AN EVALUATION OF PERSONALIZED NEUROFEEDBACK TRAINING FOR ROLANDIC EPILEPSY: A CASE STUDY

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SUMMARY

Background: The aim of this study was to determine whether relative beta neurofeedback training (RBNT), applied to regulate brain dynamics, would be useful for a patient with benign partial epilepsy with Rolandic Spikes (BPERS), accompanied by symptoms of attention deficit-hyperactivity disorder (ADHD).

Case study: The patient, AG, age 8:2, presented with neuropsychiatric symptoms, cognitive dysfunction (especially attention deficits), and behavioral disorders, rendering her unable to function independently in many home, preschool and school situations. She was being treated for epilepsy, without apparent progress. Forty sessions of RBNT were applied to regulate the dynamics of frontal lobe functioning. In standardized neuropsychological testing and ERPs before and after the neurotherapy program, there were multiple cognitive and neurobehavioral deficits at baseline, along with Rolandic spikes. After RBNT, there was a statistically significant reduction of slow activity frontally and a considerable lowering of the theta:beta ratio. These changes were accompanied by decreased reaction time and fewer omission errors in the cued GO/NOGO task. The post-training changes in EEG spectra were also accompanied by complete cessation of Rolandic spikes in resting EEG. AG's verbal and non-verbal IQ also increased significantly, and her cognitive functions improved, including immediate and delayed logical and visual recall on the WMS-III, attention on the WMS-III, and executive functions.

Conclusions: RBNT was effective in the treatment of this patient with BPERS, accompanied by ADHD symptoms. ERPs in a GO/NOGO task can be used to assess functional brain changes induced by neurotherapeutic programs.

Key words: ERPs, ADHD, attention problems, graphomotor deficit, behavioral problems

INTRODUCTION

Many different kinds of epileptic syndromes are most common in childhood. Among these is Rolandic epilepsy (RE), which is more and more frequently diagnosed by pediatricians and pediatric neurologists [1,2,3,4,5].

According to the 2006 classification published by the International League Against Epilepsy (ILAE), RE is a developmental epilepsy. The complex genetic background of this particular type of epilepsy has not yet been fully explained [4,6,7,8]. The presence of centrotemporal spikes (CTS) in the EEG is pathognomonic [4,5,7,8].

RE is also designated as an idiopathic epilepsy syndrome. It has been described in the literature as "benign Rolandic epilepsy in children" (BREC) or "benign epilepsy with centrotemporal spikes" (BECTS) [9,10]. The term "Rolandic" stems from the fact that the seizures begin in the Rolandic region of the brain. These are classified as "partial" seizures, since only this one part of the brain is involved [11,12,13].

RE usually manifests between the ages of 3 and 10 years, and often resolves in adolescence (14-18 years) [14,15]. It is considered a "benign" epilepsy precisely because the prognosis is positive: nearly all children with RE grow out of it as they mature. It occurs in almost one out of every five children with epilepsy, which means that it is one of the most common types of childhood epilepsy. Boys and girls are equally affected.

Some children with RE function relatively well and display no learning difficulties, though some may have special difficulties with reading and/or writing, or with drawing and other visuospatial skills [8,10,16,17,18]. The great majority of children with RE, however, display symptoms similar to Attention Deficit Hyperactivity Disorder (ADHD) [4,5,18,19]. These symptoms may be associated with a neuropsychiatric syndrome [9], though there has been relatively little attention given in the literature to this co-occurence of syndromes [5,20].

There have been reports of a close connection between benign partial epilepsy with Rolandic spikes (BPERS) and acquired epileptic aphasia (Landau-Kleffner syndrome), which was the first example of a primarily "cognitive" epilepsy in children [8,18]. Later, some children were diagnosed with persistent but reversible oral-motor deficits during the active phase of RE [18].

With further research, it has become apparent that this type of epilepsy can cause long-term "epileptic" deficits [20]. There has been some neuropsychological research supported clinical observations that children with BPERS display normal intelligence, but a larger percentage have attention disorder and other deficits (linguistic, visuospatial, etc.) when compared to a control group [4,5,18, 20]. In the last decade or so, research on transient cognitive and behavioral disturbances occurring in children with BPERS has suggested that these symptoms are directly related to the epilepsy [18,20,21]. Though studies of this kind have been infrequent, some longitudinal neuropsychological research has recently indicated that acquired transient cognitive and behavioral symptoms correlate in

some children with epileptic activity in the EEG [4,5,8,18,20], which may explain some of the learning and school-related problems experienced by these children when they are in the active phase of the syndrome [5]. Many of these studies have found deviations from the norm in categories similar to those of the very familiar pathophysiological patterns in the population of children with ADHD, such as an increased theta-beta ratio [18,22], a decrease in the P3b component [4,14,15,18], and a decrease in the P3 component in a NOGO task [4,20,21]. Quantitative analysis has also been used to analyze the ERP data without any a priori decisions about peak performance, amplitude, or latency. It turned out that the frequency of Rolandic spikes in children with ADHD is much higher than epidemiological research would predict [23]. ERPs have also shown much greater amplitudes in persons with epilepsy in the frontal and central regions during the temporal window between 250 and 425 ms after stimulus, which corresponds with the temporal window of differentiation between target and non-target stimuli [8,10,13].

