral-motor deficits were subsequently recognized during the active epilepsy phase in some children with an otherwise typical syndrome and good final prognosis. These cases showed that this epilepsy syndrome could cause prolonged "epileptic" deficits. Several neuropsychological studies confirmed the clinical experience that children with BPERS had normal intelligence but that a certain percentage of them showed variable attention or selective deficits (linguistics, visuospatial etc.), as compared to normal controls [1,8,9].

Recent studies on the frequent temporary cognitive-behavioral disorders encountered in BPERS have placed emphasis on their probable direct epileptic origin [9]. This was thought to possibly explain some of the learning and school problems that many of these children experienced in the active phase of the syndrome. Rare longitudinal correlative EEG-neuropsychological studies have recently shown that acquired temporary cognitive-behavioral problems correlate with epileptic activity (EEG) in some children [6,10,11]. It is now an open question whether this epilepsy can cause a specific developmental learning disability, or more general cognitive disability, if the onset is severe, early, and affects brain areas other than the strictly "rolandic" [10].

To this end, event-related potentials (ERPs) were measured during a visual 1-backmatching task. EEG spectra and ERPs in a patient with Rolandic interictal spikes were compared with the normative data (HBIdatabase) in order to estimate the main neurophysiological deficits found in this patient.

The deviations from the normality are discussed in term of well-known pathophysiological patterns in the ADHD population, such as increased in the theta-beta ratio [5], decrease of the P3b component [6–8] and decrease of the P3 NOGO potential [9,10]. A quantitative analysis technique to analyze the ERP data, without any "a priori" decisions on "peak" presence, amplitudes or latencies, is used. The frequency of rolandic spikes in children with ADHD is significantly higher than that expected from epidemiologic studies. Also, ERPs are of significantly higher amplitude in the epilepsy group compared to the control group over frontal and central regions within the time window between 250 and 425 ms post-stimulus, which coincides with the time window of target-nontarget stimulus discrimination [1,3,4].

The question arises how ADHD symptoms are related to rolandic spikes in this ADHD subgroup and how these symptoms can be treated by neurotherapy. What follows is a case study, in which we address this question.

The aim of this study was to find out:

1. whether this boy with rolandic epilepsy shows different cortical activation patterns compared to non-epileptic children during the performance of a working memory task;
2. does relative beta training, which activates the frontal cortex by enhancing beta activity recorded over the frontal electrodes, helps:
   a. to decrease the number of spikes?
   b. to reduce the neuropsychiatric symptoms, executive dysfunction, and behavioral changes, rendering him able to function independently in school and in many situations of everyday life.

Case Report

This patient with rolandic epilepsy, E.Z. age 9.3. suffered from neuropsychiatric symptoms, cognitive dysfunction, especially attention deficits[1], and behavioral changes, rendering him unable to function effectively in school and in many situations of everyday life.

In the initial descriptions of the syndrome, his mother and teachers observed behavioral and learning disabilities which caused serious school problems. At the age of 7.5. he developed severe handwriting problem. It was diagnosed as acquired isolated graphomotor deficit, as an example of selective "epileptic" developmental deficit (Figure 1).

At the age of 7.6. the first seizure was observed, and later active epilepsy (mainly in sleep), which was slowing the normal process of his development. At the age of 9.9. the epilepsy was worsening. The seizures were simple partial motor and sensory seizures involving the lower face and the pharyngeal region, the so-called "sylvian" seizures, and tended to occur during sleep (after falling asleep or before arousal in the morning) sometime with extension to the hemibody or with generalization. Such seizures sometimes started as

1. The mechanisms underlying attention deficits are still unknown and appear to be different between focal and generalized epilepsy [13].

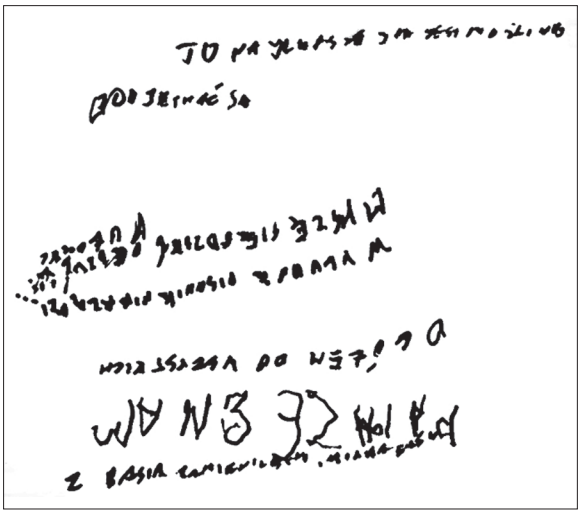

Figure 1. Writing sample: acquired isolated graphomotor deficit in E.Z. at the age of 7.5 as an example of selective "epileptic" developmental deficit.

