Neurofeedback treatments enable the EEG-normalization and total seizure control of epilepsy – A Case Study

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Abstract:

Over the past 40 years, researchers have examined various non-drug approaches to the treatment of epilepsy. Neurofeedback is an approach that has been proven efficient in reducing seizure frequency.

Our experience in treating children and adolescents with epilepsy shows that QEEG guided amplitude training enables one to reach total elimination of seizure activity in a relatively short time (3-5 months of training). In addition, our patients' EEG, which before treatment included numerous appearances of spike & wave complexes, was sampled again towards the end of treatment and was found to be normal (with no spikes and discharges).

This article presents two cases of epilepsy patients who were treated with QEEG guided neurofeedback at our clinic. Both of them experienced total cessation of seizures at an early stage in the treatment and displayed substantial behavioral and cognitive improvement during the course of treatment. Both patients displayed a normal EEG at the end of the treatment.

The cases described in this article lend support to the assumption that neurofeedback treatments enable the EEG-normalization and total seizure control of epilepsy patients who do not respond (or only partially respond) to anti-convulsant medicines. Many of these patients do not have an epileptic focus, and therefore are not considered suitable candidates for neurosurgery. Other alternatives are expensive and have low efficiency. These results lend support to the idea that tailoring the neurofeedback treatment protocol specifically to each individual patient by doing QEEG tests improves treatment quality and precision in a way which enables the achievement of full control over seizures and full EEG-normalization.

Introduction:

Epilepsy is a seizure disorder characterized by abnormal electrical brain activity.

According to the World Health Organization, about 50 million people around the world suffer from epilepsy, which amounts to 0.8% of the world population[1]. Epilepsy has traditionally been treated with anticonvulsant drugs. However, about one third of patients do not respond to medication and are unable to control their seizures[2,3]. Their situation is often complicated by the drugs' aversive side effects, which are detrimental to their health. Also, women who want to become pregnant are warned against severe harm that might be caused to their fetus by the drugs[4]. In several cases in which seizures are not controlled by medication, patients might undergo neurosurgery, whereby specific neural pathways are severed to prevent extensive epileptic activity. Seizures are caused by abnormal, excessive electrical activity in the brain. The pattern is one of over-synchronization of neuronal activity[5].

In many cases, the cause of the disorder is not clear, but its onset sometimes follows the appearance of brain cancer, brain trauma or stroke[1]. Abuse of alcohol and/or drugs might also be a factor.

Another option for treating epilepsy, aside of anti-convulsant medication or neurosurgery, is neurofeedback (aka EEG Biofeedback), a non-medicinal method that has been gaining momentum and recognition over the past two decades, with high success rates in the treatment of epilepsy as well as other neuropsychiatric disorders (such as ADD/ADHD, learning disabilities, anxiety and more)[6]. The method's success in reducing seizure frequency and intensity for epileptic patients is highly impressive, given the fact that

most studies published have dealt with patients whose seizures were uncontrollable using orthodox medicines[7].

Neurofeedback:

Neurofeedback is a form of neurotherapy that is based on the principle of operant conditioning. By this method, the brain is trained to normalize its own electrical activity through receipt of congruent feedback for its different EEG patterns (i.e: positive, reinforcing feedback for normative activity patterns; and negative, frustrating feedback for abnormal activity patterns). After a few trials, the brain starts to realize that there is a correlation between the kind of feedback it receives and the kind of EEG pattern that brings it about. Once such a realization takes place, the brain starts a slow, gradual process of changing its own activity patterns in order to avoid negative feedback and win more of the positive feedback. Such learning leads to the (partial or total) normalization of the EEG patterns. When the EEG activity starts to normalize, neuropsychiatric symptoms (which characterize non-normative EEG activity) start to diminish in size and in frequency.

One of the most common neurofeedback protocols is the up-training of Sensory Motor Rhythms (SMR; i.e: frequencies of 12-15 Hz over the somatosensory and motor strips of the brain). SMR up-training as an efficient treatment for epilepsy was first discovered in the mid-'70s by Prof. Barry Sterman of the UCLA School of Medicine[7]. Since then neurofeedback as a whole has evolved in both form and scope to include treatment of a host of other neuropsychiatric disorders, such as ADD/ADHD, learning disabilities, communication problems of the autistic spectrum, anxieties, depression and more. The

method is widely researched in clinical and academic institutes worldwide, with high success rates reported for the treatment of the various disorders.

History of neurofeedback treatment for epilepsy:

Research in most institutes has focused on up-training of 12-15 Hz EEG waves (i.e: SMR) as a neurofeedback treatment protocol for Epilepsy[8-10]. Most research studies report high success rates in reducing seizure frequency and magnitude for patients who do not respond to anti-convulsant drugs[11]. Sterman et al.[10] have demonstrated for the first time the applicability of SMR up-training to four epileptic patients who, following treatment, experienced significant improvement in their ability to control their seizures.

Cott et al.[12] found that 5 out of 7 patients experienced a reduction in the frequency of their previously uncontrolled seizures after three months of SMR up-training.

Kaplan[13] found clinical improvement in 80% of patients treated with neurofeedback. Similarly, Finley et al.[14] found reduction of seizures and normalization of EEG in a severely epileptic patient following SMR up-training.

Lantz and Sterman[15], in a large, double-blind controlled clinical trial on SMR up-training, found a 61% seizure reduction in the experimental group only.

Tan et al.[16] in their meta-analysis of 10 carefully selected research studies which answer their criteria for inclusion, determined that SMR up-training consistently decreased seizure rate among severe cases of epilepsy, which could not otherwise be controlled.

Andrews & Schonfeld[17] found that out of a sample of 83 patients, 80% managed to gain control over their seizures using SMR up-training neurofeedback protocol together with other methods of intervention (such as diaphragmatic breathing).

Two different studies[18,19] found that SMR up-training influences epileptic EEG also during sleep, when no conscious effort is done by the patient to control it: as SMR was up-trained, night-time epileptiform activity decreased.

