**Saudi Journal of Medical and Pharmaceutical Sciences** **ISSN 2413-4929 (Print)**
Scholars Middle East Publishers **ISSN 2413-4910 (Online)**
Dubai, United Arab Emirates
Website: https://saudijournals.com/ DOI: 10.36348/sjmps.2017.v03i02.002

**Original Research Article**

# The Effect of Carbamazepine on EEG Tracings of People with Seizure Disorders in Calabar, Nigeria

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**Abstract:** The Effect of Carbamazepine on EEG tracings of some people with Seizure Disorders was studied on human subjects. This study conducted within 12-24 weeks was a prospective descriptive study involving 30 people with age range of 18-81years coming to University of Calabar Teaching Hospital (UCTH) on account of seizure disorders. The electrical activity of the brain was scanned with the placement of EEG Electro cap (with electrodes connected to the EEG equipment) on each person's head while in sitting position and subjects were asked (at regular intervals) to close/open their eyes, hyperventilate and under photic stimulation. The results obtained from the voltage/amplitude showed no significant difference in the varying states of eye open and photic stimulation (734.23 ± 0.034) in all the regions of the head at $p \leq 0.05$. The background frequency showed a significant difference after carbamazepine treatment regimen (479.88 ± 0.86) in all regions of the head at $p \leq 0.01$ different from before treatment. From the total analysis obtained, carbamazepine decreases the voltage/amplitude, increases the background activity and reduces epileptiform activity observed during recording. From the results on voltage/amplitude of scalp EEG tracings of people, the treatment with the anti-epileptic drug has a dampening effect and suppresses the abnormal electrical impulse of the nerve cells in the human brain, thereby improving the transmission of electrical signals inside the brain. These results showed that carbamazepine reduces epileptiform activity which are indicated by spikes, sharp waves, electrographic seizures, and some other stereotyped phenomena which are strongly associated with seizures.
**Keywords:** Carbamazepine, Electroencephalography, Seizures

## INTRODUCTION

Seizure is the manifestation of an abnormal, hypersynchronous discharge of a population of cortical neurons [1]. This discharge may produce subjective symptoms or objective signs, in which case it is a clinical seizure, or it may be apparent only on an electroencephalogram (EEG), in which case it is an electrographic (or subclinical) seizure.

One of such seizures, epileptic seizure is a brief episode of signs or symptoms due to abnormal excessive or synchronous neuronal activity in the brain [2]. The outward effect can vary from uncontrolled jerking movement (tonic-clonic seizure) to as subtle as a momentary loss of awareness (absence seizure) [3]. Diseases of the brain characterized by an enduring predisposition to generate epileptic seizures are collectively called epilepsy, but seizures can also occur in people who do not have epilepsy.

The cause of most cases of epilepsy is unknown, although some people develop epilepsy as a result of brain injury, stroke, brain tumour and substance use disorders. Genetic mutations are linked to a small proportion of the disease [4]. Epileptic seizures are the result of excessive and abnormal cortical nerve cell activity in the brain. The diagnosis typically involves ruling out other conditions that might cause similar symptoms such as fainting [5].

Making the diagnosis involves determining if any other cause of seizures is present such as alcohol withdrawal or electrolyte problems [6]. This may be done by imaging the brain and performing blood tests. Epilepsy can often be confirmed with an electroencephalogram but a normal test does not rule out the condition [7]. Seizures are controllable with medication in about 70 percent of cases. In those whose seizures do not respond to medication, then surgery, neurostimulation, or dietary changes may be considered [5]. However, not all cases of epilepsy are life long, and some people improve to the point that treatment is no longer needed [4].

About 1% of people worldwide have epilepsy, and nearly 80 percent of cases occur

in developing countries. In the developed world, onset of new cases occurs most frequently in infants and the elderly. The most common type (60 percent) of seizure is convulsive [8]. Of these, one-third begins as generalized seizures from the start, affecting both hemispheres of the brain. Two-thirds begin as partial seizures (which affect one hemisphere of the brain) which may then progress to generalized seizures.

Partial seizures are often preceded by certain experiences, known as auras. They include sensory (visual, hearing, or smell), psychic, autonomic, and motor phenomena. Jerking activity may start in a specific muscle group and spread to surrounding muscle groups in which case it is known as a Jacksonian march [6]. Automatisms may occur, which are non-consciously-generated activities and mostly simple repetitive movements like smacking of the lips or more complex activities such as attempts to pick up something [6].

Carbamazepine (Tegretol) among others is a medication used primarily in the treatment of seizures such as epilepsy and neuropathic pain [9]. It is not effective for absence seizures or myoclonic seizures [10]. It may be used in schizophrenia along with other medications and as a second line agent in bipolar disorder.

EEG is only a brief time sample of electrical activity of the patient's brain showing interictal (between seizures) epiletiform activity [11]. It provides corroborating evidence, but is not proof, unless the patient has a seizure during the EEG (in which case the epiletiform activity is ictal rather than interictal) [11]. Epiletiform activity includes spikes, sharp waves, electrographic seizures and some other stereotyped phenomena which are strongly associated with seizures. Spikes and sharp waves are interictal epiletiform events [12]. Background abnormalities indicate localized or diffuse cerebral dysfunction, and may reflect a transient postictal disturbance or the underlying process responsible for the seizure [13].