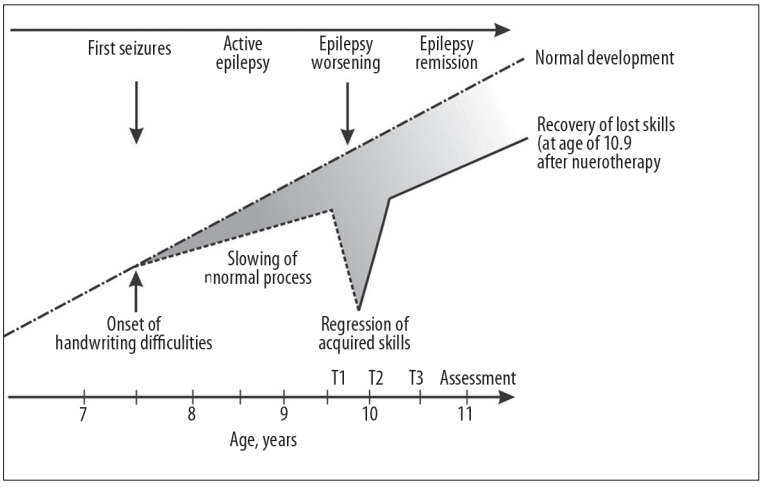

Figure 2. Acquired isolated graphomotor deficit in E.Z. at the age of 7.5 as an example of selective "epileptic" developmental deficit.

the child was waking up in the morning. There was a feeling of tingling (like pins and needles) on one side of the mouth involving the tongue, lips, gum and inner side of the cheek. Sometimes the seizure also involved the throat which may have caused speech to be unclear and therefore difficult to understand. The child made strange throaty or gurgling noises from time to time, and it was often this which alerted the parents to the fact that something may be wrong. He often knew what he wanted to say but he was not able to speak properly.

Although he was treated for epilepsy (carbamazepine), his condition was worsening, and a dramatic regression of acquired skills occurred (Figure 2). However, all this could be explained by the psychological consequences of the disease [11–14].

In brain MRI, frFSET2 sequence, coronal plane dysgenesis of the fornix was found, along with asymmetry of the lateral ventricles, more prominent on the left. In the axial plane, there were subarachnoid cysts in the posterior fossa. In 3DFSPGRT1 sequence, after contrast, in the axial plane there was local atrophy of the brain parenchyma in the right frontal area parasagittal. In frFSET2 sequence, coronal plane, local atrophy of the brain parenchyma was found in the right frontal area parasagittal (Figure 3A–D).

The patient took part in the neurotherapy program which included 20 sessions of relative beta training; the goal of the training was to activate the frontal cortex by enhancing the beta activity recorded over the frontal electrodes. In more detail the procedure was as follows: electrodes were placed at Fz and Cz – bipolar recording. The procedure was to increase the ratio of beta EEG power/EG power in the theta and alpha frequency bands. The beta frequency band was from 13 to 21 Hz. The combined theta and alpha frequency bands were from 4 to 12 Hz. Each session included approximately 20 min of neurofeedback training [11]. The entire program was administered by the same therapist team, simultaneously. We used neuropsychological testing as well as ERPs before the experiment, as well as after the completion of the program. The basic clinical background is provided in Table 1.

The experiment was reviewed and approved by the respective medical ethics committees, and the parents gave written informed consent for the anonymous publication of his case history.

The patient underwent standardized neuropsychological testing: electroencephalogram recording, Wechsler Intelligence Scale for Children-revised, Wechsler Memory Scale-III Polish version (WMS-III, Polish version), Peabody Picture Vocabulary Test-III (PPVT-III) and Boston Naming Test, Polish version (BNT-vPl), at baseline during active disease (Exam 1) and at follow-up after the completion of the neurotherapy program, during which recovery from epilepsy occurred (Exam 2).

Neuropsychological testing at baseline (Exam 1) showed multiple deficits (see Table 1). At follow up, after conclusion of the neurotherapy program (Exam 2), patient E.Z. showed improvements in neuropsychological functioning. His verbal and non-verbal IQ, and most of his cognitive functions increased significantly, including immediate and delayed logical and visual recall on the WMS-III (cf. Table 1).