In a review of research studies spanning the years 1972-1996, Sterman[8] concluded that 82% of 174 patients who participated in these studies gained clinical improvement, while around two thirds of them managed to achieve change in their EEG towards normalization. Other researchers (like Lubar and Bahler[20], Zhao, Wu, Liang and Hu[21], Johnson and Meyer[22]) reported decreases in seizure activity or even total cessation of seizures in some of the patients following neurofeedback treatment. Walker and Kozlowski[23] claim that a QEEG guided coherence training improves treatment outcomes. The question remains to be asked: how is the neurofeedback effect achieved?

Possible mechanisms mediating the neurofeedback SMR effect:

The Sensory Motor Rhythms (SMR) seem to emanate from the thalamus (specifically, from the ventrobasal nuclei of the thalamus, which conduct afferent somatosensory information[24]). During SMR up-training, the firing pattern of these thalamic nuclei becomes more systematic and rhythmic, which suggests suppression of somatosensory information passage[25]. The GABA neurotransmitter participates in this process. This is also influenced by nonspecific cholinergic and monoaminergic neuromodulation, which can affect excitability levels in the thalamus and in cortical areas receiving the thalamic input[11]. SMR up-training is thought to result in better control over excitation in that system. Epilepsy is characterized by over-excitation of the cortical and/or thalamocortical areas. SMR training raises excitation threshold in these areas, and thus exerts its

therapeutic effect. Another structure implied in this process is the striatum complex of the basal ganglia[11]. Froemke[25] discusses coincidence detection and synaptic plasticity, and his concepts might be in line with an LTP (long-term potentiation) based explanation for the neurofeedback effect. All in all, it appears that the SMR up-training neurofeedback effect is achieved through the enhancement of inhibitory mechanisms in sensorimotor pathways[26].

Case 1 report

There are many kinds of epilepsy. Among children, the most common type is known as Rolandic epilepsy, which is characterized by spikes over centro-temporal regions of the brain (the Rolandic Strip)[27]. The seizures are considered to be partial, because they occur over the Rolandic region of the brain only[28]. This kind of epilepsy usually starts in early childhood (sometimes as early as the age of 3), and often naturally recedes in adolescence[29,30]. Other neuropsychiatric and cognitive symptoms may accompany this disorder in some cases, such as: attention deficit disorder and learning disabilities (specifically, difficulties with oral and written language or with drawing and visuo-spatial skills)[30,31]. Despite this, children with this kind of epilepsy usually have normal levels of intelligence. This kind of epilepsy is also related to acquired epileptic aphasia (Landau-Kleffner Syndrome). Oral-motor deficiencies appear during the epileptic phase in some children. Recent studies have shown that adjustment difficulties at school, experienced by some of these children, are in correlation with their epileptic electroencephalographic activity[31].

This case study involves a patient with Rolandic epilepsy, A.B., age 8 years, who suffered from neuropsychiatric symptoms, cognitive dysfunction, verbal apraxia of speech and gross motor dysfunction. She was unable to function effectively in school and in many everyday life situations.

The first seizure was observed when she was 4.5 years old. She had an event of a sudden and involuntary contraction of muscles in all limbs, without loss of consciousness (before sleep). Her EEG taken at that time at the hospital showed forms of epilepsy and recurrent

seizures with Rolandic temporal spikes on both sides. She was treated with Tegretol and later on with Depalept.

Although she was treated for epilepsy, her function at school and at home was very low. At the age of 7.5 her diadochokinesis was anomalously clumsy (e.g: she could not even put her shoes on and she could not speak).

A clinical EEG test was conducted in our clinic at intake for 10 minuets, with eyes open and eyes closed (Figure 1). Her parents were asked to fill a Conners' parent questionnaire before intake and during subsequent assessments.

EEGs were recorded from 19 scalp sites. The electrodes were applied according to the International 10-20 system. The EEG was recorded in the average montage.

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

The EEG was analyzed with EEG/QEEG software (WinEEG).

The analysis consisted 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.

We used the automatic spike detection, analysis and average spike calculation system. This resulted in evidence of significant paroxysmal activity consistent with the spike & wave pattern in centro-temporal regions, mostly on the left side (Figure 2). There were significant 257 events detected over 10 minutes of recording. We used low resolution

brain electromagnetic tomography (Loreta) analysis to locate the source distribution. It was located by Loreta in the middle temporal gyrus, Brodman area 22 (Figure 3).

2) Fast-Fourier Transformation (FFT) of the corrected EEG for extracting EEG power. We computed EEG with eyes open and eyes closed average and compared to age-regressed, normative database (HBI), for absolute power (Figures 4,5) . There were obvious power-excesses in the 3-9Hz and 11-15 Hz band frequencies (Theta, Alpha and low Beta wave bands).

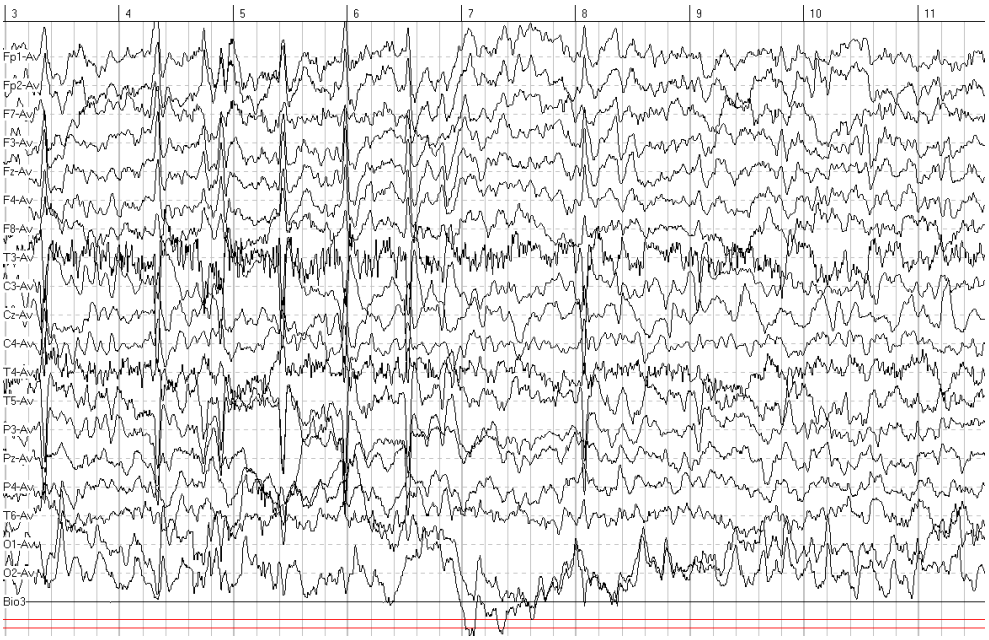


Figure 1: complexes of spike & wave on the patient raw EEG (eyes open), at intake.