Yet another question raised by researchers is the efficacy of therapy for children with RE. Promising results have been obtained by applying carefully chosen neurotherapy programs. Our previous research has shown that "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" [5]. Relative beta neurofeedback training (RBNT), applied to regulate the dynamics of frontal lobe cortical function by increasing beta waves, has shown promise in reducing epileptic spikes around the Rolandic fissure [24].

The next question involves the connection, if any, between the symptoms of ADHD and the presence of Rolandic spikes in this subgroup of the ADHD population, and how these symptoms could be treated using RBNT. In what follows, a case that addresses this question will be presented.

1. The purpose of the present study was to determine:
   - whether or not a child with BPERS, accompanied by symptoms of attention deficit-hyperactivity disorder (ADHD), presents with:
   - a specific developmental deficit in learning or, more generally, disturbances of cognitive and executive functions;
   - variant patterns of cortical activation while performing operations in working memory in comparison to children without epileptic seizures;

2. whether or not RBNT, applied to regulate the dynamics of frontal lobe cortex function by increasing beta waves, registered with an electrode placed in the frontal region, can help to:
   - alleviate the disturbances of cognitive and executive functions;
   - reduce the number of spikes
   - alleviate the syndrome of neuropsychiatric disturbances and cognitive and executive dysfunctions characteristic for ADHD;
   - improve the patient's independent functioning in school.

CASE HISTORY

Female patient AG was first examined when she was six-and-a-half years old. Neuropsychiatric symptoms had been occurring for some time, along with disturbances of cognitive and executive functions, especially attention deficits. This was making it difficult for her to function independently in daily life, as well as in the preschool setting (she changed preschools three times) and in school. Since AG's problems with learning new material were considerable, so that she was basically unable to learn anything, her teachers suggested that she should be transferred to a special school.

After neuropsychiatric and neuropsychological consultations, AG was diagnosed with ADHD, according to DSM-IV-TR diagnostic criteria, and she initially received neuropsychological therapy for her attention problems. In her mother's opinion, these disturbances could be attributed to an intensification of the symptoms of a particularly troublesome neuropsychiatric syndrome diagnosed by a psychiatrist, which had persisted since her first seizure at the age of 3. The symptoms consisted primarily in behavioral disturbances, such as impulsivity and aggressiveness, along with defiance (i.e. refusal to follow parental instruction).

The symptoms began to intensify, with stronger nocturnal "episodes" [5], until the development of active epilepsy, which slowed the normal course of AG's development. The seizures were simple, partial, motor and sensory, and involved the lower part of the face and the throat. Tremors most often occurred during sleep (immediately before falling asleep and just before waking up in the morning), and involved mostly an upper or lower limb, sometimes the entire half of the body. The seizures sometimes began when AG awoke at night or in the morning; she would experience a prickling sensation (something like needles) at one corner of her mouth, involving also the tongue, the lips, and the inside of her cheek. There were also automatisms, consisting in lateral movements of the jaw. She usually cried out or wept, and could not be comforted. At times the seizures involved the throat as well, causing slurred and nearly incomprehensible speech. She would produce odd throaty, gurgling sounds, and in this way she often alarmed her parents that something bad was happening. She usually knew what she wanted to say at such times, but could not pronounce the words properly.

AG was also observed to have increasing and serious problems with drawing and writing. The writing disorders were diagnosed as an acquired, isolated graphomotor deficit, an example of a selective "epileptic" developmental deficit (see Fig. 1A).

