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The clinical utility of electroencephalographic studies in children and adolescents at Tara Hospital

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The clinical utility of electroencephalographic studies in children and adolescents at Tara Hospital

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, in partial fulfilment of the requirements for the degree of Master of Medicine in Psychiatry.

Johannesburg, 2019

DECLARATION

I, Dr Othelia Omphemetse Mahuma, declare that this research report is my own work. It is being submitted for the degree of MMed Psychiatry at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other university.

……

The …… day of ……., 2019

DEDICATION

To: Mlinga for your love and unwavering support and my daughters Rato and Luchi for your understanding and encouragement and patience throughout this process.

ABSTRACT

Background Electroencephalography (EEG) can be useful in the diagnosis of epilepsy and other neuropsychiatric disorders. There is a need to evaluate whether it is appropriately utilised, particularly among children and adolescents.

Aim To assess the clinical utility of EEG studies in children and adolescents at Tara Hospital.

Objectives To describe the reasons for referral for EEG studies, to determine the prevalence of abnormal EEGs and to examine their impact on the diagnosis and management of patients.

Methods A retrospective record review of all patients under age 18 referred for EEGs over a five-year period was conducted. Descriptive statistics were used to analyse the clinical profile and EEG findings, and inferential statistics to test associations between clinical variables and EEG findings.

Results 249 participants were enrolled; 63.4% were males, 56.9% were between 5-12 years and 43% were between 13-18 years. 13.7% had an abnormal EEG, of which 70.6% were males. The proportion of abnormal EEGs was significantly higher in the 5-12 years age group compared to the 13-18 years age group (76.5% vs. 53.7%, p=0.013). The most common abnormal EEG finding was focal epileptiform activity (27%), followed by generalised epileptiform activity (24%) and focal slowing (18%). The rate of abnormal EEGs was found to be clinically significant in those with a history of seizures (p= 0.027), traumatic brain injury (p= 0.18) and comorbid medical conditions (p= 0.07). The overall demonstrable impact of the EEG in our study was 22%.

Conclusion Our results confirm that EEG studies add value in the diagnosis and psychiatric management of children and adolescents.

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Mrs Maureen Chalom, senior EEG Technician at Tara Hospital.

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TABLE 1: ABBREVIATIONS AND DEFINITIONS	
ADHD	Attention Deficit Hyperactivity Disorder
AIDS	Acquired Immunodeficiency syndrome
BECTS	Benign Epilepsy with Centro-Temporal Spikes
CPSO	College of Physicians and Surgeons of Ontario
CT	Computed Tomography
DSM	Diagnostic and Statistical Manual
EEG	Electroencephalogram
HIV	Human Immunodeficiency Virus
HPE	HIV-associated Progressive Encephalopathy
MCA	Middle Cerebral Artery
MRI	Magnetic Resonance Imaging
SAS	Statistical Analysis System
TBI	Traumatic Brain Injury
Participant and patient were used interchangeably to refer to the subjects enrolled in our study	

CHAPTER 1

INTRODUCTION

1.1 Background

The discovery of electroencephalography (EEG) was made when electric potentials were recorded from activated muscles and nerves of animals, and later in the 19th century from the cerebral cortex of animals (Stone & Hughes, 2013). In the 1920's a neuropsychiatrist, Hans Berger, recorded the first EEG electrical potentials from skulls of patients with skull defects, and from intact subjects with more sensitive equipment a few years later (Stone & Hughes, 2013).

1.2. Uses

Hans Berger's breakthrough work established the EEG as an important non-invasive tool that can assist in the investigation and diagnosis of psychiatric and neuropsychiatric disorders. His original research looked for encephalopathic changes in patients with psychiatric disorders, including schizophrenia (O'Sullivan S. S. et al., 2006). The EEG has great value in assisting with the identification of epilepsy and of organic mental disorders. It can aid to confirm or rule out various conditions including epilepsy, encephalitis, encephalopathy, sleep disorders etc. Many psychiatric disorders have a neurobiological base, as such the term 'organic mental disorders' is unsatisfactory. The EEG, typically marked by generalised or focal slowing in patients with acute or chronic encephalopathies is useful to the clinician in differentiation of these disorders from psychiatric disorders (O'Sullivan S. S. et al., 2006).

1.3. EEG studies in psychiatry

Hughes and John (1999) propose that the EEG is most useful to the psychiatrist in the following:

- Paroxysmal activity identification,
- Identification of gross alterations in the background frequencies of the EEG,

- Identifying intermixed slow activity that may be related to delirium or dementia,
- Sleep disorders evaluation.

They also suggest inclusion of the EEG in the diagnostic workup of the following:

- An acute confusional state,
- The first presentation of schizophrenia,
- A major mood disorder or mania, and
- Refractory behavioural problems such as violence, panic or obsessions (Hughes & John, 1999).

The association between EEG abnormalities and organic brain disease is well established. Clinicians are also often required to make a differentiation between functional psychiatric disorders and organic brain syndromes, sometimes without the aid of advanced neuroimaging studies (Rascher, Connor, & Jeena, 2004).

1.4. International studies

Lam et al. (1988) undertook a study to survey the pattern of clinical use of the EEG in the general adult psychiatric population. The study was based on a review of charts of 150 psychiatric inpatients referred for EEG between April 1984 and July 1985. They excluded neuropsychiatry patients because the study focused on the use of EEG by general psychiatrists. Charts were also reviewed to record any organic factors that were identified before an EEG was ordered. Clinical indications for EEGs were possible organic disorder (30%), possible temporal lobe epilepsy (19.3%), pre-ECT workup (12.7%), dementia workup (11.3%), epilepsy (14%), possible delirium (5.3%), previous abnormal EEG (3.3%) and other indication (4%).

The only clinical indications significantly associated with an abnormal EEG were a history of epilepsy and a suspicion of a recent seizure. Seventeen of the EEGs performed were abnormal. None of the abnormal EEGs helped to identify an organic aetiology that was not already

diagnosed before the EEG examination. For 14 patients who had both organic factors and an abnormal EEG, the EEG result had been noted on the chart, and further clinical investigation had been pursued. They found that the abnormal EEG had been useful in the management of these patients by confirming a seizure focus, ruling out complex partial status epilepticus, or leading to clarification of the role of organic factors in the psychiatric presentation (Lam et al., 1988).

A more recent study by Raybould et al. (2012) looked at EEG and clinical data of 406 adult patients presenting with acute psychosis, including both recurrent and first-time psychoses, from 2001 to 2006. All the EEG referrals were made by psychiatrists in an academic hospital to rule out ictal or inter-ictal psychosis in patients presenting with an acute psychotic episode. None of these patients had witnessed seizures before or after the onset of psychosis. A routine EEG was obtained in all patients with new-onset psychosis, and in cases of recurrent psychoses, the EEG was obtained during one of the presentations in order to rule out seizures. After exclusions, 240 participants had an EEG. Of the 240 participants, 141 patients had brain MRI scans and 39 patients had brain CT scans. There was no ictal epileptiform activity recorded in any of the patients. However, 7 patients had intermittent epileptiform discharges, 10 patients had posterior basic rhythm slowing, 2 patients had right-sided slowing, 9 patients had left-sided slowing and 19 patients had bilateral slowing. There were no significant associations between EEG and MRI findings and between EEG and CT findings.