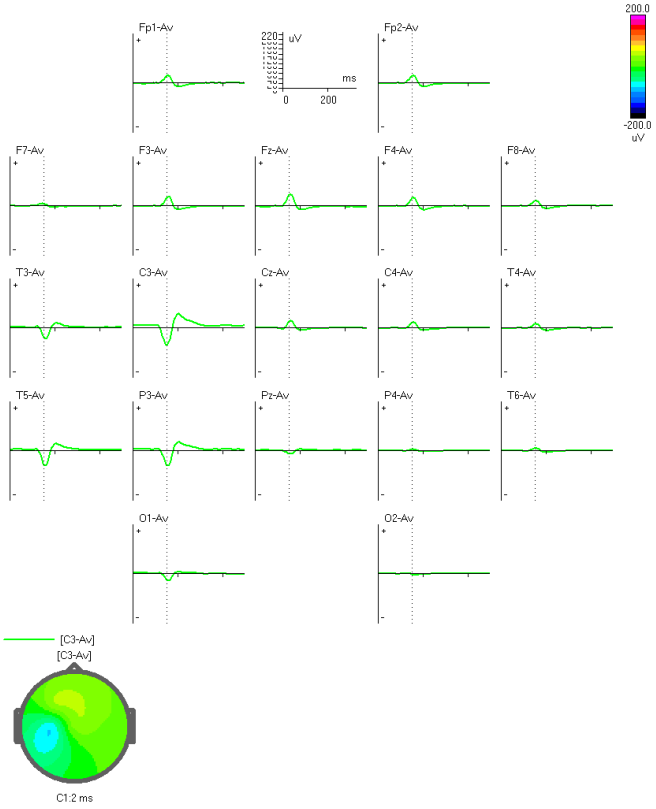


Figure 2: Spike & wave pattern detected by the automatic spike averaging system on C3 at intake.

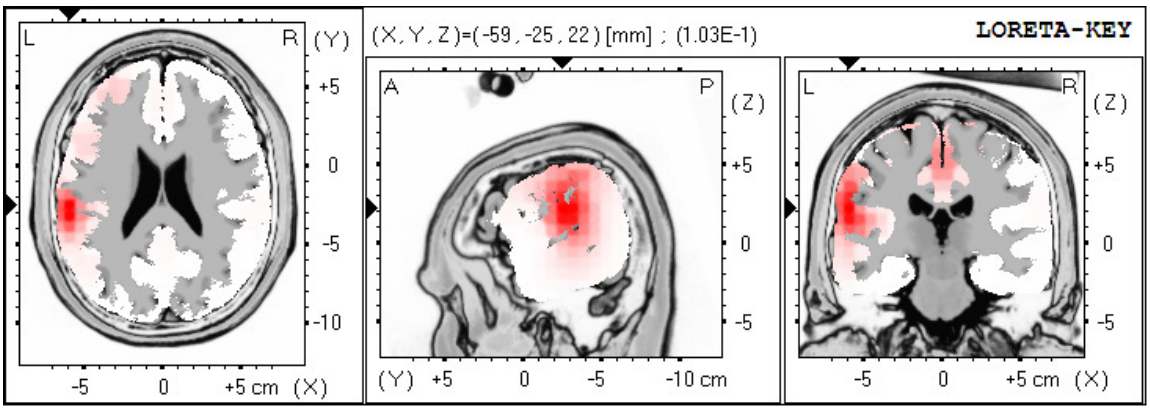


Figure 3: Loreta analysis for source distribution indicates source in middle temporal gyrus, Brodman area 22.

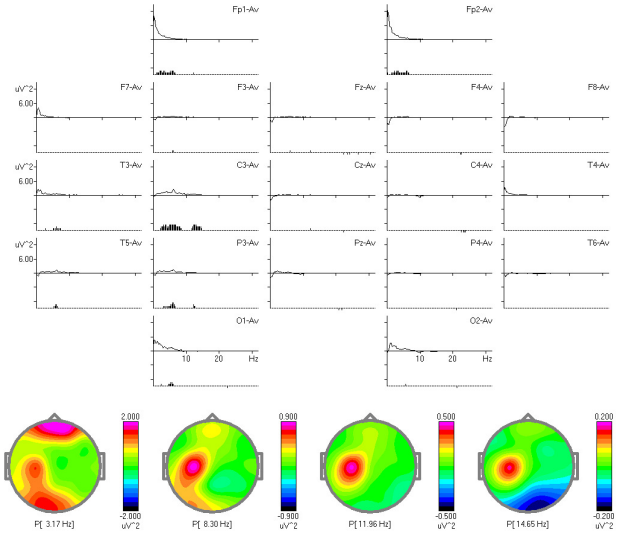


Figure 4: Graphs of EEG power spectra (eyes open) compared to a normative database, at intake.

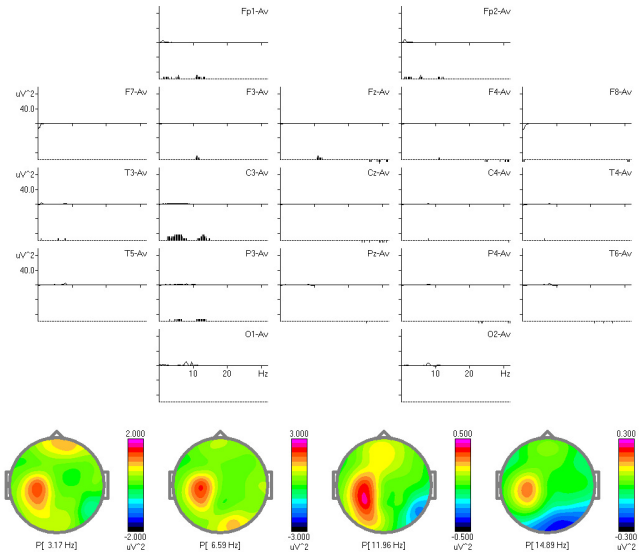


Figure 5: Graphs of EEG power spectra (eyes closed) compared to a normative database, at intake.

