A Physical Analysis of an Epileptic State EEG. A Possible Way to Treat the Epilepsy Seizure

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Keywords
EEG; Epilepsy; Seizure; Delta waves; Harmonic analysis; Multisim

1. Introduction
The existence of electric voltage fluctuations in the human brain was first proved by H. Berger in 1924. He used an amplifying machine, that he called electroencephalograph to realize the first recording of the electric activity of the human brain: electroencephalogram (EEG), measuring tiny changes in electrical flow between a pair of electrodes placed on a patient’s skull. Then, he performed several scientific papers on it, treating the spontaneous activity, the evoked potentials and the bioelectric events produced by single neurons. In particular, he showed that this electric activity change according to the functional status of the brain, such as in sleep, anesthesia, hypoxia (lack of oxygen) and in some nervous diseases, such as epilepsy [1].

Epilepsy remains one of the most common neurologic disorders affecting both adults and children alike. It is characterized as a seizure disorder with electrophysiologic abnormalities and is most often diagnosed by electroencephalography (EEG) [2].

Abstract

Background and Purpose
An electroencephalogram (EEG) is a test that measures and records the electrical activity of our brain. Moreover, it is the most useful and important test in confirming a diagnosis of epilepsy. The purpose of this work is to perform a deep physical analysis of an epilepsy EEG signal in view to contribute to finding a way to treat this disease.

Methods
Firstly, we present a concise harmonic analysis of EEG for a sleeping adult and for a person at an epilepsy-“petit mal” state. Secondly, we realize an electronic simulation for the two cases and give their equivalent electronic circuits.

Results
Comparing between the two studied EEG signals, we firstly find that the frequency increases enormously in the epileptic state by a factor greater than 4 than in the healthy state. Secondly, the amplitudes remain globally stable in the two cases. Thirdly, during each cycle in the epileptic state, it appears an additional peak, in comparison with the healthy case.

Conclusions
One concludes that the cerebral activity during the epileptic state is disturbed by means of some disorderly discharging of the brain’s nerve cells. These are the so-called seizures. They occurrence is due to a purely neurobiological reason, such as a disturbance of motor, sensory or mental function. Basing upon these results, and in view to treat this disease, we propose to equip the patient with an electronic device in order to re-establish his crisis state by restoring it to normal.
The main aim of this work is, first, to perform a harmonic analysis of the spikes of an epilepsy-petit mal (or absence seizure) as well as of the Delta waves of a sleeping adult, and to use Multisim for an electronic circuit simulation corresponding to the two EEG signals. Comparing between the two results, we try to give some interpretation to the seizures that occur in the epilepsy case and we propose a possible way to treat this pathology using an electronic equipment.

2. Electroencephalography

Firstly, let us recall some basic notions concerning electroencephalography. From an EEG signal, it is possible to differentiate between the Alpha (α), Beta (β), Delta (δ) and Theta (θ) waves as well as the spikes of an absence seizure [3].

Figure 1: EEG waves

The frequency band of the Alpha waves is 8-13 Hz and can be measured from the occipital region in an awake person with closed eyes. The frequency range of the Beta waves is of 13-30 Hz and these are detectable over the parietal and frontal lobes. The Delta waves have the frequency spectrum of 0.5-4 Hz and are detectable in infants and sleeping adults. The Theta waves have the frequency range of 4-8 Hz and are obtained from children and sleeping adults.

Although in principle the EEG can be performed, the complexity of brain structure and its electrophysiological features have thus far precluded the evaluation of the source function. It follows that the quantitative study of the EEG differs, for instance, from that of the electrocardiogram (ECG), in which it is possible to evaluate the source function. Under these conditions, the quantitative EEG is based on a statistical study, whereas the clinical EEG is largely empirical.

Figure 2: Frequency spectrum of normal EEG

The EEG signal is closely tied to the level of consciousness of the person. As the activity increases, the EEG shifts to higher dominating frequency and lower amplitude. When the eyes are closed, the Alpha waves begin to dominate the EEG. When the person falls asleep, the dominant EEG frequency decreases. In a certain phase of sleep, rapid eye movement called (REM) sleep, the person dreams and has active movements of the eyes, which can be seen as a characteristic EEG signal. In deep sleep, the EEG has large and slow deflections called Delta waves. No cerebral activity can be detected from a patient with complete cerebral death.