Despite antiseizure mediation with carbamazepine (Tegretol), her condition worsened, and she exhibited a dramatic loss of skills already acquired (see Fig. 2). This situation could be explained, however, in terms of the psychological and neuropsychological sequelae of the epilepsy [23,24,25,26].
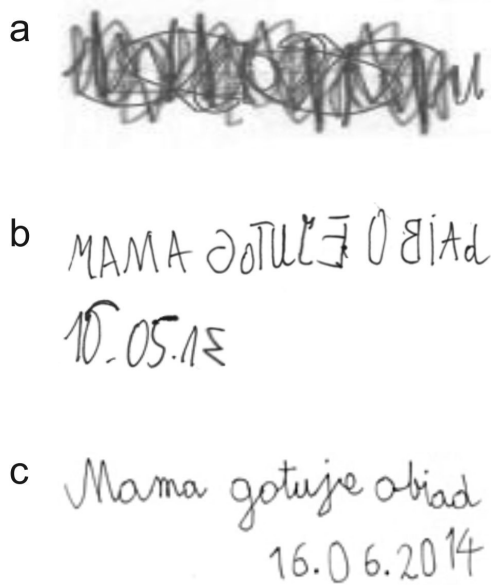
Fig. 1. Writing samples from AG, a female patient diagnosed with RE, at the age of 6.5 years. In B and C, the patient was asked to write from dictation the Polish sentence "Mama gotuje obiad," which means "Mom is cooking dinner," and then to write the date beneath. [Please note that the European convention is used: DD/MM/YY, not the American MM/DD/YY]: a). writing sample at the age of 6:5, indecipherable scribble; b). at baseline, age 7:1: acquired, isolated graphomotor deficit, an example of selective "epileptic" development disorders; mixing of upper and lower case letters, rotation; c). at followup (after 40 sessions of neurotherapy), age 8:2: correct writing. Date of examination corrected by AG spontaneously: initially, she said the wrong date aloud (April 23), but quickly corrected to the right date, August 3
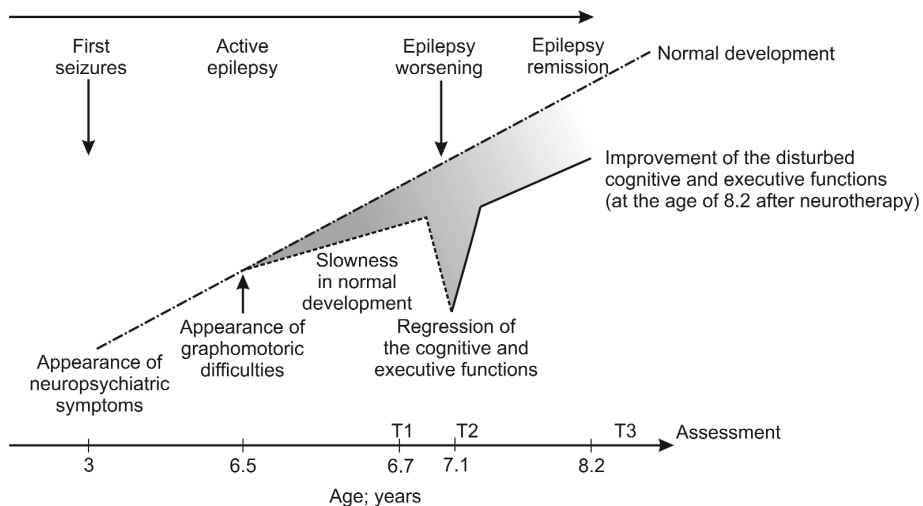

Fig. 2. Acquired, isolated graphomotor deficit in AG at the age of 6:5, an example of selective "epileptic" development problems. Retardation of the process of development. Cognitive and executive dysfunctions at age 7:1. Significant recovery of disturbed cognitive and exectuve functions at age 8:2. From: [5], with modifications

THE NEUROTHERAPY PROGRAM

Based on the data given above, a neurotherapy program was administered to AG, involving 40 sessions of relative beta training, conducted by a neurofeedback therapist.

Electrodes were placed at the Fz and Cz points, for bipolar recording. The goal of the therapy was to increase the activization of the frontal cortex by increasing beta activity in the EEG in the combined theta and alpha frequency bands. The beta frequency band was in the range from 4 to 12 Hz, and the combined theta and alpha bands were in the same range. During the first four sessions, neurofeedback training was administered daily for 30 seconds. In subsequent sessions, the duration was gradually increased, as the patient's capacity increased. The last 20 sessions of neurofeedback lasted for about 20 minutes [26].

AG was evaluated twice, using a comprehensive diagnostic protocol (psychometric and neurometric). The baseline examination was performed in an active period of the epilepsy, before the application of neurotherapy, when the patient's age was 7:1, while the followup examination took place after 40 sessions of neurotherapy, at the age of 8:2.

In neuropsychological examination, standard neuropsychological batteries were used, including clinical observation, a structured interview, the Wechsler Memory Scale (WMS III, Polish version), the Wechsler Children's Intelligence Test (Polish version), the Wisconsin Card Sorting Test (WCST, Polish version for children), the Trail-Making Test (TMT) and Pachalska's Semantic Figure Test for children [26]. For the neurophysiological testing, we used EEG, quantitative electroencephalography (QEEG), and event-related potentials (ERPs). The patient's EEG and ERP spectra with inter-seizure Rolandic spikes were compared to a normative base (the HBI data base) in order to evaluate AG's neurophysiological deficits.

The experiment was consulted with and approved by the local ethics commitee. The parents gave written consent to the anonymous publication of AG's history.

RESULTS

Neuropsychological results

Table 1 shows the results of neuropsychological testing. The baseline examination showed disturbances of cognitive and executive functions. In the follow-up examination, AG showed considerable improvement in all tested neuropsychological functions. The greatest improvement was seen in the verbal and nonverbal IQ (WCIS), concentration (WMS III), visuospatial functions (WMS III), immediate and delayed logical memory (WMS III), and executive functions (WCST), where at baseline, as Table 1 shows, there were errors in every category (Table 1). Of particular interest is the disappearance of perseveration, which was no longer present in either drawing or writing (cf. Figs. 1 and 3).

