

Conference Paper

Follow-Up to Case Study: Neurofeedback as a First Choice Treatment in an ADHD and Comorbidities

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INESP – Instituto Neurológico de São Paulo, São Paulo, Brazil**Abstract**

The purpose of this case study is to present the evolution in the neurofeedback treatment of a 7-years-old boy with ADHD and comorbidities – OCD, Anxiety and Aggression. The main complaint of parents and school was the lack of control of his impulses, beating his classmates, not sitting quietly in the classroom, disrupting classmates, biting fingernails and toes compulsively – getting hurt. He was asked to withdraw from the previous school and, the parents saw neurofeedback as an alternative non-drug treatment, since the psychiatrist suggested an antidepressant. Brain training by neurofeedback occurred twice a week, in a total of 70 sessions, where the brain areas with the greatest impairment were trained. With only two months of treatment, the boy's aggressiveness was no longer a problem. At the end of the treatment, the functional impairments were better, validated by mean evolution evaluations. The patient was released, with a high approval rating from parents and school, since their symptoms disappeared. After 1 year and 9 months of the end of treatment at the age of 9, a new assessment was performed to see if the gains remained. The results showed that through Neurofeedback training the brain had the ability to normalize his electrical activity and maintain the gain over time.

Keywords: ADHD, neurofeedback, rehabilitation

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Received: 25 July 2018

Accepted: 9 August 2018

Published: 1 November 2018

Publishing services provided by
Knowledge E

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Selection and Peer-review under the responsibility of the Fifth International Luria Memorial Congress Conference Committee.

1. Introduction

The human brain is the largest data processing system on our planet: a 100 billion neuron structure that forms more than 10 trillion connections between them and has an extraordinary ability to modify its own structure through neural plasticity.

In the last decades, there has been a great advance in neuroscience with the advent of new technologies that allowed the visualization of internal structures of the brain (tomography and magnetic resonance), going beyond the structure, regarding

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anatomy and pathology, but being possible to visualize the brain functioning caused, for example, by a specific motor action [1].

In the midst of this universe of neuroscience and neuropsychology and its attempts to understand brain functioning, it consolidated in the 1960s, 70s neurofeedback, as a field of science, which has since then proven its benefits through serious scientific studies with the most renowned Ph.D.s [2].

Many of the efforts in the area of neurofeedback have focused on ADHD studies and learning problems with Dr. Joel Lubar, who since 1970 has repeatedly demonstrated, through controlled and randomized studies, that it is possible to re-train the brain. One of his published studies covers 10 years of follow-up, where 80% of neurofeedback-trained patients have shown substantial improvements in ADHD symptoms, and these changes have remained over the years [2].

The importance of these studies is due to the fact that ADHD is one of the most important public health problems in the world. It is estimated that the prevalence is 5% of the population (DSM-V, 2014). The disorder manifests itself in childhood and is characterized by three main symptoms: distraction, impulsivity and hyperactivity [4].

In addition to problems with school performance, family and social relationships, psychosocial adjustment and at work, 50% of ADHD patients have comorbidities with learning disorder, mood and anxiety disorder, substance and alcohol abuse disorder, which gets worse the clinical picture [5].

According to Dr. Ana Beatriz [4], identifying a person with ADHD always has been a challenge, due to the diagnosis being essentially clinical based on the criteria derived from the DSM classification system. ADHD treatment is multidisciplinary (speech therapist, psychologist and pedagogue) and medication, but medication still the first choice treatment in nowadays.

While some doctors advocates for the use of medications in the treatment of ADHD as a tool that should be used for a better quality of life [4], Demos (2005) says that the use of drugs only alters the electrochemistry of the brain, but does not cure the problem, while neurofeedback is a training that promotes change in the cellular level of the brain, responsible for a definitive change. A breakthrough in science!

But how does neurofeedback work?