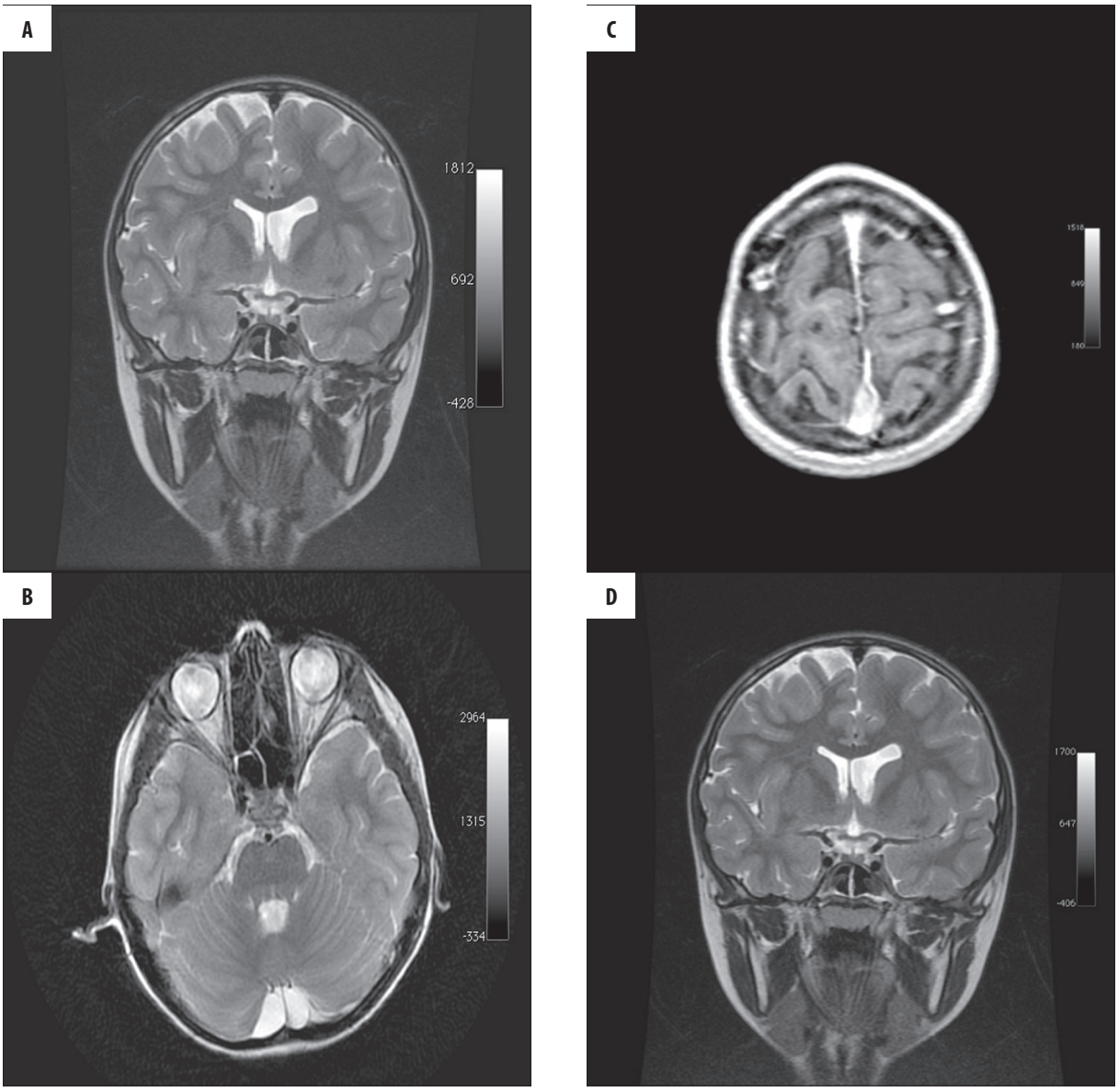

Figure 3. EZ (9;9) with benign partial epilepsy with Rolandic Spikes (BPERS) and the diagnosis of ADHD. (**A**) Brain MRI, frFSET2 sequence, coronal plane. Fornix dysgenesis, lateral ventricles asymetry – left more prominent. (**B**) Brain MRI, frFSET2 sequence, axial plane. Subarachnoid cysts in posterior fossa. (**C**) Brain MRI, 3DFSPGRT1 sequence post contrast medium injection, axial plane. Local atrophy of the brain parenchyma in the right frontal area parasagital. (**D**) Brain MRI, frFSET2 sequence, coronal plane. Local atrophy of the brain parenchyma in the right frontal area parasagital.

His results for maintaining attention on the WMS-III also improved (34/40 points). In other cognitive functions E.Z's results also improved in the 2nd examination. On the auditory learning task, he had forgotten all the words after a 15-minute filled delay in the 1st examination, and achieved 5 words in recognition; however, in the 2nd examination he remembered 2 words after the delay, and achieved all the words in recognition. This general pattern was repeated in nearly all the neuropsychological parameters.

Neuropsychological testing at baseline (Exam 1) showed also executive dysfunction in drawing of Semantic Figure (Figure 4). Patient E.Z. was not able to copy the semantic figure (Figure 4A), as he worked very fast and then abandoned the task. However, at follow up, after completion of the neurotherapy program (Exam 2), the child showed major improvement in executive functions (Figure 4C), even though these were the most disturbed of all his neuropsychological functions.

Event related potentials (ERPs) were used to assess the functional changes in the patient induced by rehabilitation programs. We used this approach for the following reasons. First, ERPs have a superior temporal resolution (on the order of milliseconds) as compared to other imaging methods, such as fMRI and PET (which have time resolution of 6 seconds and more) [12], Secondly, ERPs have been proven to be a powerful tool for detecting changes induced by neurofeedback training in ADHD children [12,13]. And finally, in contrast to spontaneous EEG oscillations, ERPs reflect the stages of information flow within the brain [14,15].

Table 1. Neuropsychological testing of the patient E.Z. in examination 1, and 2.

Measure	Exam. 1	Exam. 2
WAIS-R		
IQ – full	62.5/100	93.5/100
IQ – verbal	63.5/100	98.5/100
IQ – nonverbal	58.5/100	87.5/100
Attention		
WMS-III spatial span	3 (1st%)	12 (75th%)
Visuospatial ability		
WAIS-III block design	3 (1st%)	8 (25th%)
Logical memory		
WMS-III immediate logical memory	11/24	18/24
WMS-III delayed logical memory	9/24	19/24
WMS-III immediate visual recall	12/41	36/41
WMS-III delayed visual recall	6/41	25/41
Verbal memory		
CVLT short delay free recall	0/9 (<1st%)	2/9 (<1st%)
CVLT long free recall	0/9 (<1st%)	2/9 (<1st%)
CVLT long delay cue recall	0/9 (<1st%)	2/9 (<1st%)
Executive functions		
TMT– number sequencing	150s. (<1st%)	54s. (10th%)
TMT– number letter sequencing	Discontinued	150s. (<1st%)
Stroop		
Color	90 s. (<1st%)	41 s. (16th%)
Word	29 s. (25th%)	42 s. (63rd%)
Interferences	Discontinued	128 s. (<1th%)
WCST		
Categories	0 (2–5th%)	2 (>16th%)
Perseverative errors	46 (<1th%)	19 (37th%)
Conceptual level responses	63 (<19th%)	48 (45th%)
Fail to maintain sets	Discontinued	4 (2–5th%)

TMT – trial making test. Level of performance corresponding to the percentiles 98–99% – very superior; 91–97 % – superior; 75–90% – high average; 25–74% – average; 9–24% – low average, 3–8% – borderline; 2nd% ; below – impaired.