Figure 3: EEG activity depends on the level of consciousness
For further technical details on the EEG, see references [3, 4].

3. Epilepsy Seizures

In other respects, epilepsy is a neurological aspect that affects the nervous system. It is is characterized by recurrent seizures that often occur in a sudden and spontaneous manner, and is the third most common neurological disorder in the world, with 0.5-1% of the human population suffering from the disease.

Seizures are manifestations of abnormal, hypersynchronous discharges of a large number of brain cells (cortical neurons) that are activated at the same time, resulting of a temporary disturbance of motor, sensory or mental function. In fact, they are paroxysmal neurological symptoms caused by episodic and pathologic neuronal discharging, that look like electrical storms in the brain. The official definition of a seizure retained by International League Against Epilepsy (ILAE) is “a transient occurrence of signs and/or symptoms due to an abnormal excessive or synchronous neuronal activity in the brain.”

Seizures have a large number of associated signs/symptoms that are dependent upon its anatomical origin and subsequent spread. The underlying causes of “epilepsy” (recurrent seizures) are many and include genetic factors, congenital and/or developmental anomalies, infections, trauma, and tumors. In fact, the nature of the seizures depends on many factors, such as the person’s age, the sleep-wake cycle, prior injuries to the brain, genetic aspects, medications, and many others.

4. Classification of Seizure types

There are several types of seizures, depending primarily on what part of the brain is concerned. Epilepsy is usually diagnosed after a person has had at least two seizures that were not caused by some known medical conditions like alcohol withdrawal or extremely low blood sugar. According to the (ILAE), epilepsy can sometimes be diagnosed after one seizure, if a person has a condition that places him at high risk for having another one.

Classifying seizures into different types helps guide further testing, treatment, and prognosis or outlook. Then, it is very important to use a common language for seizure classification since it will make easier the communication among epileptologists and influenced both basic and clinical research on this neuropathology [5].

In this context, several classifications of epilepsy syndromes and epilepsies have been proposed since 1970; the most recent one was published in 1989 by the ILAE. Since 1997, the ILAE Task Force on Classification and Terminology has been evaluating this classification and some modifications have been introduced. Although the 1989 classification needs to be updated, it has been widely accepted and is universally employed [6].

Historically, for decades, the most common words to describe seizures were grand mal and petit mal. Although the medical meaning of these terms was fairly precise, some people often used them loosely when referring to any big or little seizure.

Since 1981, the following terms were used to describe different types of seizures [7]:

✓ Partial seizures: seizures starting in one area or side of the brain. This type of seizure was defined by whether a person was aware or conscious during the event. It is divided into:

> Simple partial seizures: Person is aware of what happens during the seizure.

> Complex partial seizures: Person has some impaired awareness during the seizure. They may be confused, partially aware, or not aware of anything during a seizure.

✓ Generalized seizures: Seizures starting in both sides of the brain at the same time. This type was divided into:

> Tonic-clonic seizures: At one time called “grand mal” seizures, where patients lose consciousness, their muscles stiffen, and they exhibit jerking movements,

> Absence seizures: Once called “petit mal” seizures, where the patient has lapses in awareness and may appear to be staring,

> Atonic seizures: Characterized by an abrupt loss of muscle tone,

> Tonic seizures: Stiffening,

> Clonic seizures: Repeated jerking,

> Myoclonic seizures: Brief shock-like jerks of a muscle...
or several muscles.

✓ **Unclassified seizures:** They include all those that didn’t fit into the other categories or those for which it was unclear where in the brain they began.

This old classification worked for many years but did not take into account many types of seizures. After that, the ILAE has approved a new and more complete version [8-10]. The main changes that were introduced concern the naming of these types of seizures. Hence, seizures formerly known as “simple partial” seizures are now called “focal aware” seizures and “complex partial” seizures are now called “focal impaired awareness” seizures, (Table 1).