After understanding brain structure and function using Quantitative Electroencephalography (QEEG) to begin to understand how de-regulated areas account for a patient's symptoms, neurofeedback involves training and learning self-regulation of

brain activity (Jay Gunkelman), based on the general principles of biofeedback. It is a non-invasive, non-drug treatment with no side effect and no addiction.

Through accurate instruments to measure brain waves activity, this comprehensive training system, in real time and with precision, send information of 'feedback' to the user. The presentation of this information supports desired physiological changes. Over time, these changes remain without the continued use of the instrument.

The purpose of this case study is to present the evolution in the neurofeedback treatment and follow up after 1 year and 4 months after the end of the treatment, to verify if the gains in neurofeedback training remains over time.

2. Methodology

The participant was a 7-year-old male patient, diagnosed with ADHD and comorbidities – OCD, Anxiety and Aggression. The parents looked for Brain Healthy Institute for a non-invasive and non-drug treatment.

Acquisitions was a Nineteen channels of EEG data acquired with a Brainmaster, Discovery 24 amplifier, and Neuroguide Acquisition software. The EEG data was gathered at 70 microvolts sensitivity and digitized at a rate of; 256 samples/second/channel. The EEG was analyzed with Neuroguide 2.8.4 software from Applied Neuroscience, Inc.

EEG data were collected during at least 5 minutes on an 'eyes open' conditions, and were free edited for 10 seconds and then automatic edited to corrected artifacts for eye movement, which obtained at least 1.00 minute of good EEG data.

During the treatment was acquired 10 assessments, and 2 assessments of follow up (one after 4 months at the end of the treatment and one 21 months after).

There were 70 training sessions, twice a week, that last 20 minutes each and the protocols were adapted after each assessment.

The treatment was interrupted twice, one after 43 sessions, the patient was 1 month absent for health reasons and when he returned he did 3 more sessions and stopped again, this time for another 2 months also for health reasons. After this last interruption, he did more 24 sessions until finish the treatment.

3. Results

On August 2014, the first normative QEEG, identified the exactly location of his functional impairments: excess of theta slowing at 7hz (absolute power) all over motor

strip (the middle line) and parietal areas, with more than 2 to 3 standard deviation (Figure 1).

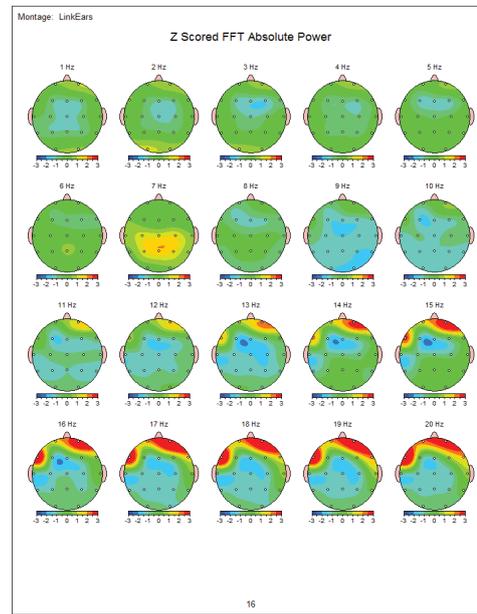


Figure 1: QEEG values (z-scores) absolute power for the Eyes Open condition, Linked Ears montage – 1st assessment 08/14/2014.

The data are presented as z scores (standard deviation units), representing the deviation of power in each area of the brain from the values in a normative database of EEG records (see color scale on the map: green z score = 0; red z score = +3; dark blue z score = -3). Power is measured in microvolts and Figure 1 represents data for Absolute Power, the amount of energy in each frequency band for each electrode placements that conform to the 10–20 International Electrode System.

Also theta/beta ratio was 10:1 at Cz, which means 10 times more slowing theta wave than beta (Figure 2). A theta-to-beta ratio greater than 3:1 configure as slow-wave disorder measured on Cz, and this is the classic ADHD pattern [6].