The patient underwent 20 guided Neurofeedback training sessions. The training involved the sensory motor strip, not directly involving the regions of the epileptiform activity. We up-trained 12-15 Hz (i.e: SMR). During the training, the patient was still receiving anticonvulsant medication. The parents reported a significant improvement in her speech comprehension and linguistic abilities.

After 17 sessions of Neurofeedback we recorded and analyzed her EEG again.

There were 115 events detected over 10 minutes of recording on C3 (Figure 6), and a significant change in the eyes-open and eyes-closed power spectra, compared with norms (HBI database), for absolute power (Figures 7,8).

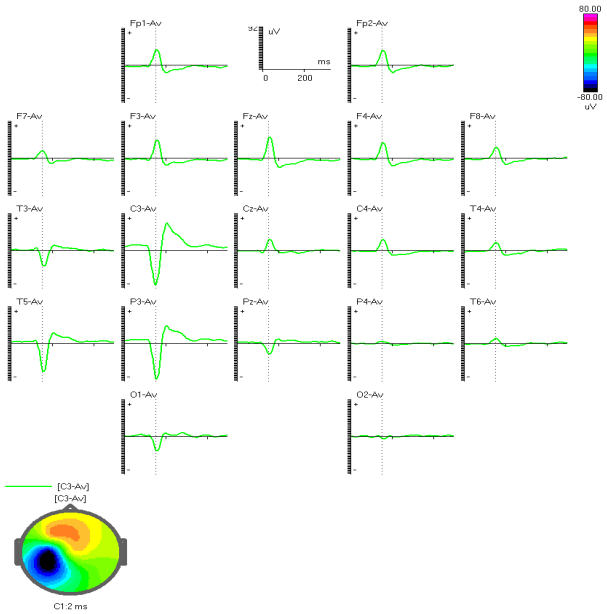


Figure 6: Spike & wave pattern detect by the automatic spike averaging system on C3, after 17 neurofeedback sessions.

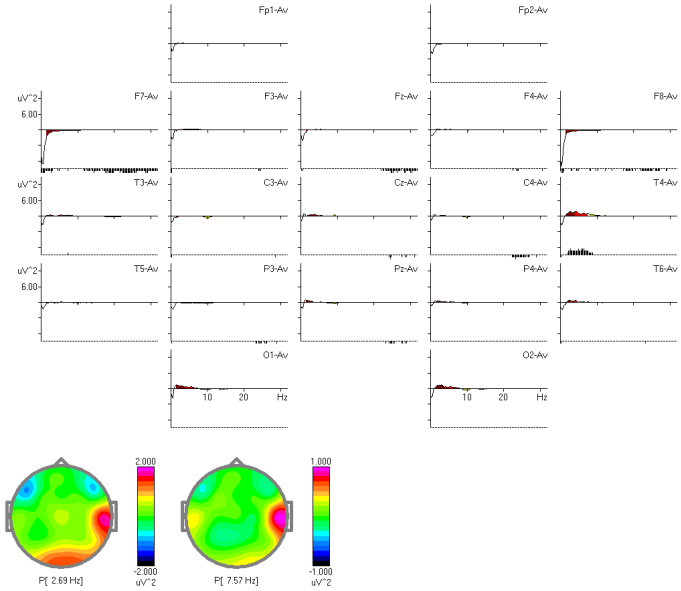


Figure 7: Graphs of EEG power spectra (eyes open) compared to a normative database, after 17 neurofeedback sessions.

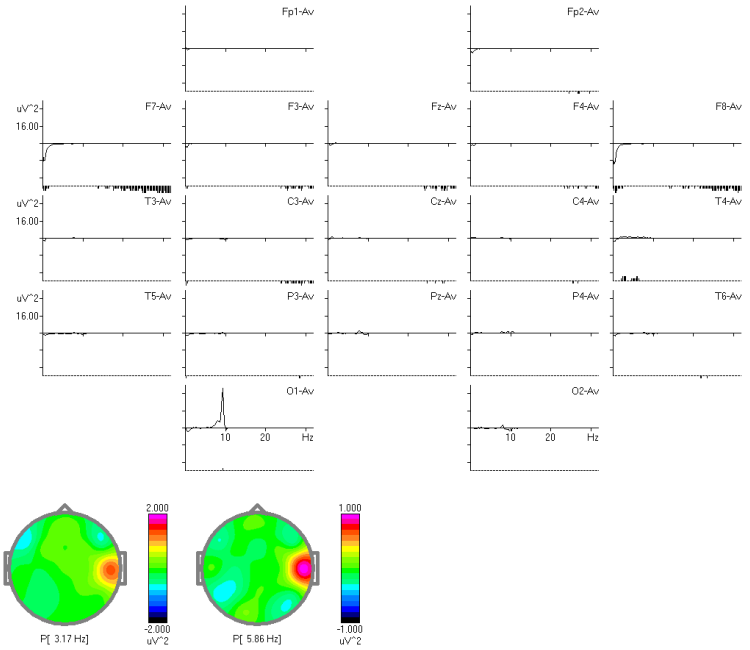


Figure 8: Graphs of EEG Eyes closed power spectra compared to a normative database, after 17 neurofeedback sessions.

The patient underwent 25 more guided Neurofeedback training sessions. Around the middle of the training period there were no seizure events, so she stopped taking anticonvulsant medication. Continued improvement of language abilities and gross motor skills was observed, and she even started riding a bicycle on her own.

After 42 neurofeedback sessions we recorded and analyzed her EEG again.

The patient's raw EEG was clear of any epileptiform discharges. There were zero events detected over 10 minutes of recording on C3. There were a few changes with eyes closed power spectra compared with norms for absolute power: mostly a lack of beta-power in the frontal lobe. These changes can be explained by her quitting the anticonvulsant medication (Figures 9,10).