These findings suggest that despite the recognised association between acute psychosis and seizures, a routine EEG may not provide additional benefits to the clinician as a screening tool in evaluation of adults presenting with acute psychosis. Although EEG recordings can detect a wide variety of pathological conditions, there is a limited number of studies looking at its usefulness in psychiatric patients. As such there is a need to evaluate the usefulness of EEG in our patients, especially in children and adolescents.

1.5. The South African context

South African studies examining the usefulness of EEG among psychiatric patients are limited. Stein (1991) performed a retrospective study at Hillbrow Hospital, Johannesburg, looking at all referrals by the department of psychiatry for EEG between 1986 and 1987 and analysed their value and benefit to patient care. One hundred and forty-five patients met inclusion criteria, with nearly half of them showing clearly demonstrable abnormalities on EEG. Definite epileptiform activity was demonstrated on 50% of his sample while 67 % had localised EEG dysfunction, with 48% of abnormalities found in the temporal lobe areas. In this study, although there was almost a 50% prevalence of EEG abnormalities, the proportion of abnormal EEGs leading to a change in diagnosis was not determined.

Molokomme and Subramaney (2016) did a retrospective record review of patients referred for EEG over 18 months at Sterkfontein psychiatric hospital in Gauteng. In a sample of 85 adult patients, 74 records were normal, 7 were abnormal, 2 were inconclusive and 2 reports were unavailable. Reasons for referral were seizure exclusion (69%) and other reasons (31%) which included treatment non-response and excluding other pathology. Seizure history was reported in 28.2% of the sample population before EEG. EEG abnormalities in 8.2% of the sample population were documented as slowing and dysrhythmia. Only one patient had their diagnosis changed from schizophrenia to temporal lobe epilepsy as a result of an abnormal EEG recording.

1.6. EEG studies in children and adolescents

The EEG in child and adolescent psychiatry may be useful in identifying unsuspected organic cause of intractable symptoms. It has also been found to sometimes be helpful in differentiating between idiopathic epilepsy and symptomatic convulsive disorders, except that certain post-encephalitic conditions can simulate EEG patterns of idiopathic epilepsy (Riviello, 2007).

Practice parameters developed by The American Academy of Child and Adolescent Psychiatry recommend that an EEG is done in assessment of youths with suspected diagnoses of

intellectual disability, autism and other pervasive developmental disorders. On the other hand, a routine EEG study is not recommended in children and adolescents with schizophrenia, depression, bipolar disorder, substance use disorders and oppositional defiant disorder (Birmaher et al., 1998; Bukstein, 2005; McClellan et al., 2007; McDonald et al., 2006; Steiner and Remsing, 2007; Szymanski and King, 1999; Volkmar et al., 1999; Waddell et al., 1999).

The College of Physicians and Surgeons of Ontario (CPSO) have recommended indications to assist with appropriate referral of patients for EEG. Recommended indications include new onset seizures, query seizure, poorly controlled epilepsy, infantile spasms, encephalitis, interval EEG and episodic rage attacks. Swart and Wahab (2010) looked at all EEGs of children from birth to 18 years, at a tertiary care centre for children over a period of two years. The reasons for referral for an EEG were compared to the guidelines by the CPSO for ordering EEGs. They also reviewed the outcomes of the EEGs performed and the actions undertaken by physicians on receipt of the results.

Fifty three percent of EEGs were ordered for reasons in accordance with CPSO guidelines indicating a significant probability of useful clinical information being obtained. EEG abnormalities were found in 49% of the participants in this category. Twenty percent of EEGs were ordered for reasons indicated in the guidelines as not likely to produce useful information. In this category, abnormal EEGs were found in 24% of patients. In patients who had EEGs done for reasons not mentioned in the CPSO guidelines, 55% had an abnormal EEG. Their conclusion was that there was an increased probability of neurological problems in children with severe mental health problems, which might have an impact on their assessment and treatment.

A retrospective study of adolescent psychiatric inpatients at Tara Hospital admitted between 1990 and 1995 was published by Szabo and Magnus (1999). They reviewed all admissions to the ward with a wide range of Axis I, DSM IV psychiatric diagnoses. 4% of the patients (16 out of 360 total admissions) had been diagnosed with complex partial seizures. A definite

complex partial seizure diagnosis was made in 44% of patients who underwent EEG studies, 33% had nonspecific abnormal findings and 22% were normal. The study highlighted that certain clinical features were found to relate to EEG findings.

The EEG, although subject to false-negative results, can be a useful diagnostic adjunct in psychiatric practice, including in the assessment of children and adolescents. The above studies have shown that the EEG has been used extensively in psychiatry to help confirm or rule out various conditions, including epilepsy and organic mental disorders. There is, however, a paucity of published South African literature on EEGs especially in children and adolescents.

CHAPTER 2

AIM, OBJECTIVES AND METHODS

2.1. Aim

The aim of this investigation is to assess the clinical utility of electroencephalographic studies in children and adolescents at Tara Hospital.

2.2. Objectives

1. To describe the reasons for referral of children and adolescents for EEG studies.
2. To determine the prevalence of abnormal EEG results in the study population.
3. To examine how EEG studies have an impact on the diagnostic assessment and management of children and adolescents.

2.3. Hypotheses

1. EEG studies can be useful in child and adolescent psychiatric patients in the diagnostic work-up to confirm or modify clinical diagnoses.
2. EEG studies may assist in child and adolescent psychiatric patients by guiding further management.

2.4. Methods

This is a retrospective descriptive record review of referrals for EEG studies at Tara Hospital in Johannesburg. The period of study was from 01 January 2012 to 31 December 2016.The participants were children and adolescents who underwent an EEG study.

Tara Hospital is a specialised psychiatric hospital in Johannesburg, South Africa. It is a referral hospital for patients from acute psychiatric units requiring long term care. It offers biological and neuropsychiatry services, an eating disorder unit, and a ward for personality disorders. It also has a dedicated child and adolescent psychiatry unit with two separate wards for children

and adolescents, and an outpatient clinic. The unit is run by three child and adolescent psychiatrists and has three registrars on a 6-monthly rotational basis. The staff includes a team comprising of nurses, psychologists, occupational therapists and social workers.

Referrals for EEG studies were received from the children's ward, the adolescent ward and the outpatient clinic at Tara. Files are kept at the clinic for regularly attending patients; or file registry for patients who had not attended the clinic for some time, those who had been transferred to local clinics or their referring institutions once they have been stabilised. Some patient files were not found. All records of EEGs done over the last decade are kept in chronological order at Tara hospital EEG department. There is a fulltime EEG technician at Tara Hospital with considerable experience who does all the EEG recordings. Interpretation of the EEG is done by a neurologist.

Data collected from EEG records includes patient demographics, reason for EEG referral, psychiatric and medical diagnoses, medications, the type of EEG study conducted (i.e. routine, sleep-deprived, or sleep EEG), and EEG findings as reported by a neurologist. Further information was obtained from patients' medical records to determine what decisions were made by the doctors after receiving the results of the EEG.