**Table 1: ILAE 2017 Classification of Seizure Types basic Version**

![Seizure Types Diagram]

The new basic seizure classification is based on 3 main factors [5]:

A. The seizure onset

B. Level of awareness,

C. Other features of seizures: Within this category, we find:

- **Generalized motor seizure:** Corresponding to the grand-mal seizure, the generalized tonic-clonic seizure term is still used to describe seizures with stiffening (tonic) and jerking (clonic). Furthermore, there are many other forms of the generalized motor seizure, and some new terms have been added.

- **Generalized non-motor seizure:** These are previously called absence seizures that correspond to the old term petit mal. These seizures involve brief changes in awareness, staring, and some may have automatic or repeated movements.

5. **Absence Seizure**

Following the new ILAE classification of seizure types, the petit mal seizures, or absence seizures, are classified as (idiopathic and symptomatic) non-motor generalized seizures [11]. They were first described by Poupart in 1705, and later by Tissot in 1770 who use the term petit access. The term absence was first used by Calmeil [12] in 1824 and it was reported as pyknolepsy (“heaped up, closely packed, aggregated attacks”) in the early 20th century. In 1935, Gibbs, Davis, and Lennox described the association of impaired consciousness and 3-Hz spike-and-slow-wave complexes on EEGs [13]. Absence seizures are common within many forms of pediatric and adult epilepsies and span all the ages, in particular, they are the hallmark seizure type in the
following idiopathic generalized epilepsy syndromes: childhood absence epilepsy (pyknolepsy), juvenile absence epilepsy and juvenile myoclonic epilepsy (impulsive petit mal). Generally, absence (petit mal) seizures are brief episodes, usually lasting 3–20 seconds, of staring with impairment of awareness and responsiveness. They begin and end suddenly, without warning before the seizure, and immediately afterward the patient is aware and attentive. Although absence seizures are known more than three centuries ago, they continue to be subject of new advances with its classification, pathophysiology, genetics, treatment, prognosis, and associated co-morbidities. These seizures are called typical absence seizures and are usually associated with generalized 3-4 Hz spike-and-slow-wave complexes on EEG [14].

In another context, it is well known that the Delta waves have the frequency spectrum of 0.5-4 Hz and are detectable in infants and sleeping adults. This is precisely why we have chosen in this work to compare these two types of EEG.

If the EEG is recorded at the moment of crisis, the obtained data should be very precious to precise the type of the crisis, their departure point, their diffusion, etc…. Between two crisis, the EEG graph may be normal, but also may bring up some epilepsy signals.

### 6. Control of seizures-Medications and Neurostimulations

The seizures in epilepsy may be related to a brain injury or a family tendency, but most of the time the cause is unknown. The term epilepsy says nothing about the cause of the person’s seizures, their types or how severe they are. Experimentally, the localization of the epilepsy source is more precise using some imaging techniques like Positron Emission Tomography (PET) or Magnetic Resonance Imaging (MRI) [15-17].

To control this neuropathology, many types of medication and non-pharmacological therapies have been introduced during the last decades. Nevertheless, in 20 - 40% of epileptic patients, medications alone are unable to adequately control seizures and are therefore diagnosed with drug-resistant epilepsy [18]. For 60% of patients with drug-resistant epilepsy, surgery can significantly reduce or eliminate seizures [19-20]. Unfortunately, despite the development of new antiepileptic drugs (AEDs), there are still many patients suffering from persistent seizures that impair their daily living [21] (15-40%). To improve the quality of life for these remaining patients, both epileptologists have developed new therapies based on neuromodulation [22] (or neurostimulation).