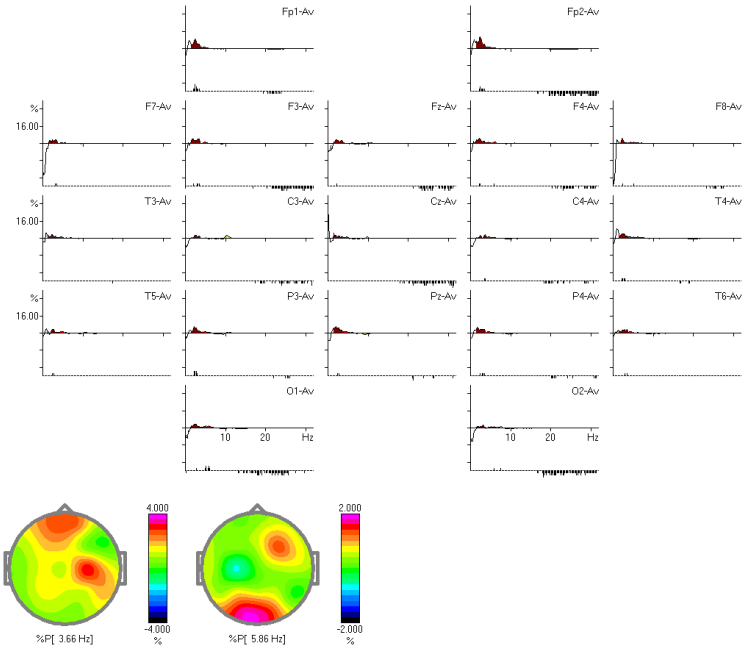


Figure 9: Graphs of EEG power spectra (eyes open) compared to a normative database, after 42 neurofeedback sessions.

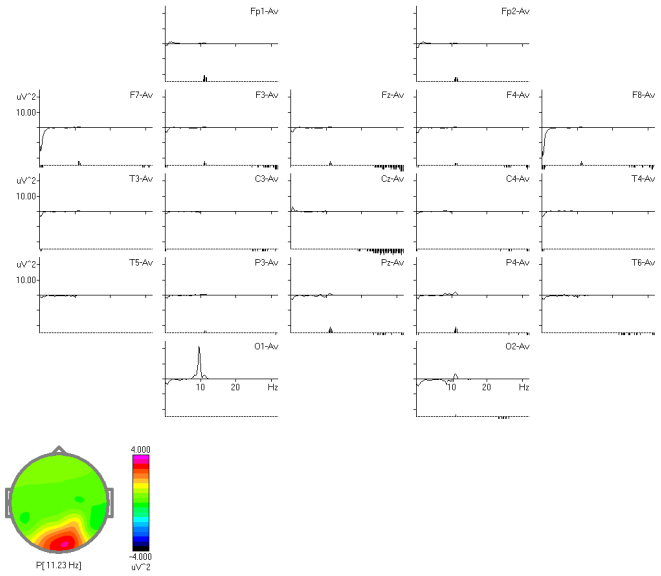
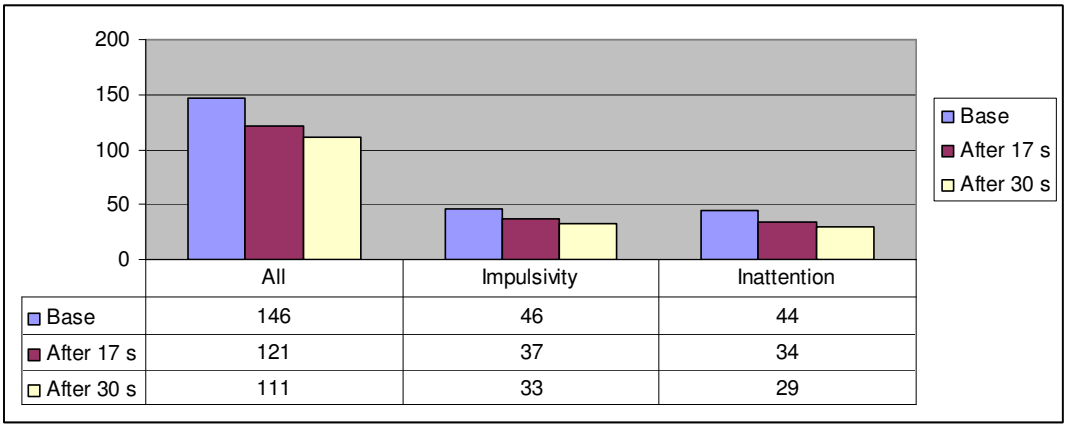


Figure 10: Graphs of EEG power spectra (eyes closed) compared to a normative database, after 42 neurofeedback sessions.

The Conner's' parent questionnaires were taken in three different points in time: before treatment, after 17 training sessions and after 30 sessions (Figure 11). Her total rate score decreased during the course of treatment (from 146 on the first assessment to 111 after 30 sessions). Attentiveness improved by 34% and impulsivity improved by 28%.



	All	Impulsivity	Inattention
Base	146	46	44
After 17 s	121	37	34
After 30 s	111	33	29

Figure 11: Chart of Conners' parent questionnaires before treatment, after 17 sessions and after 30 sessions. The total rate score decreased during treatment (from 146 on the first assessment to 111 after 30 sessions). Attentiveness improved by 34% and impulsivity improved by 28%.

Case 2 report

The patient, H.Y.,10 year old girl, was diagnosed with epilepsy and suffered from developmental delay, cognitive dysfunction, impulsivity and wild behavior.

Seizures were first observed at age 6. There were many observed instances of her disconnecting and gazing left-wards, without limb movement. The patient was treated with anti-convulsant drugs, and it seemed like her epilepsy was controlled. At the age of 8.5 there were night-events during sleep, which were accompanied by grinding of teeth, trismus, tremors all over the body and loss of consciousness for a few minutes. Despite the drug treatment, the seizure events repeated every night. The EEG chart that was done at the hospital during wakefulness, nap and sleep after sleep deprivation showed electrical status epilepticus during sleep (ESES). ESES is a typical childhood process of generalization of paroxysmal activity.

A clinical EEG was conducted in our clinic at intake for 10 minuets, with eyes open and eyes closed (Figure 12). Her parents were asked to fill a Conners' parent questionnaire before intake and during subsequent assessments.