2.5. Inclusion criteria

All children and adolescents (i.e. all patients under the age of 18) who underwent EEG studies during the study period were included.

2.6 Exclusion criteria

None

CHAPTER 3

STATISTICAL ANALYSIS

Frequencies and their percentages (proportions) were determined for all the categorical data. Where feasible, the categorical data were stratified by abnormal or normal EEG result. Abnormal EEG result was comprised of at least one of the following findings: generalised epileptiform activity, focal epileptiform activity, diffuse slowing, focal slowing and non-specific abnormal changes. Descriptive statistics including mean, median (interquartile ranges) and minimum and maximum values were determined for continuous data. Where feasible, these were stratified by the abnormal or normal EEG result. Some of the continuous measures such as age were stratified into two: 5-12 years and 13-18 years.

The number of participants with a psychiatric diagnosis or on medications before and after EEG was determined and presented graphically using counts and percentages.
Where feasible, proportions between abnormal and normal were compared by the Fishers exact test. All statistical analysis was conducted using SAS Enterprise Guide 7.1 using the PROC FREQ, PROC MEANS and PROC SGPLOT procedures.

CHAPTER 4

ETHICAL CONSIDERATIONS

Permission to conduct the study at Tara Hospital was obtained from the Chief Executive Officer. Ethics approval was granted by the Human Research Ethics Committee (M171040) at the University of the Witwatersrand. Names of patients were not collected to ensure that patient anonymity is maintained. Patients were not interviewed because this was a record review, therefore, informed consent was not required.

CHAPTER 5

RESULTS

5.1. Demographics

A total of 249 participants were enrolled but data were missing on some variables leading to varying totals across the assessed variables. Of the 249 enrolled participants, 243 (98%) had completed data on gender (63% males and 37% females); 248 had completed data on age (57% 5-12 years and 43% 13-18 years); and 248 had complete data on education (57.3% were in primary school, 29.4% were in high school, 2.4% were in special education, and 10.9% had unknown educational status).

Table 2 presents participant characteristics stratified by abnormal and normal EEG results. There were 34 (13%) participants with an abnormal EEG result and the majority were males (70.6%), aged 5-12 years (76.5%) and were in primary school (70.6%). The proportion of abnormal EEGs was significantly higher than the normal in the 5-12 years age-group (76.5% vs. 53.7%, p=0.013).

Table 2: Participant characteristics by EEG result

Variable	Overall N (%)	Abnormal N (%)	Normal N (%)	P value N (%)
Gender				
Female	89 (36.6)	10 (29.4)	79 (37.8)	0.347
Male	154 (63.4)	24 (70.6)	130 (62.2)	
Age (years)				
5-12 years	141 (56.9)	26 (76.5)	115 (53.7)	0.013
13-18 years	107 (43.1)	8 (23.5)	99 (46.3)	
Mean (SD)	11.9 (3.5)	11.1 (3.1)	12.1 (3.6)	
Median (IQR)	12.0 (9.0-15.0)	11.0 (9.0-12.0)	12.0 (9.0-15.0)	
Min-Max	5.0-18.0	6-17	5-18	
Education				
Primary school	142 (57.3)	24 (70.6)	118 (55.1)	0.091
High school	73 (29.4)	4 (11.8)	69 (32.2)	0.015
Special education	6 (2.4)	2 (5.8)	4 (2.0)	0.157
Unknown	28 (10.9)	4 (11.8)	23 (10.7)	0.86

NB: Some variables have missing data

5.2. Clinical characteristics

Of those with abnormal EEG results; 41.2% had a history of seizures, 20.6% had a history of traumatic brain injury, and 41.2% had other comorbid medical conditions (Table 3). Among those with normal EEG results; 23.4% had a history of seizures, 6.5% had a history of traumatic brain injury (TBI), and 20.7% had other comorbid medical conditions. The proportion of abnormal EEGs was higher in patients with a history of seizures (p=0.02), traumatic brain injury (p=0.18) and comorbid medical conditions (p=0.07) compared to those without a history of these conditions. There were more participants without a history of traumatic brain injury in

the group with a normal EEG group relative to the group with an abnormal EEG (70.1% vs. 58.8%, p<0.0001).

Some participants notably had more than one clinical characteristic; eg a history of seizures and a history of TBI or a medical condition. 30 participants had 2 clinical characteristics and 2 had 3 clinical characteristics. The proportion with an abnormal EEG was significantly higher than that of those with a normal EEG in those with more than 1 clinical characteristic (26.5% vs. 11.7%).

Table 3: Clinical characteristics by EEG result

Variable	Overall N (%)	Abnormal N (%)	Normal N (%)	P value
History of seizures				
Yes	64 (25.8)	14 (41.2)	50 (23.4)	0.0275
No	95 (38.3)	11 (32.4)	84 (39.3)	
Uncertain/Not stated	89 (35.9)	9 (26.4)	80 (37.3)	
History of traumatic brain injury:				
Yes	21 (8.5)	7 (20.6)	14 (6.5)	0.1844
No	170 (68.5)	20 (58.8)	150 (70.1)	<0.0001
Uncertain/Not stated	57 (23.0)	7 (20.5)	50 (23.3)	0.0238
Other comorbid medical conditions				
Yes	58 (23.5)	14 (41.2)	44 (20.7)	0.0746
Possible	13 (5.3)	2 (5.9)	11 (5.2)	0.8618
Unknown	176 (71.3)	18 (52.9)	158 (74.2)	<0.001
Participants with >1 clinical characteristic				
	34/247 (13.8)	9/34 (26.5)	25/213(11.7)	0.0206

NB: Some variables have missing data

During the process of data collection, we found that there were certain variables that were common among most of the participants, and other variables that were deemed significant enough to warrant discussion. These variables included ADHD, anxiety disorders, comorbid HIV disease, sleep deprivation, neuroimaging, psychiatric medication and psychoactive substances. Their results have been included below and elaborated on in the discussion section.

Table 4 illustrates the differences between sleep deprived EEGs and routine EEGs. There were thirty-eight sleep deprived EEGs done in our study; three were abnormal, showing mild slowing for age, focal epileptiform activity and generalised epileptiform activity. Of the two participants with epileptiform activity on EEG, one had mesial temporal sclerosis on MRI, and the other had an arachnoid cyst with hydrocephalus on CT scan.

Table 4: Sleep deprived EEGs

Variable	**Overall N (%)**	**Abnormal N (%)**	**Normal N (%)**
Sleep deprived EEG	38 (15.3)	3 (7.9)	35 (92.1)
Routine EEG	211 (84.7)	30 (14.2)	181 (85.8)

There were eight participants who had undergone a brain CT scan or an MRI scan of the brain prior to the EEG. Three of those patients had abnormalities on CT scan; two of those had focal epileptiform activity whilst the other patient had a normal sleep deprived EEG. The participant with the normal EEG had a history of seizures and abnormal left hippocampal morphology which was not diagnostic of mesial temporal sclerosis. This patient however, had antiepileptic medication added to their treatment based on the clinical history of seizures.