Seizure disorders are the most common non-infectious neurologic disease in developing African countries which include Nigeria. Even with this high prevalence rates, there is paucity of knowledge concerning the treatment of seizure disorders, most especially the changes caused by carbamazepine treatment regimen, and the use of EEG test procedure tracings to ascertain these changes. Hence this study seeks to observe or determine the changes caused by carbamazepine treatment regimen, and the use of EEG test procedure tracings to ascertain these changes.

## MATERIALS AND METHODS

### MATERIALS

The materials used for this study were gotten from Internal Medicine Department, University of Calabar Teaching Hospital of which the phoenix EEG machine is dormiciled. The EEG cap / electrodes, EEG electro gel, EEG electrogel syringe and needle, EEG electro cap measuring tape are standard consumable materials that come with the EEG machine. Other materials include; hand gloves, methylated spirit, cotton wool, cotton buds, baby wipes and handwash liquid.

### Study area

This was a prospective descriptive study involving people aged 18years and above coming to University of Calabar Teaching Hospital (UCTH), Calabar, Cross River State, Nigeria on account of seizure disorders.

### Inclusion criteria

Subjects aged 18 years and above with diagnosis of seizure disorder. Subjects on carbamazepine monotherapy and apparently healthy subjects fit enough to complete the study.

### Exclusion criteria

People who had undergone MRI or CT before acquisition of EEG test. People with poor EEG tracings and incomplete prior assessment. People below the age of 18 at time of presentation were excluded and those who had done skull plastic surgery due to trauma or accidents.

### Procedure

The first EEG test procedure on subject was before commencement of carbamazepine treatment, and then after 3 to 4 weeks after commencement of treatment, a second EEG test procedure was conducted. The pill counting method was employed to ascertain if subjects were compliant to the carbamazepine treatment regimen before commencement of a second EEG test procedure.

Subjects were required to wash their head/hair the night before the EEG test and should not apply products (such as sprays or gels) on the hair on the morning before the test. Subjects were advised to avoid consuming any food or drinks containing caffeine for at least 8 hours prior to the EEG test procedure.

An EEG test procedure which took about 30 to 60 minutes to complete typically involved the following steps:

- Subjects were asked to lie down on their back on a reclining chair or on a bed.
- Subjects' head were measured and marked at spots where each electrode was placed. These spots were scrubbed with methylated spirit using a cotton wool to help clean off sweat or hair cream/oil. A measuring tape was only used to ascertain the size of the head, and then the appropriate EEG cap with the right head size was worn on subject.

- An electro-gel was applied on 16 to 25 electrodes through a hole located on each electrode, attached to various spots on the EEG cap.
- Once the EEG test procedure begins, the electrodes send electrical impulse data from the brain to the Phoenix EEG recording machine. This machine converts the electrical impulses into visual patterns that can be seen on the EEG monitor screen. These patterns are saved on the Phoenix EEG recording machine.
- During recording, subjects were asked to lie still, avoid body movements as much as possible, no talking, laughing or body scratching.
- Recording was carried out and the subjects were told when to keep their eyes closed, eyes open, hyperventilate (breathing in and out deeply) and also photic stimulation using a photo-sensitive bulb which was placed in front of subject eyes to test for photo-sensitive epilepsy.
- Visual analysis was used to ascertain baseline measurements of amplitude, frequency and epiletiform activity.
- After completing the EEG test procedure, the EEG cap/electrodes were removed from the scalp of subjects' head. Electro-gel domiciled on each hole located on the electrode was removed with cotton buds and washed with washing liquid. Baby wipes were used to wipe the head clean of remnants of electro-gel.

**Data analysis**

Data collected were recorded in the subjects EEG tracings printout which were entered into a manuscript excel spread sheet. Entered data were double checked to ensure precision and data analysis was carried out using statistical package for the social science (SPSS) window soft version 20 (SPSS inc, Chicago, IL, USA). Tables and figures were used to present the results. P –value of $\leq 0.05$ was considered as statistically significant.

**RESULTS AND DISCUSSION**

**Table-1: Data on people with seizure disorders placed on carbamazepine Treatment regimen.**

| Variable | Sub-variable | Frequency | Percentage |
|---|---|---|---|
| State during assessment | Awake | 6 | 88.9 |
| | Drowsy | 2 | 11.1 |
| Handedness | Right | 25 | 95 |
| | Left | 5 | 5 |
| With febrile convulsion | Yes | 4 | 28.6 |
| | No | 10 | 71.4 |
| Non-febrile convulsion | Yes | 11 | 84.6 |
| | No | 2 | 15.4 |
| Family history of seizure disorder | Yes | 6 | 31.6 |
| | No | 13 | 68.4 |

**Table-2: Voltage / amplitude (µV/cm) measured on the right side of the Head before carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 0.29 | 0.43 | 0.29 | 0.30 |
| | ±0.01 | ±0.01*** | ±0.01$^{c}$ | ±0.00$^{c, z}$ |
| Temporal | 0.28 | 0.47 | 0.30 | 0.30 |
| | ±0.01 | ±0.01*** | ±0.00**$^{, c}$ | ±0.00*$^{, c}$ |
| Central | 0.29 | 0.46 | 0.29 | 0.29 |
| | ±0.00 | ±0.01*** | ±0.01$^{c}$ | ±0.00$^{c}$ |
| Parietal | 0.29 | 0.45 | 0.30 | 0.28 |
| | ±0.01 | ±0.01*** | ±0.00$^{c}$ | ±0.01$^{c, x}$ |
| Occipital | 0.30 | 0.47 | 0.29 | 0.29 |
| | ±0.00 | ±0.01*** | ±0.01$^{c}$ | ±0.01$^{c}$ |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; $p<0.05$, State: F = 701.452; df = 3; $p<0.001$) , *** = $p<0.001$, ** = $p<0.01$, * = $p<0.05$ vs eye open, c = $p<0.001$ vs eye closed, x = $p<0.05$ vs hyperventilation