Low-resolution Electromagnetic Tomography Analysis (LORETA) identify that the excess of 7 Hz theta was coming from Broadman area 5 and 6 at Frontal Lobe, Broadman area 7 – the Precuneus at Parietal Lobe, Broadman area 23, 29 and 31 in the Gyrus Cingulate at Limbic Lobe and broadman area B31 in Later Cingulate also at Limbic Lobe. All these areas have an important role in attention network: Frontal, Cingulate and Precuneus (Figure 3).

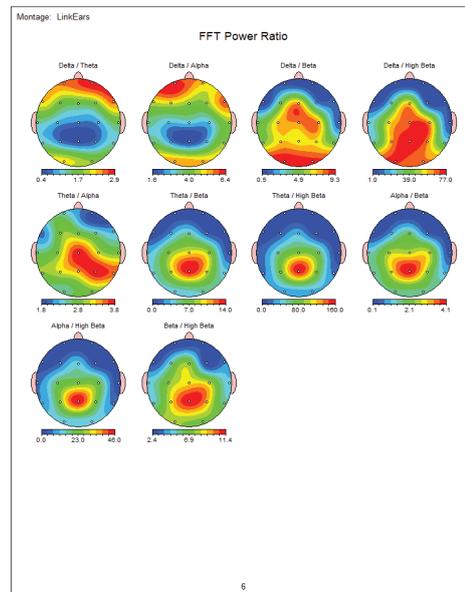


Figure 2: FFT Power Ratio - Cz - 10:1 – 1st assessment 08/14/2014.

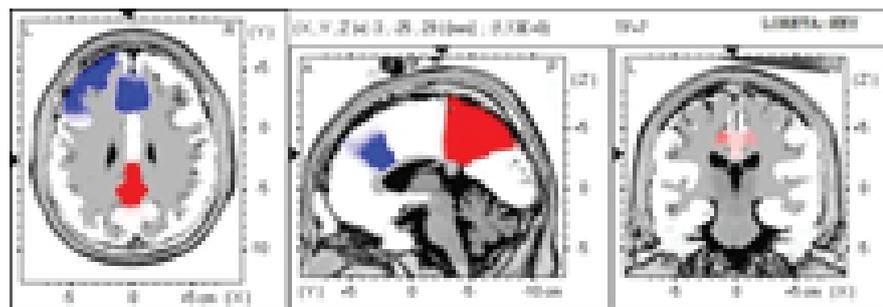


Figure 3: Low-resolution Electromagnetic Tomography Analysis (Loreta) – 1st assessment 08/14/2014.

After 70 sessions, patient was discharged. The same pattern of results was obtained at the 6-month follow-up: the slowing at 7 Hz was normalized (Figure 4), and theta/beta ratio dropped from 10 to 4.6 in CZ (Figure 5).

In addition to the clear changes on the maps, the symptoms of aggression, lack of control, disturbing colleagues at classroom, eating nails and things have disappeared. School and parents had no complains anymore.

One more assessment was done, 1 year and 9 months follow-up, and the new QEEG shows that all gain remained from the previous training (Figure 6). Table 1 show the quantitative changes on the electric activity.