Probably the most interesting result that AG achieved after neurotherapy was in the domain of memory capacity. In aural learning of verbal material, at baseline

Table 1. Results of neuropsychological testing at baseline and follow-up

| Instrument	Baseline	Follow-up
Wechsler Children's Intelligence Test (Polish version)		
I.Q. – global	62.5/100	93.5/100
I.Q. – verbal	63.5/100	98.5/100
I.Q. – non-verbal	58.5/100	87.5/100
Wechsler Memory Scale – III (Polish version)		
Attention subtest		
WMS-III Attention subtest	3 (1st%)	12 (75th%)
WMS-III Spatial subtest	3 (1st%)	12 (75th%)
WMS-III Visuospatial block subtest	3 (1st%)	8 (25th%)
Logical memory		
WMS-III logical memory(immediate)	11/24	18/24
WMS-III logical memory (30 min. delay)	9/24	19/24
WMS-III visual recall (immediate)	12/41	36/41
WMS-III visual recall (30 min. delay)	6/41	25/41
Memory storage capacity		
WMS-III list of words (after distraction)	2/12	12/12
WMS-III list of words (30 min. delay)	0/12	12/12
Executive functions		
TMT– sequences of numbers	150 sec. (<1st%)	54 sec. (10th%)
TMT– numbers and letters	interrupted	150 sec. (<1st%)
Stroop Test (**Polish version for children)**		
Colors	90 sec. (<1st%)	41 sec. (16th%)
Words	29 sek. (25th%)	42 sec. (63rd%)
Interference	interrupted	128 sec. (<1th%)
WCST (**Polish version for children)**		
Categories	2 (>16th%)	no errors
Perseverative errors	2 (>16th%)	no errors
Classification errors	48 (45th%)	no errors
Errors in changing category	interrupted	no errors
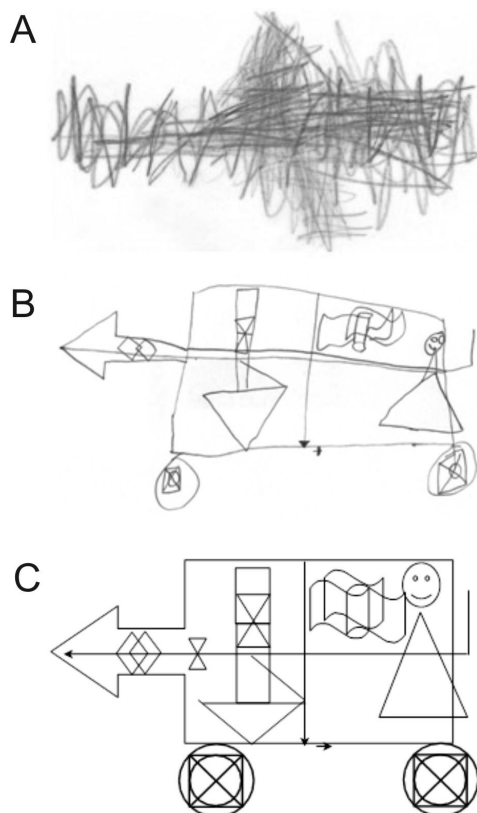
Fig. 3. Drawing of Semantic Figure by the female patient AG with benign partial epilepsy with Rolandic Spikes (BPERS) and the diagnosis of ADHD. a). The pattern for drawing; b). in the 1st examination (before neurotherapy); c) in the 2nd examination (after neurotherapy)

she remembered only 2 of 12 words after distraction, while after 30 minutes she had forgotten all of them. At follow-up, she remembered all 12 words, both after distraction and 30 minutes later. This is an above-average score compared to the Polish norm, which is 10.

Baseline testing also showed executive dysfunction in drawing a semantic figure (Fig. 3). At baseline, she was unable to copy the figure (Fig. 3A). She worked very fast but without precision, and quickly dropped the task. At follow-up, however, she was able to make a much better copy of the figure (Fig. 3C). Her writing (cf. Fig. 3C) and reading skills were also improved.

Her successes at school were particularly noteworthy. AG received the highest possible marks for a first-grader in all subjects, including conduct. The growth of her range of interests should also be emphasized, especially her excellent piano playing.

Neurophysiological analysis

Event Related Potentials (ERPs)

Event related potentials (ERPs) were used to assess functional changes manifested by the patient after rehabilitation. We used this approach for several reasons. First of all, 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 resolutions of 6 seconds and more) [4]. Secondly, ERPs have been proven to be a powerful tool for detecting changes induced by neurofeedback training in ADHD children [4,19,28,29]. And finally, in contrast to spontaneous EEG oscillations, ERPs reflect stages of information flow within the brain [4,5,18,19,23,24]. 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) [4].