**Table-3: Voltage / amplitude (µV/cm) measured on the left side of the head before carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 0.29 | 0.43 | 0.29 | 0.30 |
| | ±0.01 | ±0.01*** | ±0.01$^{c}$ | ±0.00$^{c, x}$ |
| Temporal | 0.28 | 0.47 | 0.30 | 0.30 |
| | ±0.01 | ±0.01*** | ±0.00**$^{, c}$ | ±0.00*$^{, c}$ |
| Central | 0.29 | 0.46 | 0.29 | 0.29 |
| | ±0.00 | ±0.01*** | ±0.01$^{c}$ | ±0.00$^{c}$ |
| Parietal | 0.29 | 0.45 | 0.30 | 0.28 |
| | ±0.01 | ±0.01*** | ±0.00$^{c}$ | ±0.01$^{c, x}$ |
| Occipital | 0.30 | 0.48 | 0.29 | 0.31 |
| | ±0.00 | ±0.01*** | ±0.00$^{c}$ | ±0.01$^{c}$ |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-4: Voltage / amplitude (µV/cm) measured on the right side of the head after carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 83.00 | 106.67 | 82.80 | 82.73 |
| | ±0.00 | ±2.05*** | ±0.07***$^{, c}$ | ±0.12*$^{, c}$ |
| Temporal | 83.03 | 105.90 | 82.67 | 82.50 |
| | 0.25 | ±1.96*** | ±0.12$^{c}$ | ±0.17$^{c}$ |
| Central | 82.83 | 108.33 | 82.80 | 82.73 |
| | ±0.07 | ±2.19*** | ±0.07$^{c}$ | ±0.12$^{c}$ |
| Parietal | 83.03 | 105.90 | 82.67 | 82.50 |
| | 0.25 | ±1.96*** | ±0.12$^{c}$ | ±0.17$^{c}$ |
| Occipital | 82.73 | 114.33 | 82.33 | 82.83 |
| | ±0.08 | ±2.27*** | ±0.11**$^{, c}$ | ±0.07$^{c, z}$ |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-5: Voltage / amplitude (µV/cm) measured on the left side of the head after carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 83.00 | 106.67 | 82.80 | 82.73 |
| | ±0.00 | ±2.05*** | ±0.07**$^{, c}$ | ±0.12*$^{, c}$ |
| Temporal | 83.03 | 105.90 | 82.67 | 82.50 |
| | ±0.25 | ±1.96*** | ±0.12$^{c}$ | ±0.17$^{c}$ |
| Central | 82.83 | 108.33 | 82.80 | 82.73 |
| | ±0.07 | ±2.19*** | ±0.07$^{c}$ | ±0.12$^{c}$ |
| Parietal | 83.03 | 105.90 | 82.67 | 82.50 |
| | ±0.25 | ±1.96*** | ±0.12$^{c}$ | ±0.17$^{c}$ |
| Occipital | 82.73 | 114.33 | 82.33 | 82.83 |
| | ±0.08 | ±2.27*** | ±0.11**$^{, c}$ | ±0.07$^{c, z}$ |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-6: Background frequency (Hz) measured on the right side of the head before carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 4.57 | 4.90 | 2.97 | 2.53 |
| | ±0.37 | ±0.06*** | ±0.03***, c | ±0.10***, c, z |
| Temporal | 2.93 | 4.83 | 2.93 | 2.93 |
| | ±0.05 | ±0.08*** | ±0.05c | ±0.05c |
| Central | 2.73 | 4.90 | 2.97 | 3.90 |
| | ±0.08 | ±0.07*** | ±0.03**, c | ±0.06***, c, z |
| Parietal | 3.00 | 4.93 | 3.20 | 3.93 |
| | ±0.00 | ±0.05*** | ±0.07**, c | ±0.05***, c, z |
| Occipital | 2.97 | 5.00 | 3.20 | 3.93 |
| | ±0.03 | ±0.00*** | ±0.07**, c | ±0.05***, c, z |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-7: Background frequency (Hz) measured on the left side of the head before carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 4.57 | 4.90 | 2.97 | 2.53 |
| | 0.37 | ±0.06*** | ±0.03***, c | 0.10***, c, z |
| Temporal | 2.93 | 4.83 | 2.93 | 2.93 |
| | ±0.05 | ±0.08*** | ±0.05c | ±0.05c |
| Central | 2.73 | 4.90 | 2.97 | 3.90 |
| | ±0.08 | ±0.07*** | ±0.03**, c | ±0.06***, c,z |
| Parietal | 3.00 | 4.93 | 3.20 | 3.93 |
| | ±0.00 | ±0.05*** | ±0.07**, c | ±0.05***, c,z |
| Occipital | 2.97 | 5.00 | 3.20 | 3.97 |
| | ±0.03 | ±0.00*** | ±0.07**, c | ±0.08***, c, z |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-8: Background frequency (Hz) measured on the right side of the head after carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 14.93 | 8.27 | 14.97 | 15.10 |
| | ±0.39 | ±0.17*** | ±0.40c | ±0.50c |
| Temporal | 15.10 | 8.47 | 14.77 | 14.30 |
| | ±0.37 | ±0.18*** | ±0.24c | ±0.12*, c |
| Central | 15.07 | 8.10 | 15.03 | 15.27 |
| | ±0.50 | ±0.13*** | ±0.53c | ±0.48c |
| Parietal | 14.33 | 8.80 | 14.17 | 14.63 |
| | ±0.09 | ±0.14*** | ±0.10 c | ±0.11*, c, y |
| Occipital | 15.30 | 8.53 | 14.63 | 14.57 |
| | ±0.51 | ±0.20*** | ±0.37c | ±0.11c |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-9: Background frequency (Hz) measured on the left side of the head after carbamazepine treatment regimen.**