Of the other two participants with abnormalities on CT scan, one had an arachnoid cyst on the left temporal lobe with hydrocephalus. The patient had a sleep deprived EEG done which was normal. This patient did not come back for a follow up visit, therefore, we were unable to

determine how the result impacted on the patient's management. The other patient's CT scan showed a right middle cerebral artery infarct in the temporal and parietal lobes. This participant's EEG showed right temporal delta activity, resulting in a diagnosis of temporal lobe epilepsy. The patient was already on antiepileptic medication and, of note, also on treatment for tuberculous meningitis.

One participant had mesial temporal sclerosis on MRI and a history of TBI. The patient had a sleep deprived EEG which showed generalised epileptiform activity, resulting in addition of antiepileptic medication. Of the four remaining patients, two had involutional changes, one had an old infarct, and the other had a left middle cerebral artery (MCA) aneurysm on CT scan. All these patients had normal EEGs, resulting in no change in diagnosis or management. The association between EEG findings and abnormalities found on neuroimaging could not be tested because of the small sample size of patients who had neuroimaging studies done.

Nine patients had a comorbid diagnosis of HIV and were on antiretroviral drugs. The EEG was normal in four of these participants and abnormal in the other five. Of note, the abnormal EEGs showed diffuse slowing in three participants, intermittent occipital slowing in one, and focal epileptiform activity in the remaining participant. The findings led to addition of antiepileptic medication in the patients with occipital slowing, focal epileptiform activity, and in one patient with diffuse slowing.

In addition to a diagnosis of HIV, two of the patients with abnormal EEGs had a history of seizures. One had a history of absences and EEG showed intermittent occipital slowing that is usually associated with absence seizures. Although there was frontal epileptic activity in the other patient, the diagnosis assigned on receipt of the abnormal result was not documented. Both patients had antiepileptic medication added to their treatment. Although more than half of the participants had abnormal EEGs, the numbers were too small to determine the association between the EEG and HIV.

5.3. Psychiatric diagnoses

Overall, the most common psychiatric diagnosis before EEG was attention deficit hyperactivity disorder (53%, n=132) and anxiety disorder (36%, n=90) [Figure 1].

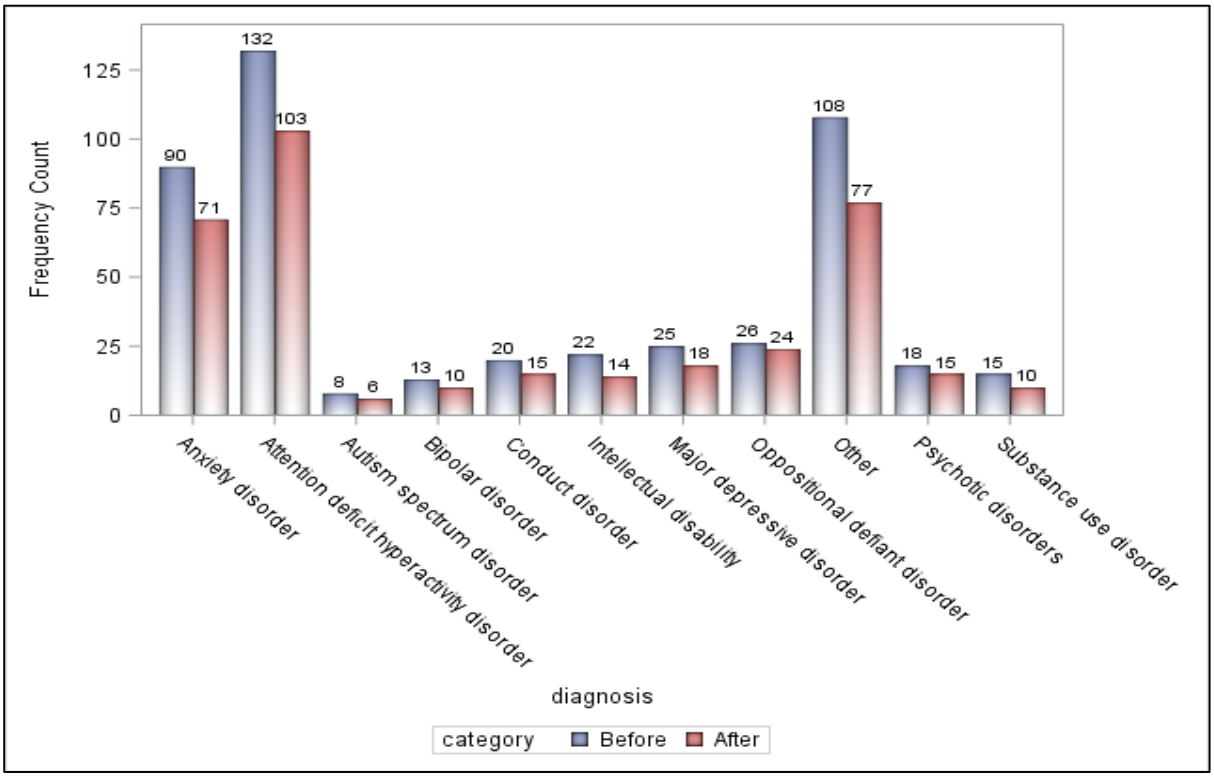


Figure 1: Psychiatric diagnoses before and after EEG

After EEG, although there was a reduction in the number of participants with a diagnosis of attention deficit hyperactivity disorder (41%, n=103) and anxiety disorder (28%, n=71), the two remained the most common diagnoses.

5.4. Medications

The distribution of medications is presented in Figure 2. Commonly prescribed medications before EEG were antidepressants (43%, n=107), methylphenidate (38%, n=97) and anti-epileptic medication (38%, n=95). After EEG, the most common medications remained antidepressants (45%, n=86), methylphenidate (47%, n=86) and anti-epileptic medication

(33%, n=83). There were slight decreases in the number of patients taking all three classes of medications.