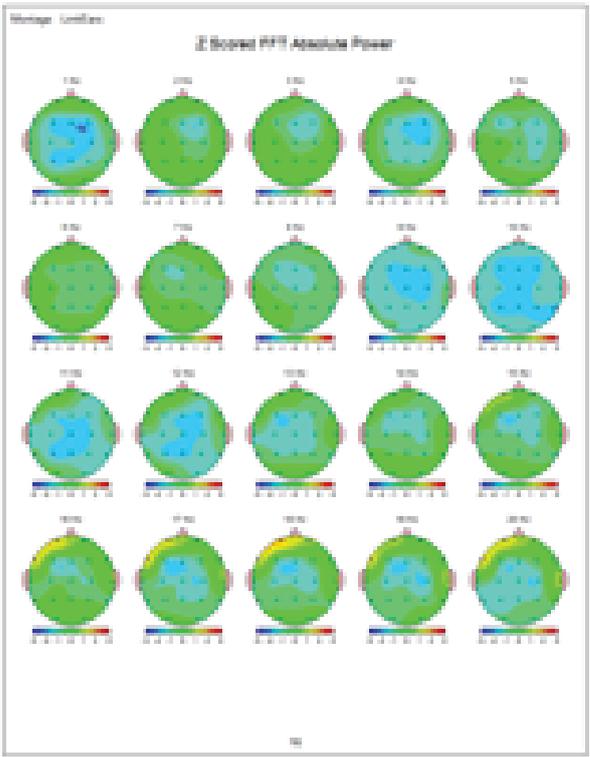


Figure 4: QEEG values (z-scores) absolute power for the Eyes Open condition, Linked Ears montage – 11th assessment 11/27/2015.

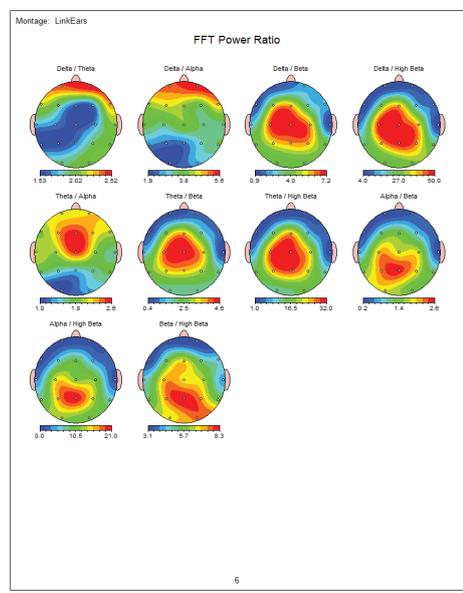


Figure 5: FFT Power Ratio - Cz - 4.6:1 – 11th assessment 11/28/2015.

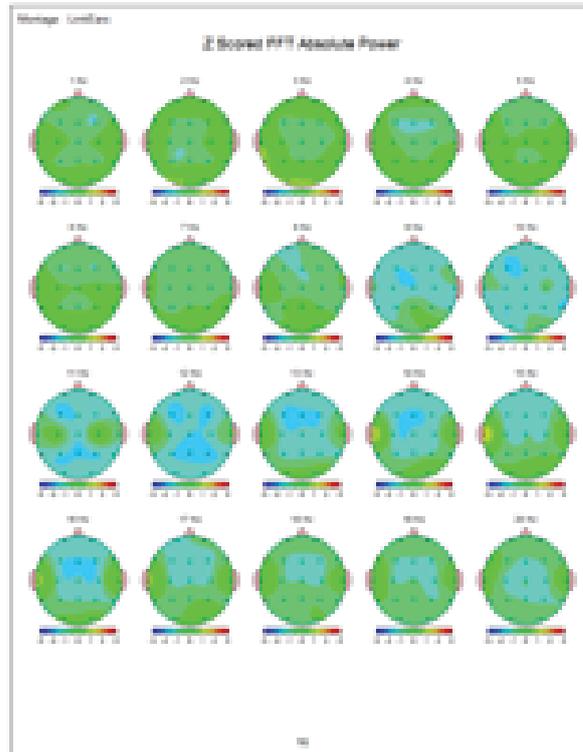


Figure 6: QEEG values (z-scores) absolute power for the Eyes Open condition, Linked Ears montage – 12th assessment 03/24/2017.

TABLE 1: Comparative table – Z score peak frequency.