A modification of the visual two-stimulus GO/NO GO paradigm was used (Fig. 4). Three categories of visual stimuli were selected:
- 20 different images of animals, referred to as "A";
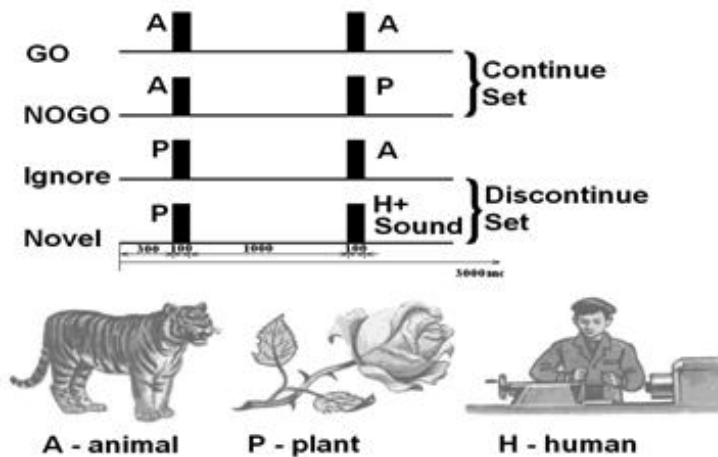- 20 different images of plants, referred to as "P";

Fig. 4. 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 occur 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 (A). 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

- 20 different images of people of different professions, presented along with an artificial "novel" sound, referred to as "H+Sound".

All visual stimuli were chosen so as 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 second. The duration of stimuli was 100 ms. Four categories of trials were used (see Fig. 3): 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 unique set of five A, five P, and five H stimuli were selected. The subject practiced the task before the recording started.

The patient sat upright in a comfortable 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 pressing the button in response to other pairs: A-P, P-P, P-(H+Sound) (Fig. 3). 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 (remontaging). The patient's performance is shown in Table 2).

The reaction time decreased by 80 ms and the number of omission errors dropped twice. The ERPs did not change. The most dramatic changes were observed in clinical EEG and EEG spectra (Fig. 5)

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

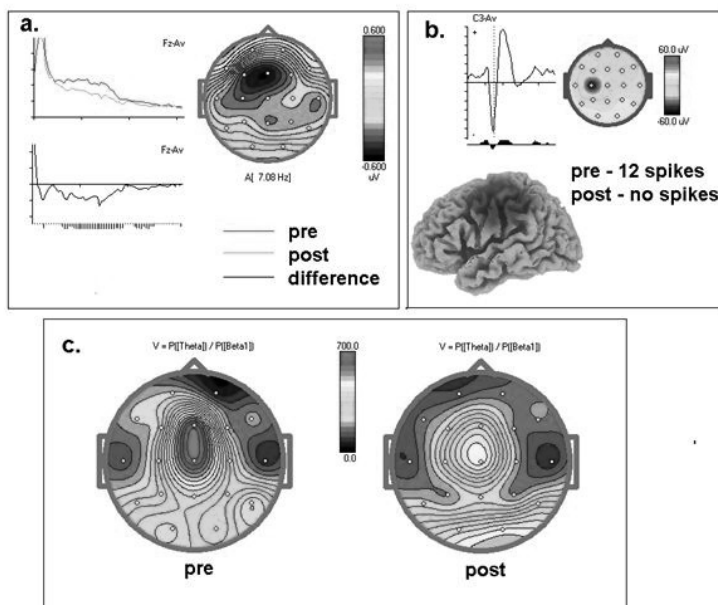|	Omission	Commission	RT1	var(RT1)
P value pre	0.15	0.61	0.61	0.25
Patient pre	16%	0%	523ms	17.3ms
Patient post	8%	2%	443ms	18.0 ms
Norms	6.3%	2.6%	480ms	13.2ms

Fig. 5. Clinical EEG/EEG spectra changes after neurofeedback treatment. a). EEG spectra diffe-rences. Red – pre, green – post, black – difference. b). Changes in spike detection. Pre – 12 Ro-landic spikes were detected during 6 minutes of resting state conditions. Post – no spikes were detected. c). Reduction in the theta-beta ratio after neurofeedback

DISCUSSION

Our study has shown that AG was displaying a specific deficit related to the epilepsy, and not a generalized dysfunction, since the cognitive symptoms re-solved along with the disappearance of Rolandic spikes. We did observe variant patterns of cortical activation during the performance of operations in working memory, as compared to children without epilepsy.

Rolandic spikes appear in the EEG of children with ADHD at a significantly higher rate than would be expected from epidemiological studies [27, 28]. The question of how ADHD symptoms are related to Rolandic spikes in this ADHD subgroup remains to be answered. On the other hand, many previous EEG stud-ies have reported an elevated theta-beta ratio in ADHD [29,14,30]. In the present study, we used the theta-beta ratio as a neurophysiological index of ADHD, and the number of Rolandic spikes as an index of the neurological status of the brain. We have shown that the QEEG based neurofeedback sessions in this patient, intended to increase the frontal beta activity, resulted in the disappearance of Rolandic spikes and a statistically significant decrease of frontal theta activity, accompanied by a substantial lowering of the theta-beta ratio.

The first application of EEG based neurofeedback for the treatment of epilepsy was done by Sterman and colleagues (for a review of these studies see

[32]). In these studies, a standard SMR (sensory-motor rhythm) training was used at one or more central sites (C3, Cz and C4).