The diagnostic power of ERPs has been enhanced by the recent emergence of new methods of analysis, such as Independent Component Analysis (ICA) and Low Resolution Electromagnetic Tomography (LORETA) [13,14].

A modification of the visual two-stimulus GO/NO GO paradigm was used (Figure 5). Three categories of visual stimuli were selected:
1. 20 different images of animals, referred to later as "A";
2. 20 different images of plants, referred to as "P";
3. 20 different images of people of different professions, presented along with an artificial "novel" sound, referred to as "H+Sound".

All visual stimuli were selected to have a similar size and luminosity. The randomly varying novel sounds consisted of five 20-ms fragments filled with tones of different frequencies (500, 1000, 1500, 2000, and 2500 Hz). Each time a new combination of tones was used, while the novel sounds appeared unexpectedly (the probability of appearance was 12.5%).

The trials consisted of presentations of paired stimuli with inter-stimulus intervals of 1 s. The duration of stimuli was 100 ms. Four categories of trials were used (see Figure 2): A-A, A-P, P-P, and P-(H+Sound). The trials were grouped into four blocks with one hundred trials each. In each block a

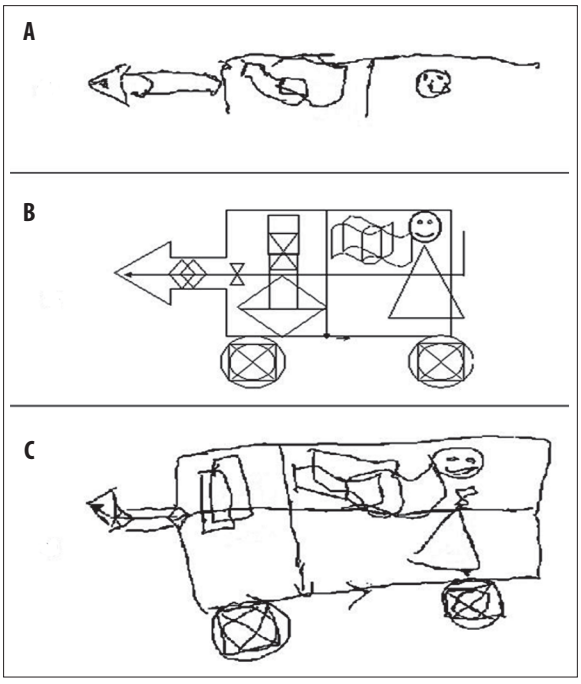

Figure 4. Drawing of Semantic Figure by EZ (9.3 years old) with benign partial epilepsy with Rolandic Spikes (BPERS) and the diagnosis of ADHD. (**A**). in the 1st examination (before the treatment). (**B**) The pattern for drawingc) in the 2nd examination (after the treatment).

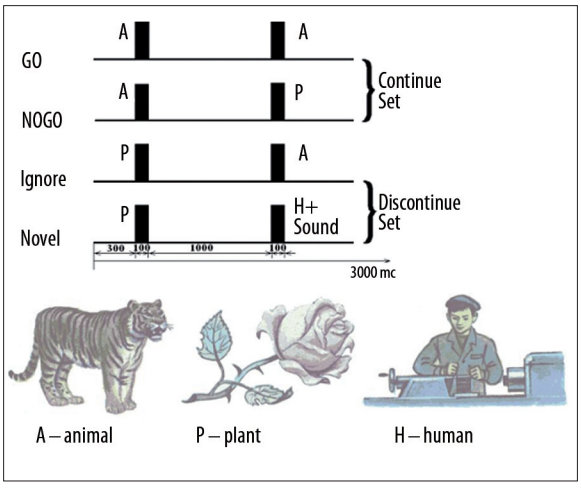

Figure 5. Schematic representation of the two stimulus GO/NOGO task. From top to bottom: time dynamics of stimuli in four categories of trials. Abbreviations: A, P, H stimuli are "Animals", "Plants" and "Humans" respectively. GO trials are when A-A stimuli require the subject to press a button. NOGO trials are A-P stimuli, which require suppression of a prepared action. GO and NOGO trials represent "Continue set", in which subjects have to prepare for action after the first stimulus presentation. Ignore trials are stimuli pairs beginning with a P, which require no preparation for action. Novel trials are pairs requiring no action, with presentation of a novel sound as the second stimuli. Ignore and Novel trials represent "Discontinue set", in which subjects do not need to prepare for action after the first stimulus presentation. Time intervals are depicted at the bottom.

unique set of five A, five P, and five H stimuli were selected. The child practiced the task before the recording started.