| Location | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|
| Frontal | 14.93 | 8.27 | 14.97 | 15.10 |
| | ±0.39 | ±0.17*** | ±0.40$^{c}$ | ±0.50$^{c}$ |
| Temporal | 15.10 | 8.47 | 14.77 | 14.30 |
| | ±0.37 | ±0.18*** | ±0.24$^{c}$ | ±0.12*, $^{c}$ |
| Central | 15.07 | 8.10 | 15.03 | 15.27 |
| | ±0.50 | ±0.13*** | ±0.53$^{c}$ | ±0.48$^{c}$ |
| Parietal | 14.33 | 8.80 | 14.17 | 14.63 |
| | ±0.09 | ±0.14*** | ±0.10$^{c}$ | ±0.11*, $^{c, y}$ |
| Occipital | 15.30 | 8.53 | 14.63 | 14.57 |
| | ±0.51 | ±0.20*** | ±0.37$^{c}$ | ±0.11$^{c}$ |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-10: Comparison of voltage / amplitude (µV/cm) measured on the right side of the head before and after carbamazepine treatment regimen.**

| Location | Treatment | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|---|
| Frontal | Before | 0.29 | 0.43 | 0.29 | 0.30 |
| | | ±0.01 | ±0.01 | ±0.01 | ±0.00 |
| | After | 83.00 | 106.67 | 82.80 | 82.73 |
| | | ±0.00*** | ±2.05*** | ±0.07*** | ±0.12*** |
| Temporal | Before | 0.28 | 0.47 | 0.30 | 0.30 |
| | | ±0.01 | ±0.01 | ±0.00 | ±0.00 |
| | After | 83.03 | 105.90 | 82.67 | 82.50 |
| | | 0.25*** | ±1.96*** | ±0.12*** | ±0.17*** |
| Central | Before | 0.29 | 0.46 | 0.29 | 0.29 |
| | | ±0.00 | ±0.01 | ±0.01 | ±0.00 |
| | After | 82.83 | 108.33 | 82.80 | 82.73 |
| | | ±0.07*** | ±2.19*** | ±0.07*** | ±0.12*** |
| Parietal | Before | 0.29 | 0.45 | 0.30 | 0.28 |
| | | ±0.01 | ±0.01 | ±0.00 | ±0.01 |
| | After | 83.03 | 105.90 | 82.67 | 82.50 |
| | | 0.25*** | ±1.96*** | ±0.12*** | ±0.17*** |
| Occipital | Before | 0.30 | 0.47 | 0.29 | 0.29 |
| | | ±0.00 | ±0.01 | ±0.01 | ±0.01 |
| | | 82.73 | 114.33 | 82.33 | 82.83 |
| | | ±0.08*** | ±2.27*** | ±0.11*** | ±0.07*** |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-11: Comparison of voltage / amplitude (µV/cm) measured on the left side of the head before and after carbamazepine treatment regimen.**

| Location | Treatment | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|---|
| Frontal | Before | 0.29 | 0.43 | 0.29 | 0.30 |
| | | ±0.01 | ±0.01 | ±0.01 | ±0.00 |
| | After | 83.00 | 106.67 | 82.80 | 82.73 |
| | | ±0.00*** | ±2.05*** | ±0.07*** | ±0.12*** |
| Temporal | Before | 0.28 | 0.47 | 0.30 | 0.30 |
| | | ±0.01 | ±0.01 | ±0.00 | ±0.00 |
| | After | 83.03 | 105.90 | 82.67 | 82.50 |
| | | ±0.25*** | ±1.96*** | ±0.12*** | ±0.17*** |
| Central | Before | 0.29 | 0.46 | 0.29 | 0.29 |
| | | ±0.00 | ±0.01 | ±0.01 | ±0.00 |
| | After | 82.83 | 108.33 | 82.80 | 82.73 |
| | | ±0.07*** | ±2.19*** | ±0.07*** | ±0.12*** |
| Parietal | Before | 0.29 | 0.45 | 0.30 | 0.28 |
| | | ±0.01 | ±0.01 | ±0.00 | ±0.01 |
| | After | 83.03*** | 105.90*** | 82.67*** | 82.50*** |
| | | ±0.25 | ±1.96 | ±0.12 | ±0.17 |
| Occipital | Before | 0.30 | 0.48 | 0.29 | 0.31 |
| | | ±0.00 | ±0.01 | ±0.00 | ±0.01 |
| | After | 82.73 | 114.33 | 82.33 | 82.83 |
| | | ±0.08*** | ±2.27*** | ±0.11*** | ±0.07*** |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-12: Comparison of background frequency (Hz) measured on the right side of the head before and after carbamazepine treatment regimen.**