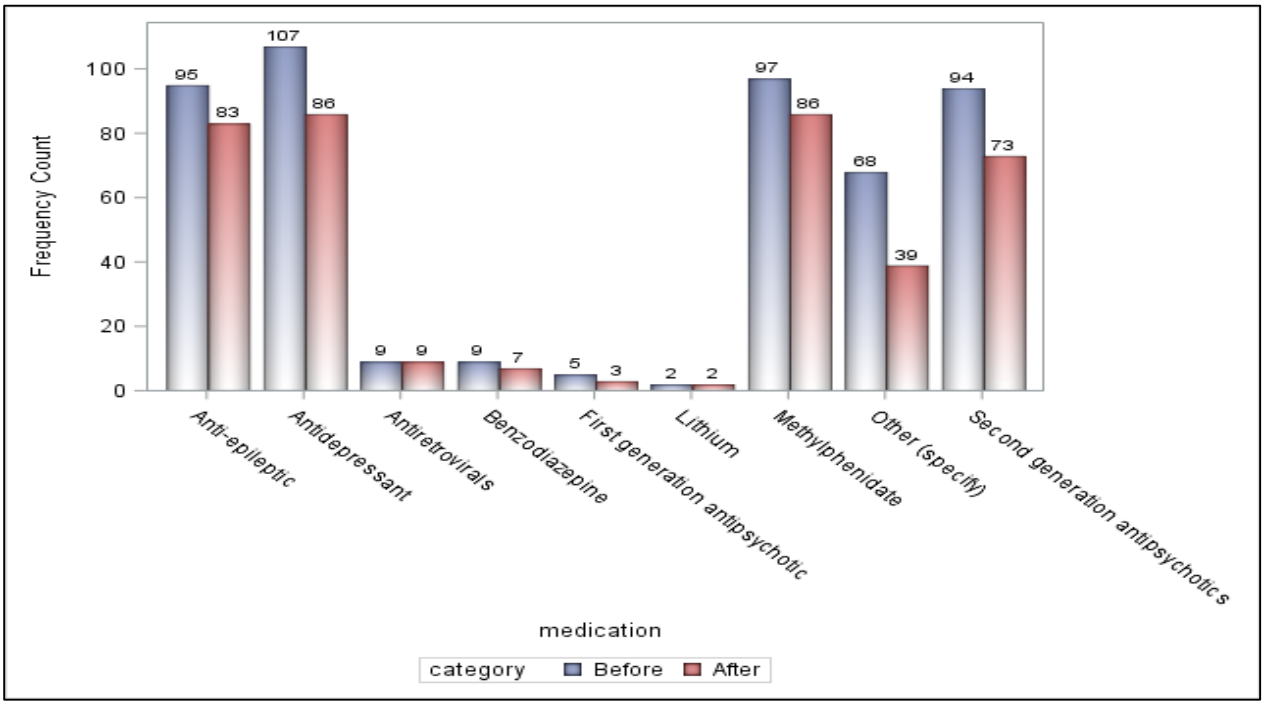


Figure 2: Medication before and after EEG

5.5. EEG and psychoactive substances

Table 5 presents a distribution of the psychoactive substances used by the participants. All those with reported substance use had a normal EEG. Only 19 participants were reported to be using substances either alone, or in combination with others. Of those with substance use, the main substances used were cannabis (79%, n=15), followed by alcohol (32%, n=6), and methamphetamine (21%, n=4).

Table 5: EEG and psychoactive substances (all had normal EEG results)

Variable	N (%)
Alcohol	6 (17.1)
Cannabis	15 (42.9)
Cocaine	3 (8.6)
Heroin	3 (8.6)
Methamphetamine	4 (11.4)
Other	4 (11.4)

5.6. Reasons for referral

Table 6 presents the reasons for referral for EEG. The most common reason for referral for an EEG was poor response to psychiatric treatment (37%, n=91) and to confirm epilepsy (34%, n=84).

Table 6: Reason for referral for an EEG

Variable	N (%)
To confirm epilepsy	84 (34.0)
New onset seizures	2 (0.8)
Poor response to antiepileptic treatment	11 (4.5)
Poor response to psychiatric treatment	91 (36.8)
Other (specify)	59 (23.9)

5.7. EEG results

There were two hundred and fourteen (86%) participants with a normal EEG result (Table 7). The majority of the abnormal EEG results were focal epileptiform activity (27%, n=9), generalised epileptiform activity (24%, n=8) and focal slowing (18%, n=6).

Table 7: EEG Results

Variable	N (%)
Normal	214 (86.3)
Abnormal	34 (13.7)
Abnormal	34
Generalised Epileptiform Activity	8 (23.5)
Focal Epileptiform Activity	9 (26.5)
Diffuse Slowing	4 (11.8)
Focal Slowing	6 (17.6)
Non-Specific Abnormal Changes	3 (8.8)
Other	4 (11.8)

5.8 Clinical impact of the EEG

The EEG resulted in change in management in 16% (n=43) of cases and confirmation of diagnosis in 9% (n=24) of cases. Change in diagnosis occurred in 4% (n=11) of the participants (Table 8). The "other" category included patients whose clinical records could not be found, those who were lost to follow-up, and those whose clinical notes did not indicate what decision was made post-EEG. It was not clear how the EEG result helped in further management of those patients in the "other" category.

Table 8: Clinical impact of the EEG

Variable	N (%)
Clinical impact documented	55 (22.1)
Confirmation of diagnosis	24 (8.8)
Change in diagnosis	11 (4.0)
Change in management	43 (15.8)
Clinical impact not documented	194 (77.9)
Clinical decisions linked to EEG not recorded	132 (53%)
Missing clinical records	6 (24.1%)
Patients lost to follow up	2 (0.8%)

The total number of patients with a documented clinical impact was lower than the three separate outcomes combined. The reason for the discrepancy is because in some patients there was more than one decision made following the EEG, eg both a change in diagnosis and a change in management.

CHAPTER 6

DISCUSSION

6.1. Reasons for referral

It was noted in our study that the most common psychiatric diagnoses among patients referred for EEG were attention deficit hyperactivity disorder (53%, n=132) and anxiety disorders (36%, n=90). The medications prescribed were antidepressants (43%, n=107), methylphenidate (38%, n=97) and anti-epileptic medication (38%, n=95).

6.1.1. EEG in children with ADHD

In our group of patients with ADHD, the high number of referrals for EEG were likely to have been as a result of overlap of symptoms, where the inattentive symptoms of ADHD were thought to be features of absence seizures, or both conditions co-occurring.

Behavioural and attention problems are known to occur in many children with epilepsy, with between 20% and 38% of the children displaying clinical symptoms of ADHD (Kaufmann, Goldberg-Stern, & Shuper, 2009). A prevalence of epileptiform EEG discharges between 5% and 60% was found by Parisi et al. (2010) in their review of ADHD in children with epilepsy. Epileptiform EEG abnormalities in children with ADHD have been quoted to have a predictive value of 14% for development of subsequent seizures. Epilepsy syndromes associated with ADHD include childhood absence epilepsy, rolandic epilepsy, frontal lobe epilepsy, and epileptic syndromes with nocturnal seizures predisposing to ADHD-like behaviour (Parisi et al., 2010). A number of mechanisms may contribute to the high rate of ADHD symptoms in children with epilepsy. These may include: effects of brain pathology underlying both the ADHD symptoms and epilepsy, the effects of chronic seizures, antiepileptic medication, and the effects of non-convulsive epileptiform discharges on vigilance, memory, and processing speed (Torres, Whitney, & Gonzalez-Heydrich, 2008).

Powell et al. (1997) reported a case of a father and son, both with orbitofrontal epilepsy presenting with symptoms of ADHD. Both had normal EEGs and brain MRIs, but a single photon emission computed tomography (SPECT) scan revealed left orbitofrontal hypo-perfusion in the father and bilateral hypo-perfusion defects in the son. The father's symptoms improved significantly on carbamazepine, which was later changed to oxcarbazepine due to side effects. His son's symptoms, on the other hand, decreased in frequency and antiepileptic medication was deferred.