Montage: LinkEars

Z Scored Peak Frequency

Intrahemispheric: LEFT

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|--------|-------|-------|-------|-------|-----------|--------|--------|--------|
| FP1-LE | -0.20 | -0.50 | 0.73 | 1.67 | 1.32 | 1.44 | 1.37 | 0.53 |
| FP2-LE | -0.18 | -0.78 | 0.50 | 1.14 | 0.74 | 0.60 | 1.01 | 0.60 |
| C4-LE | 0.41 | -2.04 | -0.70 | -0.09 | -0.42 | 0.03 | -1.30 | 0.79 |
| FP1-LE | 0.24 | -0.07 | 0.09 | 0.09 | 0.26 | 0.09 | -0.27 | 1.00 |
| FP2-LE | 0.37 | 0.28 | 0.32 | 0.09 | -0.27 | 0.09 | -1.01 | 1.00 |
| O1-LE | 0.86 | 0.24 | 0.32 | 0.09 | -0.27 | 0.09 | -1.01 | 1.00 |
| FP1-LE | 0.37 | 0.28 | 0.32 | 0.09 | -0.27 | 0.09 | -1.01 | 1.00 |
| FP2-LE | 0.39 | 0.37 | 1.46 | 0.04 | 0.11 | 0.09 | -0.40 | 0.42 |
| T8-LE | 1.54 | 0.43 | -0.48 | 0.41 | -0.38 | 0.09 | 0.22 | 0.70 |

Intrahemispheric: RIGHT

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|--------|-------|-------|-------|-------|-----------|--------|--------|--------|
| FP2-LE | -0.47 | -0.47 | 1.51 | 1.61 | 0.40 | 0.31 | 1.31 | 0.50 |
| FP1-LE | -0.60 | -0.30 | 0.54 | 0.02 | 0.47 | 0.13 | -1.00 | 0.74 |
| C4-LE | 0.10 | -2.00 | -0.54 | -0.04 | 0.24 | 0.17 | -1.02 | 0.51 |
| FP1-LE | 0.24 | -0.07 | 0.09 | 0.09 | 0.26 | 0.09 | -0.27 | 1.00 |
| O1-LE | 0.86 | 0.24 | 0.32 | 0.09 | -0.27 | 0.09 | -1.01 | 1.00 |
| FP1-LE | -1.31 | -0.50 | 0.37 | 1.00 | 0.17 | 1.11 | -1.00 | 0.51 |
| T8-LE | -0.30 | 1.00 | -0.00 | 1.10 | 0.48 | 1.00 | 1.41 | -0.13 |
| T6-LE | 0.21 | 1.00 | -0.38 | 0.70 | 1.04 | 0.41 | -0.40 | 0.30 |

Intrahemispheric: CENTER

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|-------|-------|-------|-------|-------|-----------|--------|--------|--------|
| Fz-LE | -0.17 | 0.40 | -0.00 | 0.11 | -0.20 | -0.09 | 0.93 | 0.60 |
| Cz-LE | 0.10 | -1.17 | -0.70 | -0.30 | -0.00 | 0.11 | -1.14 | 0.70 |
| Pz-LE | 0.60 | -1.10 | -1.63 | -0.30 | -0.00 | 1.01 | -1.00 | 1.20 |

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Montage: LinkEars

Z Scored Peak Frequency

Intrahemispheric: LEFT

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|--------|-------|-------|-------|------|-----------|--------|--------|--------|
| FP1-LE | -0.50 | -0.71 | -0.14 | 1.41 | 0.71 | 0.53 | 0.50 | 0.32 |
| FP2-LE | -0.50 | -0.71 | -0.14 | 1.41 | 0.71 | 0.53 | 0.50 | 0.32 |
| C4-LE | -0.10 | -0.40 | 0.00 | 0.50 | 0.00 | -0.40 | -0.44 | 0.80 |
| FP1-LE | 0.20 | -0.20 | 0.00 | 0.20 | 0.00 | 0.20 | -0.20 | 1.00 |
| FP2-LE | 0.20 | -0.20 | 0.00 | 0.20 | 0.00 | 0.20 | -0.20 | 1.00 |
| O1-LE | 0.40 | 0.40 | -0.20 | 0.20 | 0.00 | 0.40 | -0.40 | 0.40 |
| FP1-LE | 0.20 | -0.20 | -0.20 | 0.20 | 0.00 | 0.20 | -0.20 | 1.00 |
| FP2-LE | 0.20 | -0.20 | -0.20 | 0.20 | 0.00 | 0.20 | -0.20 | 1.00 |
| T8-LE | 1.00 | -0.40 | -0.40 | 0.40 | 0.00 | 0.50 | 0.50 | 0.20 |