The child sat upright in an easy chair looking at a computer screen. The task was to press a button with the right hand in response to all A-A pairs as fast as possible, and to withhold button pressing in response to other pairs: A-P, P-P, P-(H+Sound) (Figure 5). According to the task design, two preparatory sets were distinguished: a "Continue set," in which A is presented as the first stimulus and the subject is presumed to prepare to respond; and a "Discontinue set," in which P is presented as the first stimulus, and the subject does not need to prepare to respond. In the "Continue set" A-A pairs will be referred to as "GO trials," A-P pairs as "NO GO trials." Averages for response latency and response variance across trials were calculated. Omission errors (failure to respond in GO trials) and commission errors (failure to suppress a response to NO GO trials) were also computed.

EEGs were recorded from 19 scalp sites. The electrodes were applied according to the International 10-20 system. The EEG was recorded referentially to linked ears, allowing computational re-referencing of the data.

The analysis consists of the following steps: 1) eye movement artifact correction and elimination: a) using a spatial filtration technique based on zeroing the activation curves of individual Independent Component Analysis (ICA) components corresponding to horizontal and vertical eye movements, as well as b) excluding epochs with an excessive amplitude of EEG and excessive faster and slower frequency activity; 2) Fast-Fourier Transformation (FFT) of the corrected EEG for extracting EEG power and coherence for all 0.25 Hz bins in the frequency band from 0.5 to 30 Hz; 3) computation of event related potentials by averaging EEG over trials for each category of trial and each channel with a time resolution of 4 ms; 4) decomposition of an individual ERPs into independent components by applying spatial filters extracted by means of the ICA from the collection of ERPs computed for the corresponding group of healthy subjects; 5) comparison of each extracted electrophysiological and behavioral variable against the corresponding variable computed for a carefully constructed and statistically controlled age-regressed, normative database in which the variables have been transformed and confirmed for their Gaussian distribution.

Visual inspection of raw EEG was made in order to search for paroxysmal patterns that pop out of the background EEG.

Besides the visual inspection, an automated spike detection was performed. The method of automated spike detection is based on the temporal parameters of spikes, and the spatial location of the corresponding spike dipole[2]. The amplitude-temporal parameters have been defined on the basis of comparison spike detection by the program and by experienced experts from a data base of more than 300 EEG recordings in epileptic patients; paroxysmal character, high

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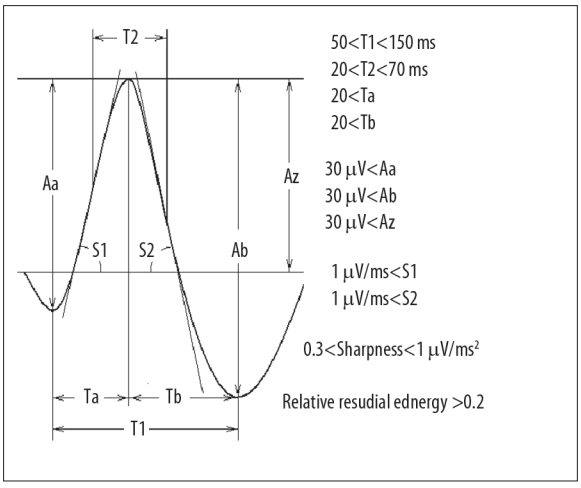

Figure 6. The amplitude-temporal characteristics used for automatic spike detection.

Behavioral data

The results of a comparison of the patient's behavioral parameters during the GO/NOGO task are presented in Table 2. The number of omission errors decreased by more than 50% after treatment, so that no deviation in behavior from norms was observed after treatment.

Spectra

Deviations from normality in the EEG spectra computed for 20 minutes of the GO/NOGO task before treatment are presented in Figure 8 left. As one can see the EEG pattern is characterized by excessive slow (around 6 Hz) activity over frontal-temporal areas. Quite large and statistically significant changes occurred after treatment. The excessive slow activity decreased after treatment (Figure 8, right).