Although 82% of patients demonstrated more than a 30% reduction in seizures, complete cessation of seizures has been rare. Walker and Kozlowski [33] were the first to report on a study of applying QEEG as a guide to neurofeedback training for people with untreatable seizures. The QEEG findings included one or more focal slow abnormalities and one or more coherence abnormalities. The neurofeedback included rewarding inhibition of slow activity in the areas with excessive slow activity (1–10 Hz) and rewarding an increase in 15–18 Hz at the same time. Within 20–35 sessions,all the studied patients became seizure-free and remained so for an average of 7 years.

In the present study we followed this strategy. The19-channel EEG of the patient was recorded in the resting state with eyes open and eyes closed, the EEG spectra were computed and compared with the HBI reference data base. We found increased slow activity frontally in comparison to healthy controls, which resulted in an elevated theta-beta ratio at the Fz and Cz electrodes. Relative beta training [23] was suggested as a neurofeedback protocol.

The implementation of a program of neurofeedback in this patient resulted in a statistically significant reduction of slow activity frontally and substantial lowering of the theta-beta ratio. These changes were accompanied by decreased reaction time and fewer omission errors in the cued GO/NOGO task. The post-training changes in EEG spectra were also accompanied by complete cessation of Rolandic spikes in the resting state EEG.

The present study thus supports the effectiveness of RBNT for mitigating the symptoms of both ADHD and epilepsy. In our opinion, however, the question about the possibility that Rolandic spikes (or rather the neurological abnormalities that lead to appearance of these spikes in EEG) are actually the cause of ADHD in this patient still remains to be answered, despite the therapeutic success.

How should we interpret the spectacular improvement achieved in this case, especially as pertains to memory capacity, which is of fundamental importance for learning?

In microgenetic theory, as in other theories of working memory, the primary challenge is to solve two basic problems:

1. How long does information remain in working memory before it is either transferred to long-term memory to be available for later recall, or forgotten?
2. How much information can be processed simultaneously in working memory?

The history of various efforts to answer these questions, based on theoretical reflection and/or experimental research, is long and complex. For the present purposes, it will suffice to say that these questions remain very much open [26].

It is difficult, but not impossible, to combine these two problems into a single concept. In the multilevel process (beginning with peripheral receptors and ending in the cortex) of sifting through the enormous mass of information that comes to the senses, we choose primarily those stimuli which have some meaning for us. This protects our brain from overload, and makes it possible to construct a

relatively stable picture of the world. Originally, the selectivity of attention served the purposes of biological survival; now, it makes it possible for us to function in a complicated social environment.

In the synchronic model of memory [26], developed according to the principles of process neuropsychology and presented in Fig. 6, the attempt has been made to determine, on the basis of neuropsychological research, the functional principles for a new clinical approach, based on process theory (i.e. microgenetic theory) and evolutionary-genetic theory [34]. This model does not presume that there exists an absolute cause-and-effect relation, but at the same time it does not deny the possibility of rigorous scientific research. It does presume, however, that there is an overarching connecting principle among the phenomena involved in explicit and implicit memory, operating alongside the principle of causation. Synchronicity is defined as the appearance in parallel lines of two (or several) phenomena, event, or mental states that have, for the observer, a common meaning, though they are not linked causally. According to some scientists, synchronicity can be offered as an alternative to complete randomness [35,36,37,38].

The synchronic model of memory is derived from the holographic model of the universe, which, according to Peat [36,37], reflects the synchronicity of reality. The spatial arrangement of the model enables it to present, on the *x* and *y* axes, the relation between the general structure of the attention and memory systems in terms of the number, content, and complexity of the items being processed, and the time needed to process them. It is assumed, as in Pribram's concept [35], that thanks to the change of the angle at which bursts radiating from two lasers affect a photographic image, it is possible to keep many different images on the same surface.

The synchronic pattern of the model (the dotted line) in turn reflects the holographic interference of waves, corresponding to what goes on in the brain: the pulsing of mental states and changes in neuronal connections (including new connections arising in brain tissue).

In this model, consciousness and self-awareness have been represented by a separate circle, since these are prerequisites for the normal course of cognitive processes (including memory) and emotional processes. The outer (yellow) spiral refers to the fractal concept of consciousness and self-awareness in relation to mind, and to the synchronic image of reality formed by the self in relation to the world and the universe.

The tunnels through which the small spheres are swimming represent the various kinds of working and long-term memory, thanks to which the conscious self forms its own synchronic reality in the relation between the self, the world, and the universe [26]. Thanks to plasticity and new connections in the brain, there appears a kind of dependency between events, in which every causal connection is possible. The large yellow circles are buffers:
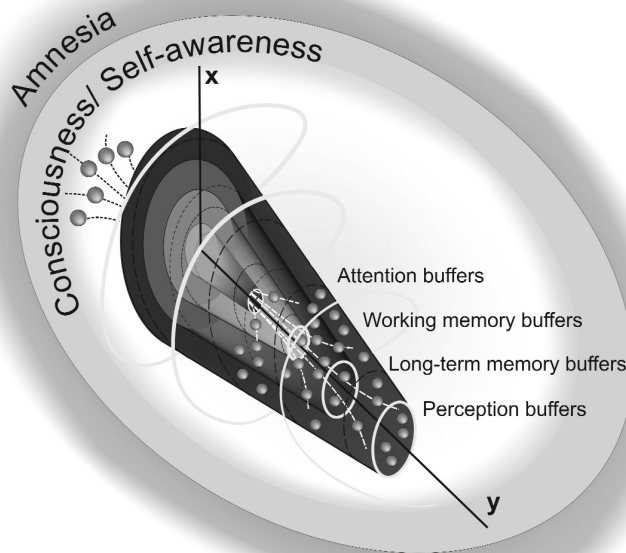- attention;
- working memory;