Intrahemispheric: RIGHT

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|--------|-------|-------|-------|------|-----------|--------|--------|--------|
| FP2-LE | 1.40 | 0.40 | -0.20 | 0.30 | 0.70 | 0.10 | 0.50 | 0.30 |
| FP1-LE | 0.20 | -0.40 | -0.10 | 0.30 | 0.40 | 0.10 | -0.10 | 0.30 |
| C4-LE | -0.20 | -0.30 | 0.00 | 0.30 | 0.10 | -0.10 | -0.10 | 0.80 |
| FP1-LE | 0.40 | -0.20 | -0.20 | 0.20 | 0.00 | 0.40 | -0.40 | 1.00 |
| O1-LE | 0.40 | 0.40 | -0.20 | 0.20 | 0.00 | 0.40 | -0.40 | 0.40 |
| FP1-LE | 0.30 | -0.30 | -0.30 | 0.30 | 0.70 | 0.44 | -0.30 | 0.30 |
| T8-LE | -0.20 | 0.30 | -0.30 | 0.30 | 1.40 | 1.20 | 0.50 | -0.20 |
| T6-LE | 1.10 | -0.30 | -0.40 | 0.20 | 0.00 | 1.00 | -0.70 | 0.50 |

Intrahemispheric: CENTER

| | DELTA | THETA | ALPHA | BETA | HIGH BETA | BETA 1 | BETA 2 | BETA 3 |
|-------|-------|-------|-------|------|-----------|--------|--------|--------|
| Fz-LE | 0.20 | -0.40 | -0.00 | 0.50 | 1.00 | -0.00 | -0.30 | 1.30 |
| Cz-LE | 0.10 | -0.70 | -0.10 | 1.10 | 0.60 | 0.00 | -0.30 | 0.90 |
| Pz-LE | 0.40 | -0.70 | -1.40 | 0.30 | 0.60 | 0.40 | 0.40 | 0.30 |

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4. Conclusion

The results showed that through Neurofeedback training, the brain had the ability to normalize its electrical activity and maintain the gain over time. It seems the brain actually learned how to function more efficiently.

This is just a single case study with the purpose of following up after the end of the training, but besides all good results, some concerns have to be taken into consideration. During the assessment the patient didn't cooperate to sit still in order to make assessments in 'eyes closed' conditions. Also the treatment had a 3 months interruptions, and it was able to observe that 43 sessions wasn't enough to regulate his abnormality in 7Hz with Z-score neurofeedback, even though some symptoms were better by this time.

More research should be done with long term follow up.

References

- [1] Andrade, V. M., Santos, F. H., and Bueno, O. F. A. (2004). *Neuropsicologia hoje*. São Paulo: Artes Médicas.
- [2] Hammond, D. C. (2011). What is neurofeedback: An update. *Journal of Neurotherapy*, vol. 15, no. 4, pp. 305-336. Retrieved (September 29, 2013) from <http://www.tandfonline.com/doi/pdf/10.1080/10874208.2011.623090>
- [3] American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th edition). Washington, DC: Author.
- [4] Silva, A. B. B. (2003). *Mentes inquietas: entendendo melhor o mundo das pessoas distraídas, impulsivas e hiperativas*. São Paulo: Gente.
- [5] Rohde, L. A. P and Mattos, P. (2003). *Princípios e práticas em TDAH: Transtorno de Déficit de Atenção/Hiperatividade*. Porto Alegre: Artmed.
- [6] Demos, J. N. (2005). *Getting Started with Neurofeedback*. New York: W.W. Norton & Company.