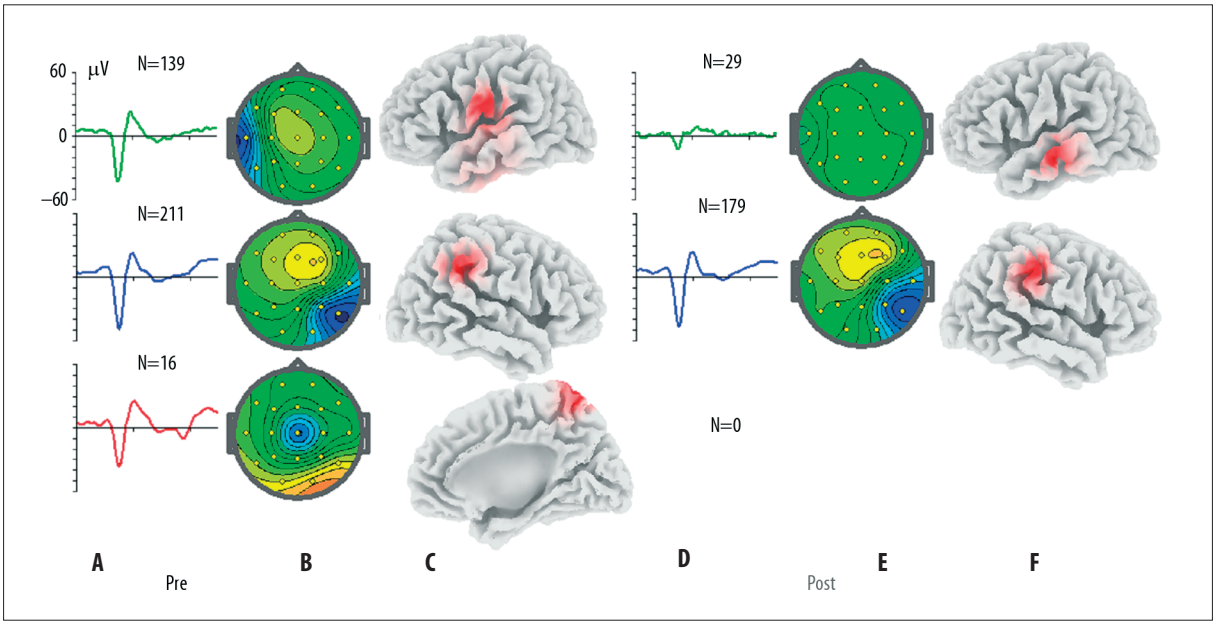

Figure 7. Averaged spikes detected in the EEG during the GO/NOGO task pre- and post-neurotherapeutic intervention. (**A**) Averaged spikes for electrodes T3, T6 and Cz before the treatment. (**B**) Maps taken at the peak of the spike before the treatment. (**C**) sLORETA images of spikes taken at the peak of the spike before the treatment. (**D**) Averaged spikes for electrodes T3, T6 after the treatment. No spikes at Cz were detected. (**E**) Maps taken at the peak of the spike after the treatment. (**F**) sLORETA images of spikes taken at the peak of the spike after the treatment. Numbers on the graphs – number of averaged spikes.

degree of sharpness, and short duration. These are its paroxysmal character, high degree of sharpness and short duration. These parameters are presented in Figure 6.

For this patient the automatic spike detection was performed on EEG in the common average montage for both eyes open and eyes closed conditions. The results of automatic spike detection in the fragment of EEG recorded during 22 min of the GO/NOGO task are presented in Figure 7.

As one can see from Figure 7. the number of spikes dramatically decreased after treatment, especially at the left temporal and central electrodes. Moreover, in EEG after treatment we were not able to detect any spikes at the Cz electrode.

Event related potentials

The results of ERP changes are presented in Figure 9. One can see dramatic changes of the NOGO ERPs with increase of the NOGO potential over the Cz electrode and decrease over the left temporal areas.

Discussion

Rolandic epilepsy, in which interictal epileptiform discharges appear, is associated with neuropsychological disorders such as cognitive impairment and behavioral problems, even in the absence of clinical epilepsy [16]. Uncontrolled reports and three preliminary randomised controlled trials

Table 2. Behavioral parameters in the GO/NOGO task.

	Omissions,%	Commissions,%	Reaction time, ms	Error of response ms
Pre	13.0	1.0	430	12.3
Post	6.0	3.0	438	14.3
Norm	6.3	2.6	489	13.2

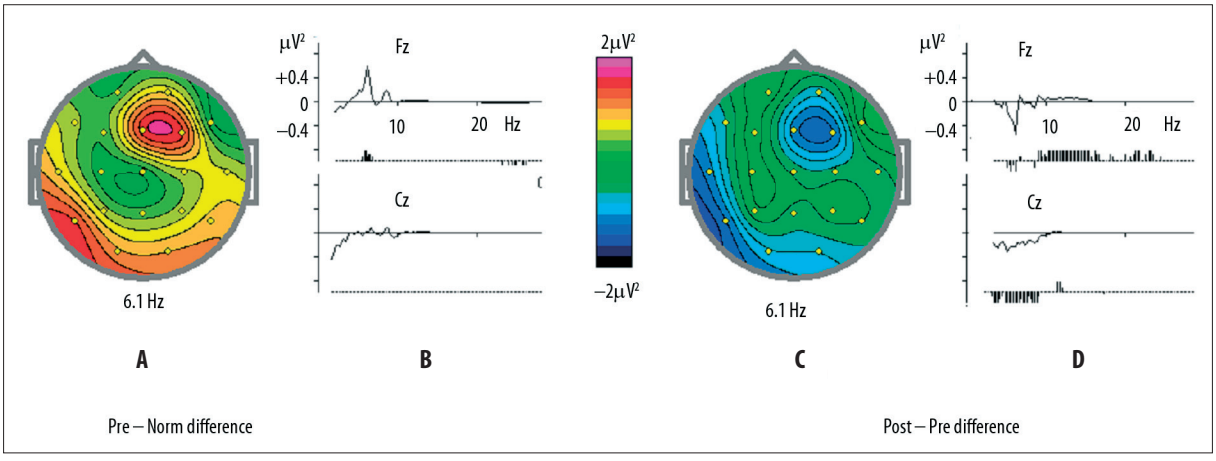

Figure 8. Differences of EEG spectra in the GO/NOGO task. Left – difference between the EEG spectra in pre-treatment condition and the average norm. (**A**) Map of the difference taken at 6.1 Hz. (**B**) The spectra difference with confidence levels below the graph (small vertical bar – $p<0.05$, medium vertical bar – $p<0.01$, large vertical bar – $p<0.001$). Right difference between EEG spectra in post and pre treatment. (**C**) Map of the difference taken at 6.1 Hz. (**D**) The spectra difference with confidence levels below (small vertical bar – $p<0.05$, medium vertical bar – $p<0.01$, large vertical bar – $p<0.001$).

of the antiepileptic treatment of interictal epileptiform discharges have suggested that suppression of discharges is associated with a significant improvement in psychosocial function. However, a greater number of controlled studies needs to be carried out in order to confirm this hypothesis [10–12].