Fig. 6. The synchronic model of memory. The spatial arrangement of the model is based on a hologram. Interpretation: x axis: the number, content, and complexity of the elements a given system must use. y axis: the time needed to process these elements. The synchronic pattern of the spiral (dotted line): pulsing of mental states and changes in neuronal connections (including new ones). The tunnels through which the small spheres are flowing: different kinds of working and long-term memory. Large yellow circles: buffers for attention, working memory, long-term memory, and perception. Outer (yellow) spiral: the fractal concept of consciousness and self-awareness in relation to the mind and the synchronic image of reality created by the self
From: Pachalska, Kaczmarek & Kropotov [26], with permission

- long-term memory;
- perception.

According to data recently obtained from neurophysiological research, the attention system buffers the transmission of data to the working memory system. The attention system processes the smallest number of elements in the shortest time (seconds, even milliseconds). When the number of elements being processed and/or the processing time exceeds a certain threshold, there is a gradual transition from the attention system (a few stimuli, a few milliseconds) to the working memory system (a dozen or more stimuli, several milliseconds, seconds, or minutes), depending on the capacity of the working memory buffer.

The transition from working memory to long-term memory takes place in a similar fashion. The boundary for transition is difficult to establish precisely, and in reality it is probably rather blurred. In the human brain there is a constant process, lasting from milliseconds to whole years, when information is committed to memory, stored, recalled, and forgotten.

The longest duration of storage is naturally provided by long-term memory, which is why we have placed it near the base of the model. Memory (both retrospective and prospective) is closely associated with the creation of a model of the world, thanks to the perception buffer placed at the very base of the model. Without a properly functioning memory system, there is no properly functioning perception system, or other cognitive processes. It is also essential to note that in the process of perception, the feeling that an object exists and that it belongs to a primitive functional category precedes the awareness of its particular perceptual features [26,34].

On the edges of the model of memory, outside the circle of consciousness and self-consciousness, is unawareness and amnesia. It requires a major commitment of brain resources to recover information from this domain.

This model makes it easier (though admittedly not easy) to understand the improvement AG displayed as a result of neurotherapy. Thanks to the reduction of the Rolandic spikes and the regulation of the brain's electrical activity, the management of brain resources was improved. As a result, the attention and working memory buffers were expanded, which in turn caused new information to become available to long-term memory. The final effects of this could be seen in AG's spectacular results, not only in neuropsychological testing (including tests for memory storage capacity), but also in her very gratifying achievements, both at school and at home, especially her piano playing skills.

CONCLUSIONS

RBNT was successful in the treatment of a patient with benign partial epilepsy with Rolandic Spikes (BPERS), accompanied by the symptoms of attention deficit-hyperactivity disorder (ADHD). Event Related Potentials (ERPs) in the GO/NOGO task can be used to assess functional brain changes induced by neurotherapeutic programs.

REFERENCES

Holtmann, M., Becker, K., Kentner-Figura, B. & Schmidt, M.H. Increased frequency of rolandic spikes in ADHD children. Epilepsia, 2003; 44, 1241-1244.

Bishop DVM, Snowling MJ. Developmental dyslexia an specific language impairment: same or different? 2004; Psychol Bull 130:858–886.

Kramer U: Atypical Presentations of Benign Childhood Epilepsy With Centrotemporal Spikes: A Review. J Child Neurol July 2008; 23(7) 785-790.

Kropotov JD: Quantitative EEG, event related potentials and neurotherapy. San Diego: Academic Press, Elsevier, 2009.

Pąchalska M., Kropotov I.D., Mańko G., Lipowska M., Rasmus A, Łukaszewska B., Bogdanowicz M., Mirski A: Evaluation of a neurotherapy program for a child with ADHD with Benign Partial Epilepsy with Rolandic Spikes (BPERS) using event-related potentials. Medical Science Monitor. 2012; 18(11) CS 94-104.

Bali B, Kugler SL, Pal DK: Genetic influence on Rolandic epilepsy. Ann Neurol, 2005; 57(3): 464–65.

Chahine LM, Mikati MA: Benign pediatric localization-related epilepsies. 2006; Epileptic Disord 8 (4): 243–58.

Clarke T, Strug LJ, Murphy PL et al: High risk of reading disability and speech sound disorder in rolandic epilepsy families: case – control study.Epilepsia, 2007; 48(12): 2258–65.

Deonna T, Zesiger P, Davidoff V et al: Benign partial epilepsy of childhood: a neuropsychological and EEG study of cognitive function. Dev Med Child Neurol, 2000; 42: 595–603.