| Location | Treatment | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|---|
| Frontal | Before | 4.57 | 4.90 | 2.97 | 2.53 |
| | | ±0.37 | ±0.06 | ±0.03 | ±0.10 |
| | After | 14.93 | 8.27 | 14.97 | 15.10 |
| | | ±0.39*** | ±0.17*** | ±0.40*** | ±0.50*** |
| Temporal | Before | 2.93 | 4.83 | 2.93 | 2.93 |
| | | ±0.05 | ±0.08 | ±0.05 | ±0.05 |
| | After | 15.10 | 8.47 | 14.77 | 14.30 |
| | | ±0.37*** | ±0.18*** | ±0.24*** | ±0.12*** |
| | | | | | |
| Central | Before | 2.73 | 4.90 | 2.97 | 3.90 |
| | | ±0.08 | ±0.07 | ±0.03 | ±0.06 |
| | After | 15.07 | 8.10 | 15.03 | 15.27 |
| | | ±0.50*** | ±0.13*** | ±0.53*** | ±0.48*** |
| Parietal | Before | 3.00 | 4.93 | 3.20 | 3.93 |
| | | ±0.00 | ±0.05 | ±0.07 | ±0.05 |
| | After | 14.33 | 8.80 | 14.17 | 14.63 |
| | | ±0.09*** | ±0.14*** | ±0.10*** | ±0.11*** |
| Occipital | Before | 2.97 | 5.00 | 3.20 | 3.93 |
| | | ±0.03 | ±0.00 | ±0.07 | ±0.05 |
| | After | 15.30 | 8.53 | 14.63 | 14.57 |
| | | ±0.51*** | ±0.20*** | ±0.37*** | ±0.11*** |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Table-13: Comparison of background frequency (Hz) measured on the left side of the head before and after carbamazepine treatment regimen.**

| Location | Treatment | Eye open | Eye closed | Hyperventilation | Photic stimulation |
|---|---|---|---|---|---|
| Frontal | Before | 4.57 | 4.90 | 2.97 | 2.53 |
| | | ±0.37 | ±0.06 | ±0.03 | ±0.10 |
| | After | 14.93 | 8.27 | 14.97 | 15.10 |
| | | ±0.39*** | ±0.17*** | ±0.40*** | ±0.50*** |
| Temporal | Before | 2.93 | 4.83 | 2.93 | 2.93 |
| | | ±0.05 | ±0.08 | ±0.05 | ±0.05 |
| | After | 15.10 | 8.47 | 14.77 | 14.30 |
| | | ±0.37*** | ±0.18*** | ±0.24*** | ±0.12*** |
| Central | Before | 2.73 | 4.90 | 2.97 | 3.90 |
| | | ±0.08 | ±0.07 | ±0.03 | ±0.06 |
| | After | 15.07 | 8.10 | 15.03 | 15.27 |
| | | ±0.50*** | ±0.13*** | ±0.53*** | ±0.48*** |
| Parietal | Before | 3.00 | 4.93 | 3.20 | 3.93 |
| | | ±0.00 | ±0.05 | ±0.07 | ±0.05 |
| | After | 14.33 | 8.80 | 14.17 | 14.63 |
| | | ±0.09*** | ±0.14*** | ±0.10*** | ±0.11*** |
| Occipital | Before | 2.97 | 5.00 | 3.20 | 3.97 |
| | | ±0.03 | ±0.00 | ±0.07 | ±0.08 |
| | After | 15.30 | 8.53 | 14.63 | 14.57 |
| | | ±0.51*** | ±0.20*** | ±0.37*** | ±0.11*** |

Values are presented as mean ±SEM, n = 30, (Location: F = 0.0136; df = 4; p<0.05, State: F = 701.452; df = 3; p<0.001) , *** = p<0.001, ** = p<0.01, * = p<0.05 vs eye open, c = p<0.001 vs eye closed, x = p<0.05 vs hyperventilation

**Comparison of voltage / amplitude (µV/cm) measured on the right and left side of the head at varying states before carbamazepine treatment regimen.**

As shown in table 2 and 3, the mean frequency of voltage / amplitude (µV/cm) measured on the right and left sides of the head at varying states before carbamazepine treatment regimen were 759.10 ± 0.198 for the right side of the head and 701.45 ± 0.013 for the left side of the head respectively. There is a significant difference noted in the central region of the head different from the frontal, temporal, and parietal regions and a significant difference noted in the occipital region of the head different from the frontal, temporal, parietal and central regions, both occurring in a state of eyes open. There is a significant difference noted in the temporal region of the head different from the frontal, central and parietal regions and a significant difference noted in the occipital region of the head different from the frontal, temporal, central and parietal regions, both occurring in a state of eyes closed. There is a significant difference noted in the temporal region of the head different from the frontal, central, parietal and occipital regions, occurring during hyperventilation. There is also a significant difference noted in the parietal region of the head different from the frontal, temporal, central and occipital regions, occurring during photic stimulation.

**Comparison of voltage / amplitude (µV/cm) measured on the right and left side of the head at varying states after carbamazepine treatment regimen.**

As seen in Table 4 and 5, the mean frequency of the comparison of voltage / amplitude (µV/ cm) measured on the right and left side of the head at varying states after carbamazepine treatment regimen were 734.23 ± 0.034 for the right side of the head and 734.23 ± 0.034 for the left side of the head respectively. There is a significant difference noted in the occipital region of the head different from the frontal, temporal, central and parietal regions occurring in a state of eye closed. There is also a significant difference noted in the occipital region of the head different from the frontal, temporal, central and parietal regions of the head occurring during hyperventilation. There is no significant difference noted in the varying states of eye open and photic stimulation in all the regions of the head.