In a review done by Williams et al. (2016), they found studies which suggested that in patients with epilepsy the inattentive presentation of ADHD was more prevalent than the combined presentation. The rates of the inattentive presentation ranged from 24% to 52%, while the combined presentation had rates between 3% and 11%. The prevalence of ADHD symptoms in children with epilepsy has been found to be higher than rates of ADHD in the general population (12-39% vs 3-5%), with predominance of inattentive symptoms (Torres et al., 2008). In addition, other studies generally agree that impairment in attention is likely to be found in patients with generalised epilepsy than focal epilepsy (Schubert, 2005).

This is in contrast to Shermal et al. (2007) who found that in a group of children with severe epilepsy, there was a higher likelihood of generalised epilepsy in the children with combined ADHD. Their study found a higher proportion of children with generalised epilepsy in the ADHD group with combined presentation than the group with no ADHD (25.7% vs 14.4%, $p = 0.008$). Although there were conflicting findings on the correlation between ADHD symptoms and type of epilepsy in the above studies, the association between epilepsy and ADHD is quite clear.

In our study, the subtypes of ADHD were often not recorded in the clinical files and as a result, we could not assess whether there was a relation between the subtypes of epilepsy and ADHD. However, quite a number of the participants diagnosed with ADHD were referred for EEG because they had symptoms which were thought to be due to absence seizures. It can therefore

be extrapolated that the majority of these patients had the inattentive ADHD subtype, in line with observations by Williams et al. (2016) and Torres et al. (2008).

One patient in our study was thought to have seizures induced by methylphenidate. Their EEG was normal, but the patient nonetheless had their methylphenidate switched to atomoxetine. Gucuyener et al. (2003) assessed the safety of methylphenidate in children and adolescents with ADHD: one group with ADHD and epilepsy, and the other group with ADHD and EEG abnormalities but no seizures. The group with EEG abnormalities and no seizures had no new seizures on methylphenidate, and there was no significant increase in the seizure frequency in the group with epilepsy. An additional finding was a decrease in EEG abnormalities or normalisation in some patients from both groups, associated with the administration of methylphenidate. Clarke et al. (2002) found similar results in their study on the effects of stimulant medication in children with ADHD. They found changes in the EEG that progressed towards normalisation and concluded that this trend was likely due to cortical arousal from the stimulant medication, resulting in normalisation of brain activity.

Results of studies by some authors suggest that patients with ADHD and benign epilepsy with centro-temporal spikes (BECTS) are at a high risk of seizures when they are treated with methylphenidate (Millichap, Millichap, & Stack, 2011). This is similar to the findings reported by Hemmer et al. (2001) in a study of children without clinical epilepsy treated with methylphenidate for ADHD. Seizures were found to occur in 16.7% of those with BECTS, in 10% of the children with epileptiform discharges, and in 0.6% of children with no epileptiform discharges.

We found two patients with benign epilepsy with BECTS in our study. One was on methylphenidate while the other was not on any medication. The patient on methylphenidate was continued on it, and both participants had antiepileptic medication added to their treatment. The patient who was not on any medication had a history of seizures, whilst the one on

methylphenidate had no documentation of previous seizures. This observation might suggest that methylphenidate contributed in raising that patient's risk of seizures, similar to the finding in studies by Hemmer et al. (2001) and Millichap et al. (2011).

In cases where seizures are precipitated by medication it is not surprising to find a normal EEG. However, other causes of seizures should without a doubt be considered and explored. Because a normal EEG does not rule out epilepsy, in certain cases clinicians need to use their clinical acumen in formulating a management plan. It is clear from our study and other authors that the EEG is necessary in the evaluation of children and adolescent with ADHD because of various factors: including the commonality of symptomatology between epilepsy and ADHD, the effect of stimulant medication on the seizure threshold, and the additional risk of seizures in children who are on psychostimulants, particularly those with ADHD and centro-temporal spikes. Given the above findings, it is prudent therefore to exercise caution in children with seizure tendencies when given stimulants to treat ADHD. On the other hand, development of seizures in those being treated with stimulant medication cannot be entirely attributed to stimulants. Other factors like nocturnal seizures, subclinical seizures or inadequately controlled epilepsy should be considered, which would be compounded by lowered seizure threshold caused by stimulant medication.

6.1.2. EEG in children with anxiety disorders

Anxiety has been recognised as a common feature in patients with epilepsy, presenting as an ictal, postictal, or inter-ictal phenomena. It is important therefore, to distinguish between anxiety due to epilepsy and co-morbid anxiety in patients with epilepsy. Diagnosis of anxiety and epilepsy, including differentiating between the two remains a challenge in children. This is especially the case in children who present with irritability and disruptive behaviour (Gaitatzis, Trimble, & Sander, 2004).

There appears to be higher risk of anxiety in patients with seizures arising from the temporal lobe than other types of epilepsies. Anxiety presenting as an ictal phenomena has been reported

in about 20% of patients (Beyenburg, Mitchell, Schmidt, Elger, & Reuber, 2005). Seizure onset can be preceded by pre-ictal anxiety by hours or days. Prevalence of inter-ictal anxiety has been found to be between 10% and 25% in community and hospital based studies of epilepsy (Jackson & Turkington, 2005), with other authors also reporting rates between 20% and 30% (Beyenburg et al., 2005).

The high rate of patients with anxiety disorder referred for an EEG in our study indicates the problem that clinicians encounter in differentiating anxiety from epilepsy. Therefore, based on literature reports and our findings, screening patients presenting with anxiety for epilepsy should not be neglected. This is especially true in the paediatric population where elicitation of symptoms and the presentation of conditions is often not straightforward, making their diagnosis difficult. The EEG should undoubtedly be considered if there is suspicion of an underlying ictal process presenting as anxiety.

6.1.3. EEG and HIV

In our study, abnormal EEGs in the HIV positive patients mainly revealed slowing, with only one patient having a focal epileptic pattern. Diffuse slowing in these patients most likely indicates encephalopathic changes.

HIV-associated progressive encephalopathy (HPE) can be seen in children, and is characterised by cognitive, behavioural and motor features. The frequency of HPE in children is between 12% and 67% overall, with a prevalence rate of 50% in children who are not on antiretroviral medication. Chiriboga et al. (2005) found an HPE and arrested HPE prevalence of 1.6% and 10% respectively, in children with HIV encephalopathy taking antiretrovirals. There was also a 23% relapse rate of HPE in a group of patients who had previously been diagnosed with HPE. It is therefore imperative that clinicians obtain a detailed history and perform a full physical examination and correlate the findings with EEG results.

In a study by Kellinghaus et al. (2008) of HIV-infected children with neurological involvement, the EEG was consistent with encephalopathy in 24% of patients. Only one patient had epileptiform activity in their study, as was the case in ours. Vigliano et al. (1994) assessed the diagnostic value of the EEG on HIV infection in childhood. There were two hundred and thirty-eight EEGs performed and divided into 3 groups according to the patient's disease state and age: those with an indeterminate HIV result, those with asymptomatic infection, and those with symptomatic disease. All the EEG results were normal in the indeterminate group, the asymptomatic group, and the controls. The EEG results in the symptomatic group showed significant changes related directly to HIV infection.