EEGs were recorded from 19 scalp sites. The electrodes were applied according to the International 10-20 system. The EEG was recorded in the average montage. Visual inspection of raw EEG was made in order to search for paroxysmal patterns that pop out of the background EEG. The EEG was analyzed with EEG/QEEG software (WinEEG). The analysis consisted 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.

We used the automatic spike detection, analysis and average spike calculation system. This resulted in evidence of significant paroxysmal activity consistent with the spike & wave pattern in medial frontal gyrus regions F7 and Fz (Figure 13). There were significant 121 events detected over 10 minutes of recording. We used low resolution brain electromagnetic tomography (Loreta) analysis to locate the source distribution. It was located by Loreta in the middle frontal gyrus, Brodman area 9 (Figure 14).

3) Fast-Fourier Transformation (FFT) of the corrected EEG for extracting EEG power. We computed EEG with eyes open and eyes closed and compared to age-regressed, normative database (HBI) for absolute power (Figures 15 ,16) . There were obvious power-excesses of high beta-waves (25-30Hz) on frontal-central-parietal areas and of Theta waves (2-6 Hz) over central areas.

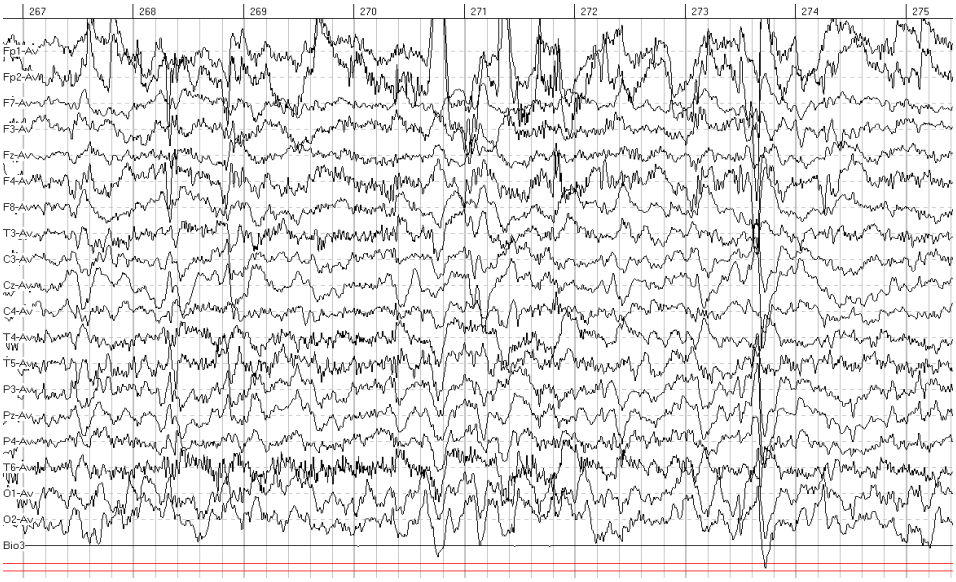


Figure 12: complexes of spike & wave on the patient row EEG (eyes open), at intake.

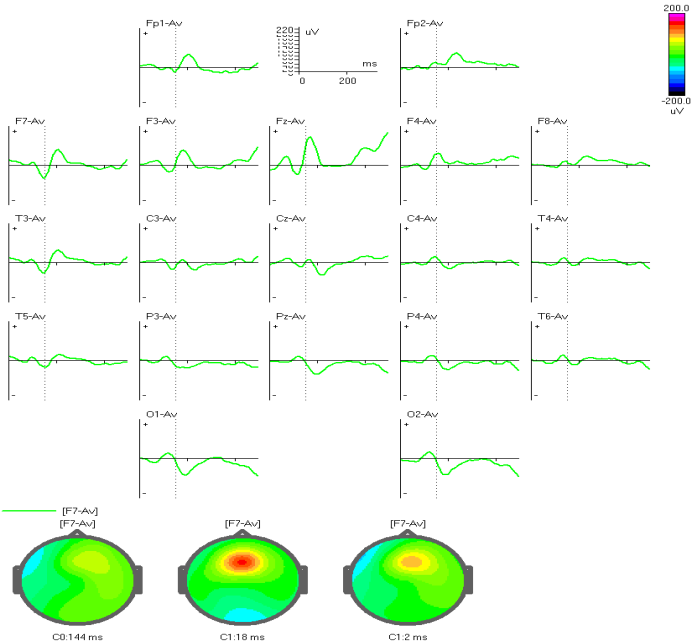


Figure 13: Spike & wave pattern detect by the automatic spike averaging system on Fz at intake.

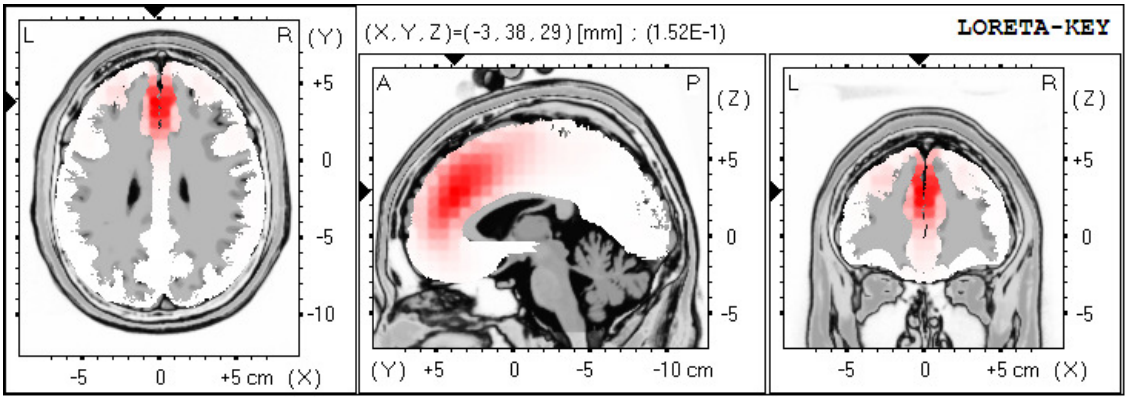


Figure 14: Loreta analysis for source distribution indicates source location in the middle frontal gyrus, Brodman area 9.