**Mean background frequency (Hz) measured on the right and left side of the head at varying states before carbamazepine treatment regimen.**

As seen in table 6 and 7, the mean frequency of the comparison of background frequency (Hz) measured on the right and left side of the head at varying ststes before carbamazepine treatment regimen were 358.75 ± 9.54 for the right side of the head and

352.02 ± 9.56 respectively. There is significant difference noted at the temporal, central, parietal and occipital regions of the head different from the frontal region which occurs in the state of eye open. There is significant difference noted at the central region of the head different from the frontal, temporal, parietal and occipital regions which occurs in the state of eye closed. There is significant difference noted at the parietal and occipital regions of the head different from the frontal, temporal and central regions which occurs during hyperventilation. There is also significant difference noted on the central, parietal and occipital regions of the head different from the frontal and temporal regions which occurs during photic stimulation.

**Mean background frequency (Hz) measured on the right and left side of the head at varying states after carbamazepine treatment regimen.**

As seen in table 8 and 9, the mean frequency of the comparison of background frequency (Hz) measured on the right and left side of the head at varying states after carbamazepine treatment regimen were 479.88 ± 0.86 for the right side of the head and 479.88 ± 0.86 for the left side of the head respectively. There is a significant difference noted at the parietal region of the head different from the frontal, temporal, central and occipital regions which occurs in the state of eye closed. There is also a significant difference noted at the temporal region of the head different from the frontal, central, parietal and occipital regions which occurs during photic stimulation. There is no significant difference noted in the varying states of eye open and hyperventilation in all the the regions of the head.

**Background frequency (Hz) measured on the right and left sides of the head in the state of eye open before and after carbamazepine treatment regimen.**

Figure 1 shows the background frequency (Hz) measured on the right and left sides of the head in the state of eye open before and after carbamazepine treatment regimen. The mean frequency of the background frequency (Hz) measured on the right and left sides of the head in the state of eye open before and after carbamazepine treatment regimen were 3.24 ± 14.94 for the right side of the head and 2.96 ± 14.94 for the left side of the head respectively. There is significant difference after carbamazepine treatment regimen in the frontal, temporal, central, parietal and occipital regions of the head different from before treatment.

**Background frequency (Hz) measured on the right and left sides of the head in the state of eye closed before and after carbamazepine treatment regimen.**

Figure 2 shows the background frequency (Hz) measured on the right and left sides of the head in the state of eye closed before and after carbamazepine treatment regimen. The mean frequency of the background frequency (Hz) measured on the right and left sides of the head in the state of eye closed before and after carbamazepine treatment regimen were 4.91 ± 8.43 for the right side of the head and 4.91 ± 8.43 for the left side. There is significant difference before and after carbamazepine treatment regimen in all regions of the head.

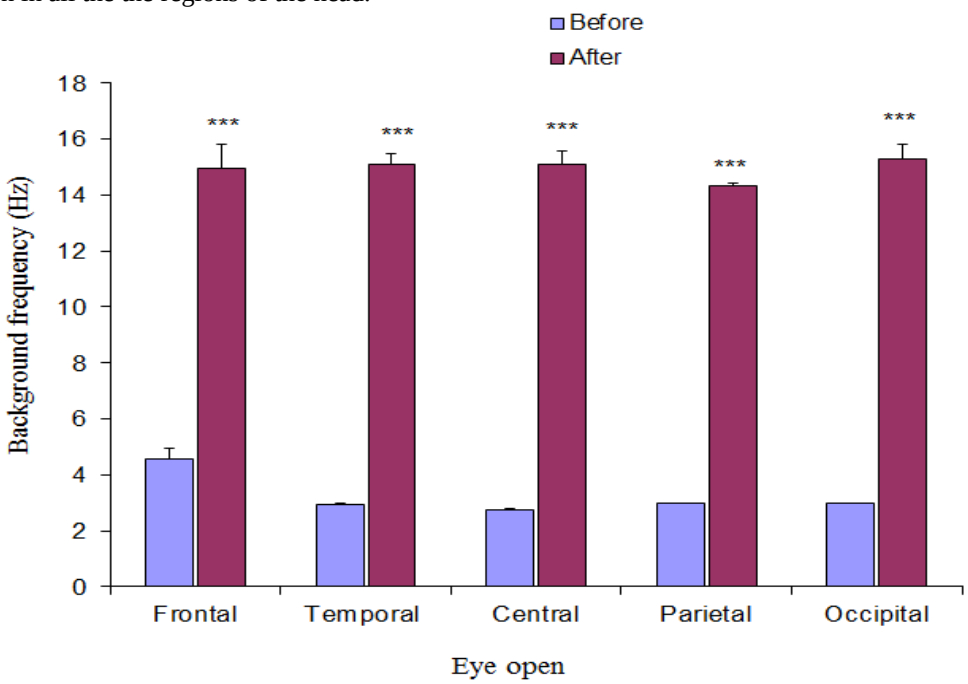


**Fig-1: Background frequency (Hz) measured on the right and left sides of the head in the state of eye open before and after carbamazepine treatment regimen.llllll**
**Values are expressed as mean ±SEM, n = 30**
***** = significant**