Among the non-pharmacologic therapies available, neuro-stimulation has been considered as a treatment for epilepsy for almost 20 years in the form of vagus nerve stimulation (VNS). It is actually the most accessible and best studied therapy. In fact, it has been approved since 1994 in Europe and since 1997 in the United States. Its efficacy may be compared with that of a newer AED [23]. Two new types of neurostimulation are actually being developed: Deep brain stimulation (DBS) and Responsive Neurostimulator System (RNS). The first one is a therapy that has been studied in different forms, but only a double-blind study of bilateral stimulation of the anterior nucleus of the thalamus [24], has been accepted as showing efficacy and is now approved as a therapy for epilepsy in Europe and not yet in the United States. The second therapy, the so-called closed-loop system, is also actually under development, and a doubleblind study has also been completed [25], but approval for use in refractory focal onset epilepsy patients is still pending.

VNS is an invasive therapy, developed by Cyberonics, that is designed to stimulate the peripheral vagus nerve, in particular the left vagus nerve, which is composed of 80% afferent fibers [26]. Through animal experiments, it has been shown that the stimulus converge on the nucleus of the tractus solitarius, which then converges on the locus coeruleus. Inhibition of noradrenalin release from the Locus Coruleus inhibits the anti-seizure response in animals [27]. Animal experiments and research in humans treated with VNS have included electrophysiological studies (EEG, EMG, Evoked Potentials), as well as functional anatomic brain imaging studies [28-30] (PET, SPECT, fMRI, c-fos, densitometry). For instance, PET and fMRI studies [31], have shown that peripheral stimulation of the vagus nerve causes increases in brain metabolism in different areas of the brain (in particular, thalamus, cerebellum, orbitofrontal cortex, limbic system, hypothalamus, and medulla). Several hypotheses have emerged through the years concerning the mechanism of action of VNS. Among them consists to say that VNS could simply be a peripheral variation of thalamic stimulation, or also that peripheral VNS may have a network-modifying influence in the brain, changing synaptic connections. However, the precise mechanism of action of VNS, and how it reduces seizures, is still elusive and not well understood. Nevertheless, this therapy is found to be generally as effective as antiepileptic drugs for...
select seizure populations and serious complications are uncommon.

Concerning the DBS therapy, developed by Medtronic, it was conceived for delivering electrical stimulation to areas in the brain that control movement and muscle function for movement disorders. Hence, the rational for DBS of the thalamus is that this latter can serve as a relay station through thalamocortical networks, thereby inhibiting or disrupting rhythmic depolarization signals from spreading and causing overt seizures. The stimulation of the *anterior nucleus of the thalamus* (ANT) that is implicated in seizure spread has been shown to reduce synchrony and increase inhibition in hippocampus or neocortex [32]. From animal experiments, it has been shown that low-frequency stimulation of ANT generates recruiting rhythms and synchronizes the pattern of EEG activity, making the cortex more susceptible to seizures, and that high-frequency stimulation of ANT induces EEG desynchronization which could produce an anti-seizure effect [33].

Unlike VNS and DBS therapies, which deliver continuous stimulation, a closed-loop neuromodulation system (RNS) only produces stimulation when it detects the starts of seizure activity. This individualized treatment, whereby a detecting electrode is placed near the seizure focus as well as a stimulating electrode, is designed to work through seizure detection to either reduce the risk of having a seizure altogether or stops seizures from spreading to other parts of the brain [34], exactly like a pacemaker does to stop abnormal heart rhythms. In this case, the seizure focus or foci must always be identified before implantation of the device. Then, the system will provide real-time electrographic analysis and automatic delivery of a responsive stimulation to a signal that is detected as being epileptiform [35].

Finally, it is important to notice that the exact mechanism of action of a number of the AEDs is also not totally understood, and new is still an experimental and theoretical research topic, even concerning the oldest antiepileptic drugs.

7. Methods

In this work, we have considered the EEG cerebral waves of a sleeping adult and spikes of an absence seizure given in Figure 1. Our main aim is to perform a harmonic analysis of one cycle of each type. Our approach follows the following steps:

7.1. Digitalization

For this purpose, we use the software *FindGraph* which is a good tool for digitalizing curves given by pictures or designs. For this purpose, we first consider an amplitude averaged cycle for the two types of EEG, and then we reproduce them as paint images.