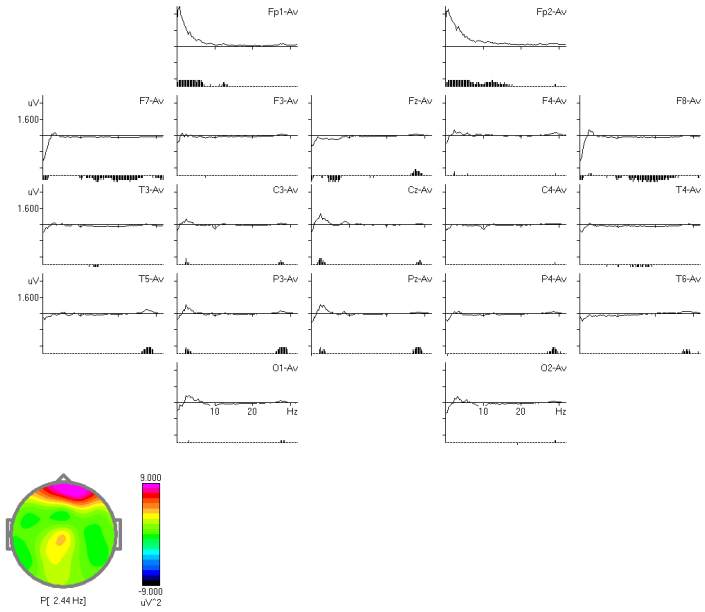


Figure 15: Graphs of EEG power spectra (eyes open) compared to a normative database, at intake.

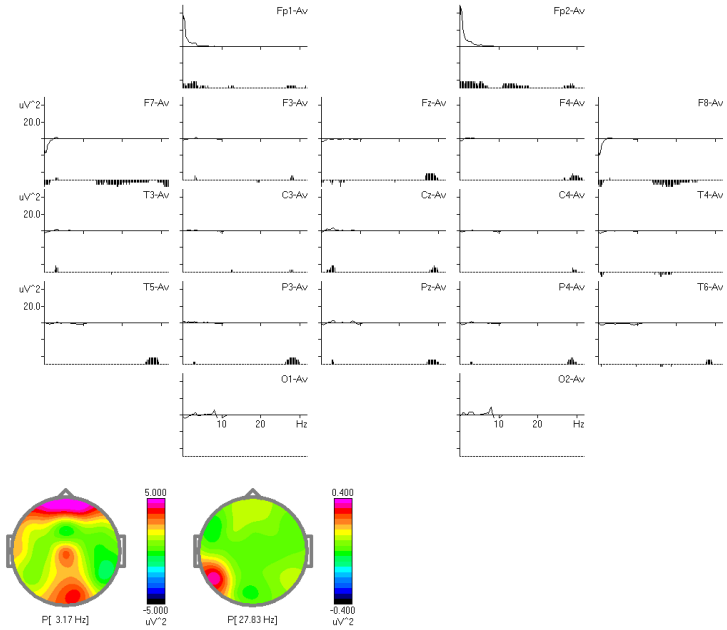


Figure 16: Graphs of EEG power spectra (eyes closed) compared to a normative database, at intake.

The patient underwent 37 guided neurofeedback training sessions. The active electrode was on Cz. We down-trained both 2-6Hz and 20-25Hz. During the training, the patient was still receiving anticonvulsant medication as before. The parents reported a significant improvement in her behavior at school and at home.

After 37 neurofeedback sessions we recorded and analyzed her EEG again.

There was only 1 event detected over 10 minutes of recording on Fz (Figure 17), and a significant decrease in theta-power in the eyes-open and eyes-closed power spectra, compared with norms, for absolute power (Figures 18, 19).

Analysis of the conners' parent questionnaires taken before treatment, after 20 neurofeedback sessions and after 35 sessions shows a 26% improvement in attentiveness and a 44% improvement in impulsivity (Figure 20).

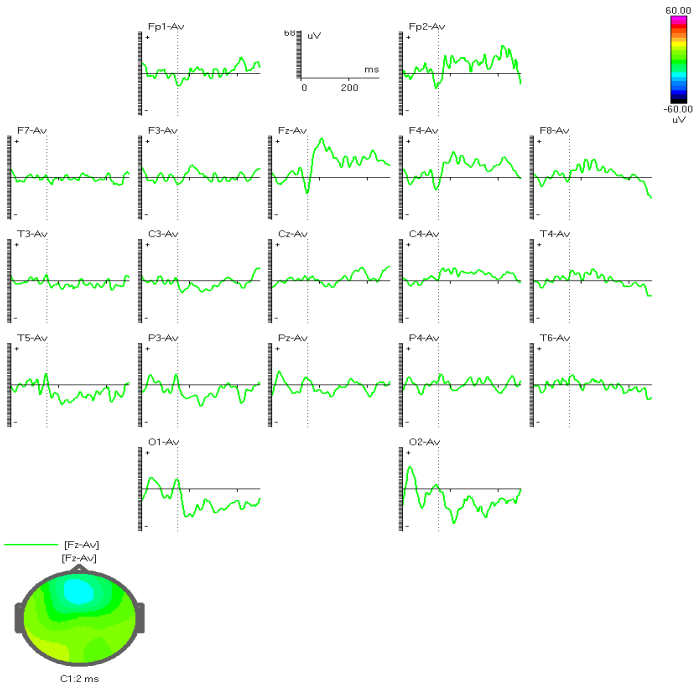


Figure 17: Spike & wave pattern detect by the automatic spike averaging system on Fz, after 37 neurofeedback sessions.

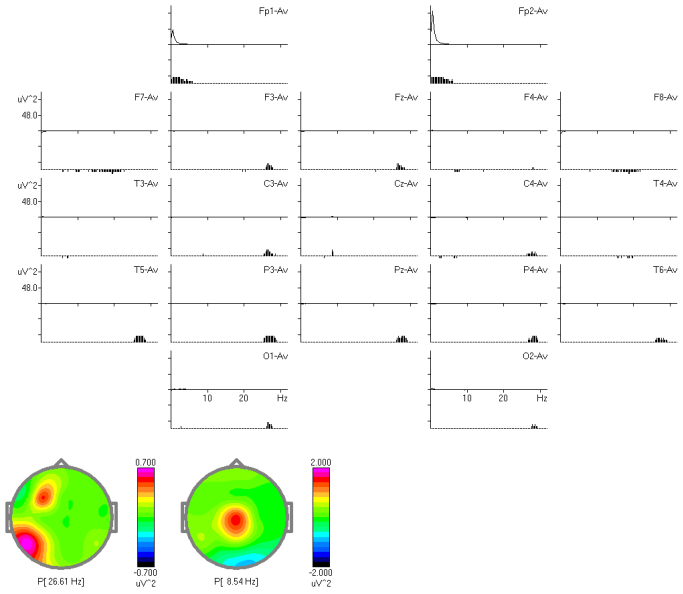


Figure 18: Graphs of EEG power spectra (eyes open) compared to a normative database, after 37 neurofeedback sessions.