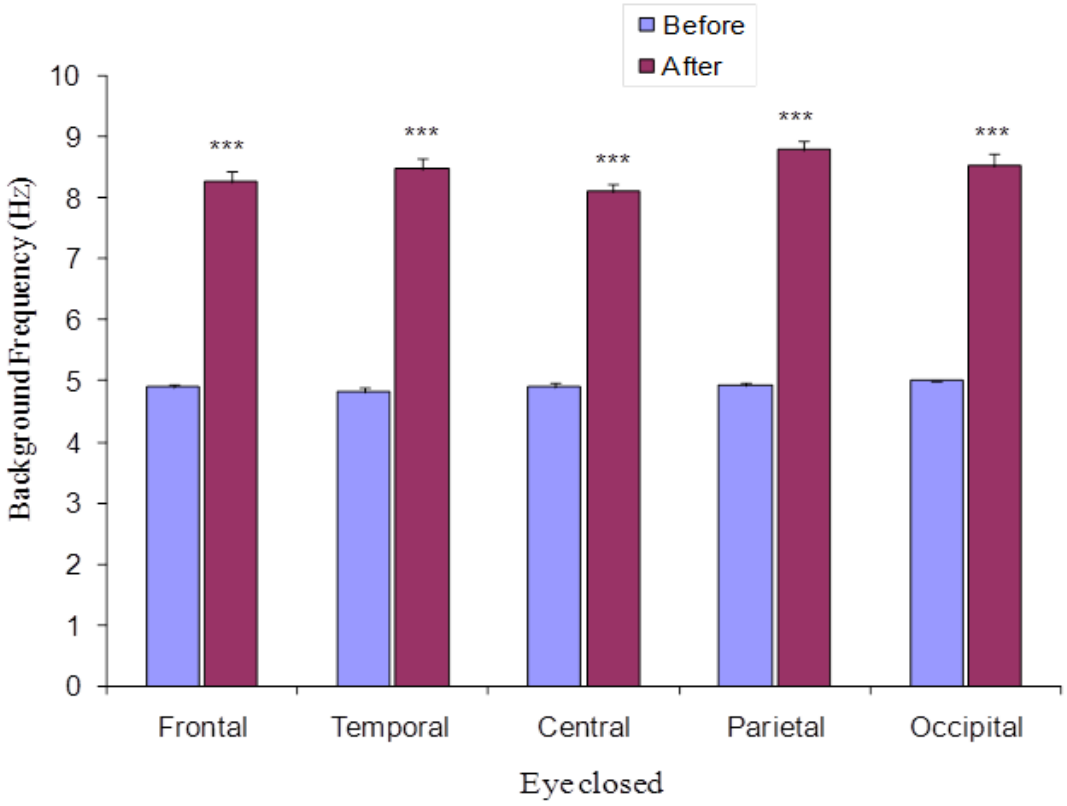


**Fig-2: Background frequency (Hz) measured on the right and left sides left sides of the head in the state of eye closed before and after carbamazepine treatment regimen.**
**Values are expressed as mean ±SEM, n = 30**
***** = significantly**

**Background frequency (Hz) measured on the right and left sides of the head during hyperventilation before and after carbamazepine treatment regimen.**

Figure 3 shows the background frequency (Hz) measured on the right and left sides of the head during hyperventilation before and after carbamazepine treatment regimen. The mean frequency for the background frequency (Hz) measured on the right and left sides of the head during hyperventilation before and after carbamazepine treatment regimen were 3.05 ± 14.71 for the right side of the head and 3.05 ± 14.71 for the left side respectively. There is significant difference after carbamazepine treatment regimen in the frontal, temporal, central, parietal and occipital regions of the head different from before treatment. Values are expressed as mean ± SEM, n=30. *** = $p<0.001$, * = $p<0.05$vs eye open, c = $p<0.001$ vs eye closed, y = $p<0.01$ vs hyperventilation.

**Background frequency (Hz) measured on the right and left sides of the head during photic stimulation before and after carbamazepine treatment regimen.**

Figure 4 shows the background frequency (Hz) measured on the right and left sides of the head during photic stimulation before and after carbamazepine treatment regimen. The mean frequency of the background frequency (Hz) measured on the right and left sides of the head during photic stimulation before and after carbamazepine treatment regimen were 3.44 ± 14.77 for the right side of the head and 3.45 ± 14.77. There is significant difference after carbamazepine treatment regimen in all regions of the head different from before treatment. Values are mean ± SEM, n=30.