Then, using *FindGraph*, we obtain the following digitalized graphs, (Figure 4a and 4b). Here, we have considered the minimal and maximal values of $x$ and $y$ for the EEG cycles respectively as follows:

$$\begin{align*}
(X_{min} = 0 \text{ cm}, X_{max} = 2.7 \text{ cm}), & \quad (Y_{min} = -0.8 \text{ cm}, Y_{max} = 0.9 \text{ cm}) \\
(X_{min} = 0 \text{ cm}, X_{min} = 0.6 \text{ cm}), & \quad (Y_{min} = -0.3 \text{ cm}, Y_{max} = 1 \text{ cm})
\end{align*}$$

Then, the digitalization is recorded into a data files of $(x,y)$ points. For the first EEG cycle, we have 1268 points, and for the second, we have 1858 points.

Figure 4: Digitalized EEG cycles a: For a sleeping adult (left); b: an epilepsy crisis (right)
7.2. Plotting

Then, we make use of the software Origin for firstly interpolating these digitalized graphs and secondly plotting the resulting data. The first digitalized EEG signal is interpolated into 2778 points, with a period T=1.3890s and a time step of 0.0005s. The second one is interpolated into 3157 points with a period T=0.3157s and a time step of 0.0001s. Then, we plot these data and obtain the following graphs, (Figure 5 and 6)

Figure 5: Plotted EEG averaged cycle graph for a sleeping adult

Figure 6: Plotted EEG averaged cycle graph for an epilepsy crisis

7.3. Harmonic Analysis

Fourier analysis is an important tool in the area of signal analysis and processing. With its help, we can determine which harmonic signals, with different amplitudes, frequencies and phases, every periodic signal consists of. Explicitly, each periodic function f(t) with a period $T_0$ (or frequency $\nu_0$) can be expanded into a Fourier series (Fourier theorem) such that [36]:

$$f(t) = \frac{a_0}{2} + \sum_{k=1}^{\infty} \left[ a_k \cos \left( \frac{2\pi k t}{T_0} \right) + b_k \sin \left( \frac{2\pi k t}{T_0} \right) \right]$$

or equivalently, as follows:

$$f(t) = A_0 + \sum_{k=1}^{\infty} A_k \cos \left( \frac{2\pi k t}{T_0} + \varphi_k \right)$$

where the coefficients $a_0$, $a_k$, $b_k$, $A_0$, $A_k$ and $\varphi_k$ can be determined by the following respective rules:

$$a_0 = \frac{1}{T_0} \int_{-T_0/2}^{T_0/2} f(t) dt$$
$$a_k = \frac{2}{T_0} \int_{-T_0/2}^{T_0/2} f(t) \cos \left( \frac{2\pi k t}{T_0} \right) dt$$
$$b_k = \frac{2}{T_0} \int_{-T_0/2}^{T_0/2} f(t) \sin \left( \frac{2\pi k t}{T_0} \right) dt$$

And

$$A_k = \sqrt{a_k^2 + b_k^2}, \quad \varphi_k = \arctan \left( \frac{a_k}{b_k} \right)$$

Henceforth, a periodic signal f(t) with period $T_0$ can be represented by a fix component measuring its average over a cycle and an infinite sum of harmonic signals whose frequencies are integral multiples of $\omega_0=2\pi/T_0=2\pi\nu_0$. The harmonic signal with frequency $k\omega_0$ is called $k$th harmonic of the fundamental oscillation ($k=1$) with the fundamental frequency $\omega_0$, and the infinite sum added to the fix component must reproduce exactly the original periodic signal. In the following, we will focus our attention on the analysis of our two EEG voltage signals $U(t)$, (Figure 5 and 6).

We proceed as follows:

Firstly, we calculate on Origin the coefficients $a_0$, $a_k$, and $b_k$ for the two digitalized EEG signals and using the corresponding time step $(t_{i+1}-t_i)$ and the number of interpolation points in the calculus of the discreet area sum for each harmonic of rank $k$, where $k=1, \ldots, 50$, i.e. the fifty first harmonics, and $i=1, \ldots, 2778$ and $T_0=1,3890s$ for the sleeping adult EEG and $i=1, \ldots, 3157$ and $T_0=0,3157s$ for the epilepsy EEG.