In the non-encephalopathic patients, there were characteristic EEG patterns that could be secondary to central nervous system damage by the HIV virus. All these patients had normal CT scans and no neurological signs. The authors suggested that because the EEG findings in this group preceeded the onset of neurological signs, the EEG might have value in predicting onset of neurological symptoms. There were nine patients in the encephalopathic group with abnormal EEGs, and seven of those had abnormal CT scan findings. They identified three EEG patterns: pattern reflecting an acute inflammatory vasculitis, pattern suggestive of neuronal loss and reduced dendritic arborisations, and a pattern seen mostly in younger children already on antiretroviral therapy. Their finding further demonstrated that the EEG can serve as a diagnostic tool in the patients with HIV and clinical signs.

In the Cohort Study of HIV-Associated Seizure and Epilepsy (CHASE), the value of the EEG in adult Zambian patients with a high HIV prevalence was assessed (Siddiqi et al., 2015). There was a 65% rate of abnormal EEGs, and patients with advanced HIV infection were more likely to have an abnormal EEG than those with early stages on infection.

It could be extrapolated in our study that the patients with abnormal EEGs had advanced HIV infection because they were already on antiretroviral treatment. It would have been helpful, however, if details of their HIV staging were available, to determine if there was a correlation between abnormal EEG and stage of infection, as suggested by the above studies.

In contrast, in a study by Harrison et al. (1998) the EEG was interpreted as showing abnormality in 13% of seronegative participants, 8% of asymptomatic HIV infected participants, and 24.5% of patients with AIDS. These findings were thought to be of borderline significance but the rate of abnormal EEGs was still higher in the patients with advanced HIV compared to the asymptomatic group.

HIV disease can present with a myriad of CNS abnormalities. Studies are not in agreement on EEG findings in HIV patients. However, if there is suspicion of HIV-related pathology, an EEG might be a useful investigative tool particularly in advanced HIV disease. We could not the determine statistical significance of the impact of HIV status on EEG findings in our study due to small numbers. However, our data suggests that the most likely EEG finding in HIV patients is diffuse slowing, as a manifestation of HIV encephalopathy.

6.1.4. EEG and history of previous seizures and other medical conditions

Our study confirms what has been found in other related studies. The likelihood of an abnormal EEG result was found to be higher in patients with a known history of seizures ($p= 0.01$), TBI ($p= 0.07$), and comorbid medical conditions ($p= 0.03$).

There were similar findings in Szabo's study, where 44% of the 36 patients who underwent EEG studies had abnormal results (Szabo and Magnus, 1999). In contrast to our study, the patients enrolled in his study were all inpatients, and all those with abnormal EEG had clinical suspicion of complex partial seizures on history. Lam et al. (1988) also found that a history of epilepsy, recent seizure and presence of organic factors identified in history or physical

examination, had a significant association with an abnormal EEG. In other studies, common reasons for referrals for EEG studies were: query seizures, poorly controlled epilepsy and previous abnormal EEG; with abnormal results found in 50%, 72% and 61% respectively (Swart & Wahab, 2010).

Our study had somewhat similar results, with a high percentage of abnormal EEGs in those with history of seizures, traumatic brain injury and other medical conditions. The results were found to be statistically significant in patients with a history of seizures and traumatic brain injury. Given our findings and those of other authors ((Lam et al., 1988; Swart & Wahab, 2010; Szabo & Magnus, 1999), the EEG is without doubt an essential investigative tool in patients with underlying medical conditions and brain abnormalities especially when there is poor response to medication, to screen for post traumatic epilepsy after a TBI and in cases where subclinical seizures are suspected.

6.1.5. EEG and neuroimaging

Eight participants in our total sample had undergone neuroimaging studies, which yielded various structural abnormalities. Four of these patients had abnormal EEGs. This finding could not be analysed for statistical significance, as the numbers were too small.

The CHASE study by Siddiqi et al. (2015) reported that abnormal imaging findings were associated with an abnormal EEG. In their study, abnormal imaging was found in 87% of those with an abnormal EEG vs 50% in those with a normal EEG. Findings from other studies have been conflicting, with other authors suggesting no correlation between the EEG and findings on imaging (Doescher et al., 2006; Raybould et al., 2012).

Doescher et al. (2006) did a study that looked at the relationship between EEG findings and MRI in children with new onset seizures. MRI results were abnormal in 32.6% of the participants; the MRI was abnormal in 42% of the fifty patients with a normal EEG, whilst in

21% of patients the MRI and EEG were both abnormal. Similar to Doescher et al. (2006), there was no significant correlation found between EEG findings and imaging in a study by Raybould et al. (2012). For patients who had an MRI done, only 4.3% had both an abnormal EEG and abnormal MRI, while 16.3% had an abnormal MRI and a normal EEG. Of those who had a CT scan done, 2.6% had both abnormal EEG and CT, 28% had an abnormal CT but a normal EEG.

It is evident from the above studies that an abnormal neuroimaging finding does not necessarily predict an abnormal EEG finding. The decision to refer patients for EEG should also be guided by history and clinical presentation.

6.1.6. Sleep deprived EEGs

In our study, there was confirmation of diagnosis and change in management for a participant with mesial temporal sclerosis on MRI who had a sleep deprived EEG. The patient's EEG showed generalised epileptiform activity. The other two patients with abnormal sleep deprived EEGs were lost to follow-up.

Sleep deprivation is known to precipitate seizures as described by Bennet in 1963, in four healthy pilots who had a seizure following sleep deprivation. The activating effect of sleep deprivation occurs in all age groups but seems to be as high or higher in children. Sleep deprived activation also occurs in patients taking antiepileptic medication (Mendez & Radtke, 2001).

A study by Geller et al. (1969) of 25 children with epilepsy and normal EEGs found that after sleep deprivation, there was a 32% activation rate. They repeated EEGs on seven of the eight patients who had activation after sleep deprivation, with no abnormalities found on the repeat EEG; ruling out the possibility of sampling effect for the effects of sleep deprivation. Roupakiotis et al. (2000) found activation rates of 22.6% in their study sample. They also

compared the activation rates in patients with definite or clinically suspected epileptic seizures, and in a group with other disorders (eg syncope, myopathy, multiple sclerosis, psychiatric disorders, etc). Sleep deprivation resulted in a 12% activation rate in the group with other disorders.

This highlights the importance of doing a sleep deprived EEG study to detect inter-ictal discharges that would otherwise be missed in routine EEGs, particularly in cases where there is high suspicion of a seizure disorder. In our study sleep deprivation did not appear to improve the chances of finding an abnormal EEG. This observation should be interpreted with caution as we did not have previous routine EEGs for comparison. In addition, our numbers were small and statistical significance could not be calculated.

6.1.7. EEG and clozapine

In the current study only two patients were on clozapine; one had a normal EEG, whilst the other patient's EEG showed diffuse slowing. EEG changes commonly associated with clozapine include both generalised slow activity and spike wave discharge in non-epileptic patients. Seizures are more likely to occur at high doses or with rapid dose increases.