The etiology is multifactorial, being affected by the type of epileptic syndrome, the cause of epilepsy, the high frequency of epileptic seizures, a previous history of status epilepticus, the age at the onset of epilepsy, the antiepileptic treatment selected, and the role of interictal epileptiform discharges [1]. Several studies have sought to analyze to what extent cognitive impairment can be attributed to interictal epileptiform discharges among the other epilepsy factors [12].

The disruptive effect of interictal epileptiform discharges on cognition is supported by a wide range of factors, such as the concept of transient cognitive impairment, the definition of epileptic encephalopathy, the natural course of epileptic syndromes with continuous spike and wave activity during slow sleep, the concept of autistic regression related to epileptiform activity, the cognitive profile of benign rolandic epilepsy, and the cognitive impact of non convulsive status epilepticus. According to this information it has been suggested that the treatment of interictal epileptiform discharges with antiepileptic drugs could improve cognition and behaviour in these children [1].

Convergent clinical data and new evidence drawn from electrophysiological studies and functional imaging [11] suggest that the cognitive and behavioral dysfunction is directly related to the particular role that the affected cortical area plays when the epileptic process becomes active. The mechanisms, however, are not well understood [16–18]. The Continuous Spike-Waves during Slow Wave Sleep [CSWS] phenomenon is probably due to a disturbance of the corticothalamic oscillatory mechanisms at work during slow sleep, which seem to play a role in the consolidation of material acquired during waking [19–21].

One especially dramatic example of how partial epilepsy can lead to a progressive dementia and/or a massive behavioral regression in children is the "acquired epileptic frontal syndrome" [21].

Cases of partial epilepsy with CSWS are increasingly described in a variety of developmental and acquired focal cortical pathologies in children (also in association with thalamic lesions) [22–26].

However, as in our case, acquired focal cortical pathologies may be not in association with thalamic lesions [27], however, the child may have an increased frequency of rolandic spikes as in other ADHD children, as was confirmed by Kropotov [13], and have associated neuropsychiatric deficits resembling the symptoms of attention deficit-hyperactivity disorder (ADHD), the most common neurobehavioral disorder of childhood [7,9,28,29].

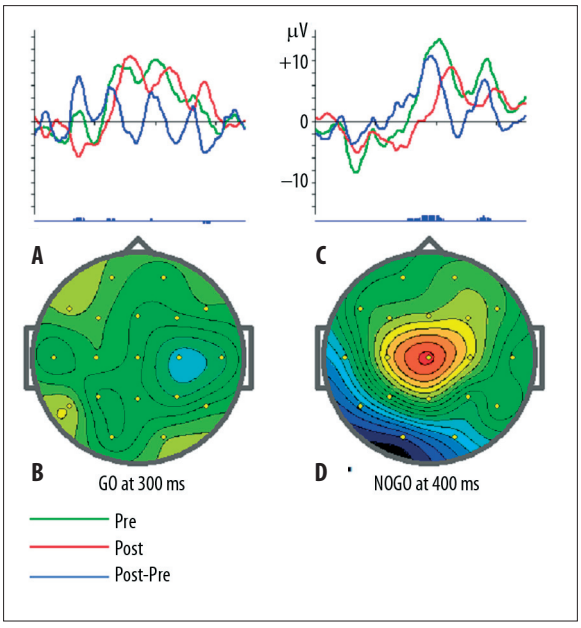

Figure 9. The ERPs and ERP difference waves (post-pre) in the GO/NOGO task. (**A**) ERP for GO condition computed at Pz for pre (red line) and post (green line) conditions and ERP difference wave Post-Pre (blue line) with the confidence levels of the difference below (small vertical bar – $p<0.05$, medium vertical bar – $p<0.01$, large vertical bar – $p<0.001$). (**B**) The map of the difference wave taken at 300 ms (peak latency of the GO P3 wave). (**C**) ERP for NOGO condition computed at Cz for pre (red) and post (green) conditions and ERP difference wave (Post-Pre) with the confidence levels of the difference below (small vertical bar – $p<0.05$, medium vertical bar – $p<0.01$, large vertical bar – $p<0.001$). (**D**) The map of the difference wave taken at 400 ms (peak latency of the NOGO P3 wave).

Graphomotor deficit is not indicated to be criterial for ADHD, though it occurs almost commonly [3]. However, it is still not clear whether there lie at its basis motor activity problems or rather the planning and organisation of behaviour. Long term observation and the carrying out of neuropsychological tests on the boy E.Z. point rather to a connection of the disturbances with all these factors cumulatively. Not without significance is the fact that the lack of allocation of brain resources in the case of performing the most difficult tasks is linked with the occurrence of epileptic brain activity.

Traditional therapies for the recovery of a child with benign partial epilepsy with Rolandic Spikes (BPERS), associated with neuropsychiatric deficits resembling symptoms of attention deficit-hyperactivity disorder (ADHD), are still not satisfactory [11,13]. To date the best approach seems to be cognitive therapy and behavioral training [13], however, the results are limited [11]. Adjunct interventions that can augment the response of the brain to the behavioral and cognitive training might be useful to enhance therapy-induced recovery in patients with rolandic epilepsy. In this context, neurofeedback self-regulation appears to be an additional intervention to standard neuropsychological therapies.