Secondly, we deduce the values of the coefficients $A_0$, $A_k$ and $\varphi_k$, for each harmonic of rank $k$, using Mathematica.
Thirdly, we add to the fix component the first harmonic and we use the above software to plot the signal. Then, we add the second harmonic and we plot the resulting signal and so on to the 50th harmonic, by means of the following instructions, for the two considered types of EEG signals.

Fourthly, at each time, we juxtapose the obtained signal with the corresponding digitalized signal, (Figure 5 and 6), and we calculate the correlation coefficient. Hereunder, we have tabulated these correlation coefficients for the two types of EEG signals for only the first 25 harmonics in the Table 2. Then, we draw the respective graphs giving the evolution of the correlation coefficient with the number of added harmonics, (Figure 7 and 8).

Figure 7: Evolution of the correlation coefficient with the number of added harmonics for the case of sleeping adult EEG signal

![Figure 7](image)

Figure 8: Evolution of the correlation coefficient with the number of added harmonics for the case of epilepsy EEG signal

![Figure 8](image)
8. Results

We decide to limit ourselves to the six first harmonics since, at this stage, the correlation coefficient is greater than 99%, and the juxtaposition of the digitalized EEG signal and the plotted one are given by Figure 9 and 10 respectively:

**Figure 9:** Juxtaposition of the digitalized EEG signal and the Plotted one with 6 harmonics for the case of sleepy adult EEG signal

![Figure 9](image_url)

**Figure 10:** Juxtaposition of the digitalized EEG signal and the Plotted one with 6 harmonics for the case of epilepsy EEG signal

![Figure 10](image_url)

Hereunder, we give a modelling of $|A_k|^2$, the square of the amplitude $A_k$, in function of the rank k of the harmonic for the two EEG examples respectively. For this purpose, we choose the allometric fitting, (Figure 11 and 12):

<table>
<thead>
<tr>
<th>Number of added harmonics</th>
<th>Correlation coefficient (Sleeping adult)</th>
<th>Correlation coefficients (Epilepsy)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>0.988</td>
<td>0.86164</td>
</tr>
<tr>
<td>2</td>
<td>0.99752</td>
<td>0.93765</td>
</tr>
<tr>
<td>3</td>
<td>0.99789</td>
<td>0.95983</td>
</tr>
<tr>
<td>4</td>
<td>0.99895</td>
<td>0.98162</td>
</tr>
<tr>
<td>5</td>
<td>0.99952</td>
<td>0.98818</td>
</tr>
<tr>
<td>6</td>
<td>0.99983</td>
<td>0.99322</td>
</tr>
<tr>
<td>7</td>
<td>0.99984</td>
<td>0.99603</td>
</tr>
<tr>
<td>8</td>
<td>0.99984</td>
<td>0.99636</td>
</tr>
<tr>
<td>9</td>
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</tr>
<tr>
<td>12</td>
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</tr>
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<tr>
<td>16</td>
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<td>17</td>
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<td>0.99881</td>
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<tr>
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<td>0.99996</td>
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<tr>
<td>20</td>
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<tr>
<td>21</td>
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<tr>
<td>22</td>
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<td>0.99932</td>
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<td>25</td>
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<td>0.99934</td>
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</table>
The idea of studying the quantity $|A_k|^2$ is guided by the fact that it is the main physical quantity we can deal with and that it gives us the occurrence probability of the $k^{th}$ harmonic for the two cases. We notice that effectively the six first harmonics have the most important occurrence probability and that their contribution in the construction of the corresponding signal is the most important.

In the last part of this work, we present a Multisim simulation of the two voltage signals using the fix component and only the six first harmonics. The fundamental characteristics of each component of the electronic circuit are given by the already calculated properties of its corresponding harmonic of rank $k$, namely: The amplitude $A_k$, the pulsation $\omega_k$ and the phase $\phi_k$. The two obtained circuits are given by Figure 13 and 14 respectively.