Huang-Pollock CL, Nigg JT, Halperin JM: Single dissociation findings of ADHD deficits in vigilance but not anterior or posterior attention systems. 2006; Neuropsychology 20:420–429.

Pan A, Luders HO: Epileptiform discharges in benign focal epilepsy of childhood. Epileptic Disord, 2000; 2S1: 29–36

de Saint-Martin AD, Carcangiu R, Arzimanoglou A et al: Semiology of typical and atypical rolandic eplepsy: a video-EEG analysis. Epileptic Disord, 2001; 3: 173–82.

Datta A, Sinclair DB. Benign epilepsy of childhood with rolandic spikes: typical and atypical variants. Pediatr Neurol 2007 March;36(3):141-5

Monastra, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., & Fenger, T. N: Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. Neuropsychology, 1999; 13, 424-433.

Pan A, Luders HO: Epileptiform discharges in benign focal epilepsy ofchildhood. Epileptic Disord, 2000; 2S1: 29–36.

Mayor C, Zesiger P, Roulet-Perez E et al: Acquired epileptic dysgraphia: a longitudinal study. Dev Med Child Neurol, 2003; 45: 807–12.

Chaix Y, Laguitton V, Lauwers-Cancès V, Daquin G, Cancès C, Démonet J.F, Villeneuve N: Reading abilities and cognitive functions of children with epilepsy: Influence of epileptic syndrome. Brain & Development 28 2006;122–130.

Barry RJ, Clarke AR, McCarthy R et al: Event-related potentials in adults with Attention-Deficit/Hyperactivity Disorder: an investigation using an inter-modal auditory/visual oddball task. Int J Psychophysiol, 2009; 71(2): 124–31.

Kropotov JD, Muller A: What can event related potentials contribute to neuropsychology. Acta Neuropsychologica, 2009; 7(3): 169–81.

Garcia-Penas JJ: Interictal epileptiform discharges and cognitive impairment in children. Revista de Neurologia, 2011; 52(Suppl.1): S43–52.

Mayor C, Zesiger P, Roulet-Perez E et al: Acquired epileptic dysgraphia: a longitudinal study. Dev Med Child Neurol, 2003; 45: 807–12.

Baglietto MG, Battaglia FM, Nobili L et al: Neuropsychological disorders related to interictal epileptic discharges during sleep in benign epilepsy of childhood with centrotemporal or rolandic spikes. DMCN, 2001; 43: 407–12.

Kropotov JD, Grin-Yatsenko VA, Ponomarev VA et al: ERPs correlates of EEG relative beta training in ADHD children. Int J Psychophysiol, 2005; 55(1): 23–34

Danielsson J, Petermann F. Cognitive deficits in children with benign rolandic epilepsy of childhood or rolandic discharges: a study of children between 4 and 7 years of age with and without seizures compared with healthy controls. Epilepsy Behav 2009 December;16(4):646-51.

Maquet P, Hirsch E, Metz-Lutz MN et al: Regional cerebral glucose metabolism in children with deterioration of one or more cognitive functions and continuous spike-and-wave discharges during sleep. Brain,1995; 118: 1497–520.

Pąchalska M., Kaczmarek B.L.J., Kropotov JuD. Neuropsychologia kliniczna: od teorii do praktyki. Warszawa: Wydawnictwo Naukowe PWN, 2014.

Diaz-Negrillo A. Benign infantile epilepsy. A new case with positive response to levetiracetam. Journal of Pediatric Epilepsy 3 (2014) 117–119. DOI 10.3233/PEP-14084.

Holtmann M, Becker K, Kentner-Figura B, Schmidt MH. Increased frequency of rolandic spikes in ADHD children. Epilepsia. 2003 Sep;44(9):1241-4.

Lubar, J. F: Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. Applied Psychophysiology and Biofeedback, 1991;16, 201-225

Monastra, V. J., Lubar, J. F., & Linden, M; The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: Reliability and validity studies. Neuropsychology, 2001;15, 136-144.

Martijn Arns, C. Keith Conners and Helena C. Kraemer A Decade of EEG Theta/Beta Ratio Research in ADHD : A Meta-Analysis. Journal of Attention Disorders. 2013; 17: 374-383

Sterman, M. B: Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. Clinical Electroencephalography, 2002; 64, 45–55.

Walker, J. E., & Kozlowski, G. P: Neurofeedback treatment of epilepsy. Child and Adolescent Psychiatric Clinics of North America, 2005;14, 163–176.

Brown, J. W: Simultaneity and serial order. Journal of Consciousness Studies, 2010: 17, 7-40.

Pribram K: The Holographic Hypothesis of Brain Functioning, [In:] S. Grof (ed.): Ancient Wisdom, Modern Science, State University of New York Press, 1984, s. 174-175.

Peat D.F:Synchronicity: The Bridge Between Matter and Mind: New York: Bantam,1987.

Peat D.F: A Flickering Reality: Cinema and the Nature of Reality, Pari Publishing, 2011.

Bloch, D. P: Complexity, Connections, and Soul-Work. Journal of Catholic Education, 2013; 11 (4): 543- 554.

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