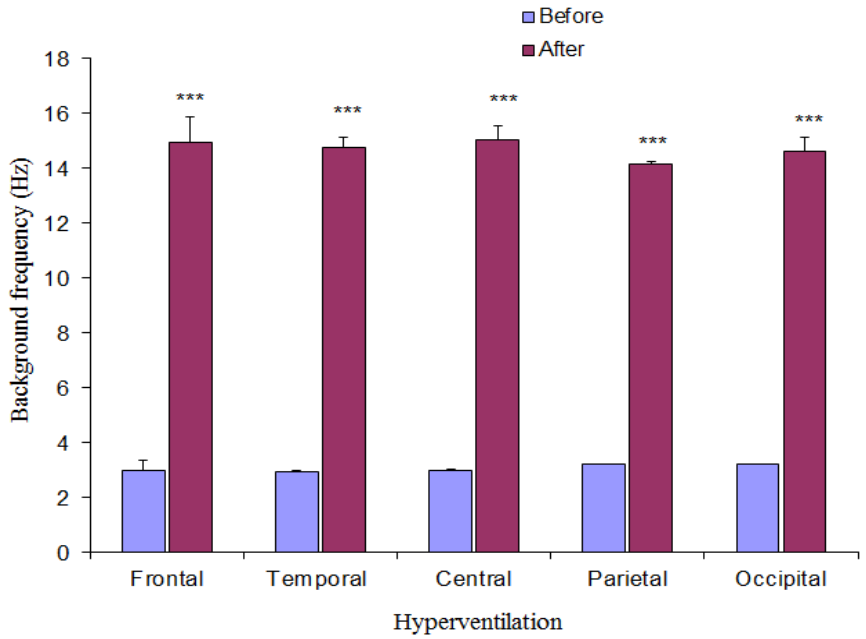


**Fig-3: Background frequency (Hz) measured on the right and left sides left sides of the head during hyperventilation beforellll and after carbamazepine treatment regimen.**
**Values are expressed as mean ±SEM, n = 30**
***** = significantly**

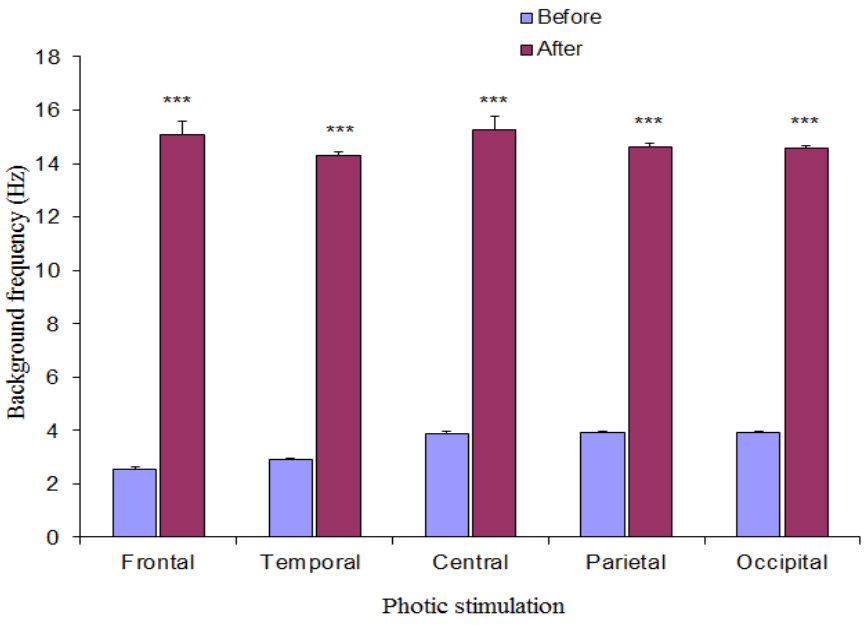


**Fig-4: Background frequency (Hz) measured on the right and left sides oleft sides of the head during photic stimulation beforel and after carbamazepine treatment regimen.**
**Values are expressed as mean ±SEM, n = 30**
***** = significantly di**

This study observes the effect of carbamazepine on EEG tracings of people with seizure disorders in Calabar, Nigeria. The parameters considered in the study include the voltage / amplitude of scalp EEG tracings which encompass localized rhythmic activity. Other parameters investigated in the study were background frequency of scalp EEG tracings and epileptiform activity.

The voltage / amplitude of scalp EEG tracings were used to study bilateral, often asymmetric, rhythmic activity (usually high amplitude and diffuse activity). The background frequency were used to study

background activity of beta, alpha, theta and delta wave patterns which indicates localized or cerebral dysfunction, transient postictal disturbance or the underlying process responsible for the seizure. The epileptiform activity was used to study spikes, sharp waves, electrographic seizures, and some other stereotyped phenomena which are strongly associated with seizures. It is pertinent to note that these parameters are however not exclusive for studying one activity as indicated. This is because some activities in the voltage / amplitude are used as measures of the underlying process responsible for the seizure. Also, parameters such as epileptiform activity could be used to assess localized or cerebral dysfunction.

The voltage / amplitude of scalp EEG tracings have been used as a test that assesses bilateral, often asymmetric, rhythmic activity (usually high amplitude and diffuse activity). It provides simultaneous measures of rhythmic activity as well as the process responsible for the seizure [2]. Physical observations of people with seizure disorders such as recurrent episodes of convulsions, accompanied by foaming from the mouth and recurrent loss of awareness, preceded by upward rolling of eyes / stiffening of the limbs are used as measures of the underlying process responsible for the seizure. These physical observations indicate high frequency in the voltage / amplitude [12].

The rhythmic activity (rhythm or voltage asymmetry) measures both partial and generalized seizures. During partial seizures it indicates quite localized or lateralized abnormal rhythmic activity, while in generalized seizures rhythmic activities is usually high amplitude and diffuse [11].

## CONCLUSION

From the result obtained on the voltage / amplitude of scalp EEG tracings of people, the treatment with the anti epileptic drug has a dampening effect and suppresses the abnormal electrical impulse of the nerve cells in the human brain, thereby improving the transmission of electrical signals inside the brain.

When the drug treatment is administered it tends to increase the background activity of theta up to the alpha and beta range and, with progressive signs of frontal and occipital fast activities. The results in this study also showed that carbamazepine reduces epileptiform activity which are indicated by spikes, sharp waves, electrographic seizures, and some other stereotyped phenomena which are strongly associated with seizures.

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