We expect from these two electronic circuits that they reproduce the two EEG voltage signals corresponding to a sleeping adult and an absence seizure. In fact, using the oscilloscope option of the Multisim software, we get nearly the same signals as given in Figure 1 (See Figure 15 and 16).
**Figure 13:** Multisim circuit corresponding to the sleeping adult EEG signal using the first six harmonics and the fix component

![Multisim circuit for sleeping adult EEG signal](image13)

**Figure 14:** Multisim circuit corresponding to the epilepsy EEG signal using the first six harmonics and the fix component

![Multisim circuit for epilepsy EEG signal](image14)

**Figure 15:** The signal obtained by means of Multisim simulation of the sleeping adult EEG signal using the first six harmonics and the fix component

![Signal obtained by Multisim simulation](image15)
9. Discussion

The classification of seizure types is essentially observational, based on how the seizures look and sometimes what the EEG pattern or other testing tells us. Even the efforts to make a classification that corresponds to the underlying brain circuitry problem causing the different seizure types, experts still don’t know enough about this. The ILAE considers epilepsy a disease. About few years ago, the ILAE adopted a new definition of epilepsy in which the disease is considered to be resolved if a patient has been seizure-free for 10 years, during the last 5 of which the patient was off all seizure medicines.

In fact, the EEG is most useful for classifying the seizure type and, in many cases, the epilepsy syndrome. A normal EEG does not exclude the diagnosis of epilepsy. The EEG is only a very brief time sample of the patient’s brain electrical activity and will miss intermittent or transient abnormalities. In evaluating a patient suspected to have had a seizure, an EEG showing interictal (between seizures) epileptiform activity provides corroborating evidence, but is not proof, unless the patient has a seizure during the EEG (in which case the epileptiform activity is ictal rather than interictal).

Over the past few decades, neuromodulation for epilepsy has grown dramatically. In fact, approval has already been given in Europe for deep brain stimulation and newer forms of vagus nerve stimulation for the treatment of drug-resistant epilepsy. Other types of neuromodulation that have been explored include trigeminal nerve stimulation (TNS) and deep brain stimulation (DBS).

In our work, the comparison between the two studied EEG signals, gives us the following main differences:

- The frequency increases enormously in the epileptic state by a factor greater than 4 than a normal state.
- The amplitudes remain globally stable.
- During each cycle in the epileptic state, it appears an additional peak (See Figure 6). This reflects that the cerebral activity at this moment is disturbed by means of some disorderly discharging of the brain’s nerve cells. These are the so-called seizures. There many types of seizures depending essentially on which part of the brain that is really concerned. They occurrence is due to a purely neurobiological reason, as consequences of a disturbance of motor, sensory or mental function.

Epilepsy remains one of the most common neurologic disorders affecting both adults and children alike. Millions of individuals in the world have a diagnosis...
of epilepsy, requiring treatment. In fact, statistically, the number of these people afflicted by clinical depression like epilepsy increases in a terrifying manner and many of them don’t respond to conventional antidepressants like Fluoxetine hydrochloride (C$_{17}$H$_{18}$F$_3$NO) and Sertraline(C$_{17}$H$_{17}$Cl$_2$N).

Nevertheless, a promising new type of medical therapy, called transcranial magnetic stimulation, is gaining wider use [37-39]. This technique uses pulses of magnetic energy to induce electric currents in specific regions of the brain. It appears that these pulses can absorb these seizures and then alleviate depression.

In conclusion of our work, we see that our physical study suggests to us a possible way to treat this disease. In fact, our work could constitute an alternative to the above-mentioned therapies to treat epileptic seizures in real time. This could be possible by means of conceiving of an electronic device that can inverse the above-mentioned differences due to the epilepsy seizure such that the patient could come back to his normal state immediately after the triggering of the crisis. We plan to study this aspect in a future work whose results could eventually be patented.

Acknowledgments

Author A.E.F. Djemaï would like to thank Prof. J. Malmivuo for having given us the permission to use the figures 1-3.

References