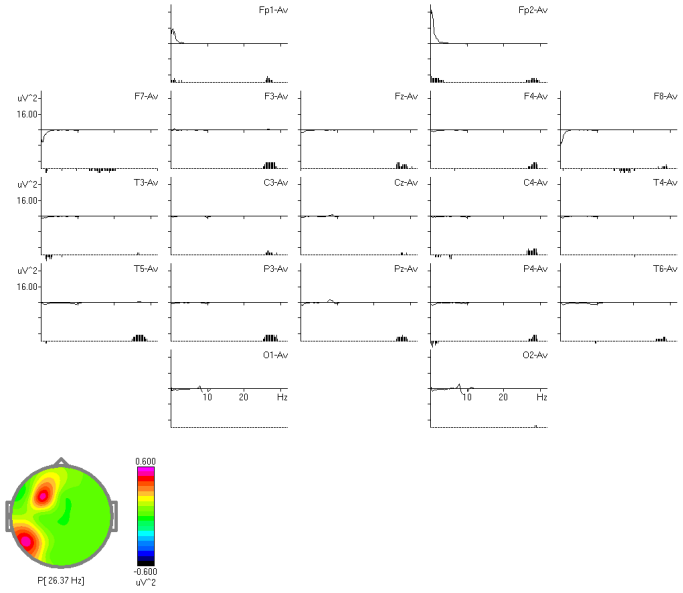
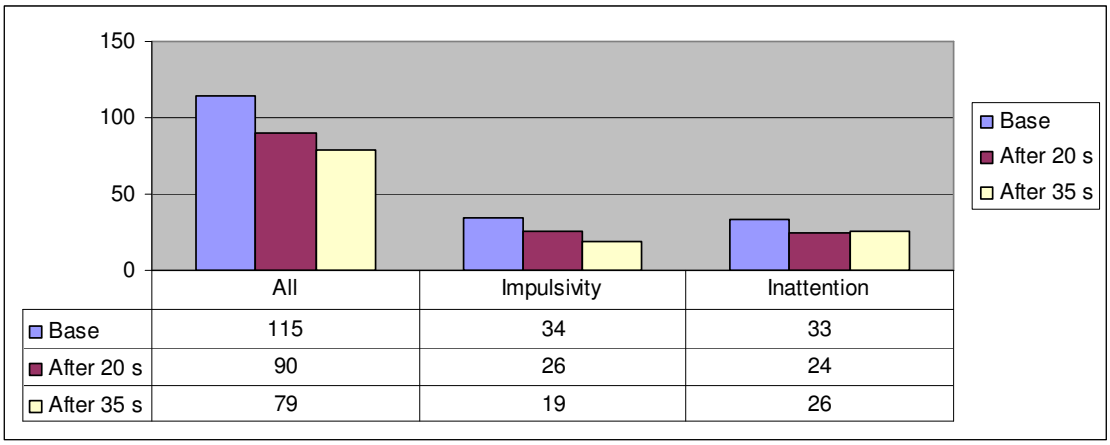


Figure 19: Graphs of EEG power spectra (eyes open) compared to a normative database, after 37 neurofeedback sessions.



	All	Impulsivity	Inattention
Base	115	34	33
After 20 s	90	26	24
After 35 s	79	19	26

Figure 20: Chart of Conners' parent questionnaires before treatment, after 20 sessions and after 35 sessions. The total rate score decreased during the treatment (from 115 on the first assessment to 90 after 35 sessions). Attentiveness improved by 26% and impulsivity improved by 44%.

Summary:

Based on our previous experience and according to previous studies published, we hypothesized that QEEG guided neurofeedback can help regulate EEG and help achieve complete cessation of seizures for patients with epilepsy that was not controlled by medications. This article reviewed two cases of epileptic patients:

1. A 7.5 year old female patient, diagnosed with benign Rolandic epilepsy with cognitive dysfunction, verbal apraxia of speech and gross motor dysfunction. Although she was treated with anticonvulsant medication, she could not function independently at school and at home.

We used EEG/QEEG and parents questionnaires to follow her progress before and during the training sessions. The baseline EEG that we took before starting the neurofeedback treatment showed significant paroxysmal activity consistent with the spike & wave pattern in centro-temporal regions, mostly on the left side (Figure 2). There were significant 257 events detected on C3 over 10 minutes of recording. The second EEG assessment taken after 17 sessions of neurofeedback training detected 115 spike & wave events over 10 minutes of recording on C3. The third EEG, taken after another 25 (total of 42) training sessions, revealed zero events of spike & wave on C3.

Over the course of neurofeedback the patient improved significantly in language understanding, speech and gross motor skills. She speaks fluently and rides a bicycle. There is an improvement in her attention and impulsivity but her cognitive functions remain relatively low for her age.

2. A 10 year old femael patient, diagnosed with epilepsy and developmental delay, cognitive dysfunction and wild, impulsive behavior.

Although she was treated with anticonvulsant medication, the seizures repeated every night during sleep and her behavior was uncontrollable at school or at home.

We used EEG/QEEG and parents questionnaires to follow her progress before and during the training sessions. The baseline EEG that we took before starting the training sessions showed significant paroxysmal activity consistent with the spike & wave pattern in frontal regions, mostly on Fz (Figure 13). There were significant 121 events detected on Fz over 10 minutes of recording. The second EEG assessment taken after 37 sessions of neurofeedback training detected 1 spike & wave event over 10 minutes of recording on Fz.

Over the course of neurofeedback the patient's behavior at school and at home improved significantly.

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