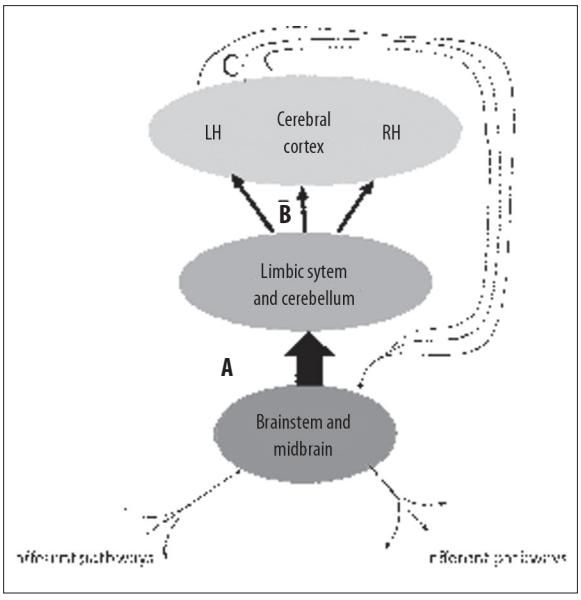

Figure 10. Schematic diagram of the pathways and brain regions involved in behavior [11].

Why did we not achieve full recovery?

BPERS is a model for the study of the cognitive manifestations of focal epileptic discharges in a developing brain, although the prolonged fluctuating and cognitive manifestations and their dynamics of onset and recovery cannot be explained in terms of simple ictal-postictal symptoms which suggest that several different mechanisms are probably involved.

Therefore, how to interpret our results?

Microgenetic theory [30] makes it possible to interpret the results we have achieved. This theory differs from other theories of brain function in that it emphasizes:

- process and change, rather than data processors connected to each other by neural "cables," as though the brain were a computer;
- the creative nature of perception, which is not just a passive collection of stimuli, but a process of creating an image of reality;
- understanding the symptom as a segment of normal behavior that is revealed prematurely by pathology, and is not just a deficit, that is, the absence of the correct behavior;
- the development of mental processes that evolve on different scales of time, assuming that the laws of behavior are the laws of evolution expressed on another temporal plane;
- processing of information from whole to part, and not, as in standard theory, from "bits" to "stacks" of information.

In particular, a fuller understanding of the essence of ADHD brings us closer to grasping the process of symptom formation.

In Figure 10, the afferent pathways bring impulses received from the sense organs to the brainstem, which constitutes the oldest and most primitive part of the brain. The brainstem reacts with a general activation of the organism, which

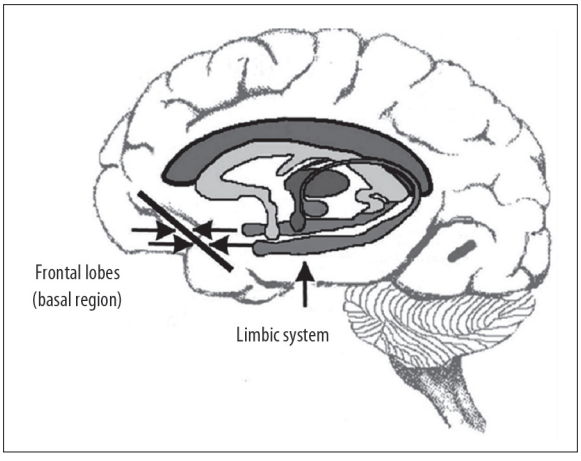

Figure 11. The bidirectional transition from emotion to mentation and action [11].

is transmitted upwards (A), to the limbic system and cerebellum. From these somewhat younger structures the activation signal becomes more complex (B), so that in the cortex it spreads to highly specialized areas. Signals from the cortex then travel by pyramidal pathways back down to the brainstem, and from there by efferent pathways to effectors in the musculoskeletal system.

Understanding this somewhat simplified diagram of activation makes it easier to interpret the mosaic of diverse disturbances that occur in children with AD/HD, associated with disturbances that are both structural (involving different areas and different levels of the brain) and functional (resulting from changes on the level of neurotransmission), as well as the resolution of these disturbances in the course of rehabilitation.

The process of symptom formation responsible for the heterogeneity and changeability of behavioral disturbances in ADHD children is explained by Figure 11, which illustrates the bidirectional transition from emotion to mentation and action. The state of arousal in the mind, which in a healthy brain can be reinforced or inhibited by the executive functions, cannot be controlled in the brains of ADHD children. Thus the behavior which these children exhibit can be diverse, variable, and capricious, depending on a whole range of factors both structural and functional in nature.

Conclusions

1. Deficits of cognitive functions characteristic for ADHD are detected in a child with benign partial epilepsy with Rolandic Spikes (BPERS).
2. The attentional dysfunctions are independent of the effects of antiepileptic treatment.
3. The parameters of attention were improved after the neurotherapy program intended to activate the frontal lobes.
4. The improvement in attention induced by the neurotherapy program was accompanied by a decrease in the number of spikes and an increase of the P3NOGO component of event related potentials in the GO/NOGO task.
5. Event related potentials can be used to assess functional brain changes induced by neurotherapeutical programs.

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