Welch et al. (1994) did a study looking at clozapine-induced seizures and EEG changes in adult patients. 35 patients with treatment resistant schizophrenia initiated on clozapine were enrolled in the study. EEGs were done at baseline, when the dose reached 300mg and repeated every three months at minimum. All baseline EEGs were normal. An EEG was done immediately when there were seizures, clinical deterioration, or change in cognitive testing. They found that 74% of the patients had EEG abnormalities at some time during clozapine treatment, the majority showing mild to moderate slowing. The conclusion from the study was that the EEG is a sensitive indicator of clozapine toxicity.

Similarly, Chung et al. (2002) found a high proportion of abnormal EEGs after clozapine treatment. All the patients had normal baseline EEGs and additional EEGs were done during the course of clozapine treatment. There were abnormal EEGs in 62% of their sample, with two patients having seizures. Most abnormalities seen were non-specific slow waves. Centorrino et al. (2002) compared EEG abnormalities in patients on typical and atypical antipsychotics. Clozapine treated patients had the highest percentage (47.1%) of EEG abnormalities that those on other agents.

We could not make any inferences on our two patients' results because of the very low participant number. There was also no record of baseline EEGs and clozapine dosages were not recorded. Clozapine is known to lower seizure threshold at high doses. Baseline EEGs for patients on clozapine might help differentiate clozapine toxicity vs pre-existing seizure disorders. Studies reviewed generally agree that the EEG provides useful information about patients on clozapine. As clozapine is used primarily for treatment refractory psychotic disorders, the feasibility of EEGs as part of the mandatory baseline work-up, should be considered, and even more so for patients with other risk factors for seizures.

6.1.8. EEG and psychoactive substances

The commonest psychoactive substance used in our sample was cannabis, followed by alcohol. Two participants used multiple substances: cannabis, alcohol, methamphetamines, and heroin. All the patients using substances had a normal EEG.

Some authors have looked at qualitative EEG findings in patients abusing substances. Trudeau et al. (1999) found a high likelihood of right temporal abnormalities and excess frontal alpha activity in adult stimulant users compared to non-users. These abnormalities were also seen in those using stimulants and cannabis together. The significance of the temporal abnormalities in the study was unclear. In other studies EEG abnormalities have been linked to probability of staying in substance abuse treatment. The authors found that adult patients with excess alpha

activity stayed in treatment longer than those with beta activity (Ceballos, Bauer, & Houston, 2009).

The relevance of findings in these studies is unclear in the absence of comparative studies in children and adolescents. The studies by Ceballos et al. (2009) and Trudeau et al (1999) were done in adults and the findings cannot be necessarily assumed to apply in the paediatric population. Based on our findings and those of other authors, it is recommended that EEG studies in patients using substances be performed only if there is a suspicion of psychiatric manifestation of an underlying medical condition.

6.2. Prevalence of abnormal EEGs

The highest proportion of abnormal EEGs in our study was found in the 5-12 years age group compared to the 13-17 years age group (75% vs 54%). We found two patients aged 6 years and 11 years old with BECTS. The other EEG abnormalities found in the 5-12 years age group were generalised epileptiform activity (19%), focal epileptiform activity (19%), focal slowing (9%) and generalised slowing (6%).

The onset of seizures occurs in childhood in over 50% of cases, with a 0.7% to 1% prevalence rate. The most common type of epilepsy in children is thought to be BECTS, with an age of onset between 1 and 14 years, and the majority presenting between 7 and 10 years (Gkampeta, Fidani, Zafeiriou, & Pavlou, 2015; Pavlou, Gkampeta, Evangeliou, & Athanasiadou-Piperopoulou, 2012). Also known as benign rolandic epilepsy, it is a type of childhood epilepsy which gives rise to epileptiform discharges in the mid-temporal and central regions of the EEG. The seizures occur infrequently and are nocturnal in 50% of patients (Riviello, 2007).

Cavazzuti et al. (1980) found a 3.54% prevalence of epileptiform patterns in a cohort of children with no history of seizures and no neurological symptoms between the ages of 6 and

13 years old (20.6% of abnormalities were rolandic spikes). A higher prevalence of epileptiform abnormalities of 6.1% was reported by Richter et al. (2002) in children between ages 5 and 16 years with ADHD, compared to the 3.54% found by Cavazzuti et al. (1980) in the general paediatric population. An epileptiform abnormality rate of 5% was found by Okubo et al. (1994) in healthy children aged 6-12 years, with the majority consisting of centro-temporal spikes (69.8%).

The prevalence of EEG abnormalities in our study was 17.2% in the 5-12 years age group, 7.5% in the 13-18year age group, and the overall prevalence in the entire study population was found to be 13.6%. The figures are not alarming and are likely representative of the prevalence in the general population. EEG abnormalities in our study also seem to be somewhat similar to findings by Cavazzuti et al. (1980), with patterns consisting of spike and slow wave complexes, focal spikes including rolandic spikes.

6.3. The impact of the EEG

The EEG resulted in a demonstrable impact of 22.1%; with change in management, confirmation of diagnosis, and change in diagnosis in 15.8%, 8.8% and 4% respectively. In the remainder (77.9%), the clinical impact was not documented in 53%, there were missing clinical records in 24.1% of cases and 0.8% were lost to follow up.

In the study by Molokomme and Subramaney (2016) 1.2% of the participants had their diagnosis changed from schizophrenia to temporal lobe epilepsy, while another 1.2% with previous seizure history but whose EEG result was unavailable had antiepileptic medication prescribed after a seizure was witnessed. Abnormal EEGs were found in 8.2% (n=7) of their study sample. 31% of EEGs were abnormal in a study by Warner et al. (1990), resulting in a change in diagnosis in 1.7% of cases. O'Sullivan et al. (2006) did not find significant clinical impact on patient management after EEG.

Although only 13.7% of EEGs in our study were abnormal, in some cases a normal EEG was found to be a useful aid to clinicians. Even though a normal EEG does not exclude a diagnosis of epilepsy, in some patients it led to confidence that the patient's symptoms were likely not due to epilepsy, and at times informed medication choices.

CHAPTER 7

LIMITATIONS OF THE STUDY

This was a retrospective study, therefore, we had no control on information provided on referral forms. Reasons for referral were often not stated and it was at times unclear how the EEG impacted on management because clinical notes were sometimes unclear. Incomplete referral forms with no reason for referral and missing clinical data might have hindered more accurate interpretation of the EEG.

Some patient records were missing and other patients did not return for follow up visits after the EEG study was done. As a result, we were unable to determine what actions were taken after the EEG was done. In the majority of cases documentation of the clinical decisions made after the EEG was not clear, making it difficult to determine how the EEG helped the clinicians with diagnosis and treatment. Another caveat is that because a negative EEG result does not exclude an epilepsy diagnosis, the number of patients with epilepsy could be higher than the proportion whose diagnosis was confirmed by an abnormal EEG result.

The small sample size of our study, and the fact that Tara is a tertiary psychiatric hospital, limits the generalisability of our findings. In addition, the results are from one hospital and the sample is not representative of the general paediatric psychiatric population. Another limitation to our study is that there were no exclusion criteria, with possible bias of our results. However, the inclusion of all records was done to examine the prevalence of EEG abnormalities in our cohort and reasons for referring the patients for the EEG studies in line with two of the study objectives. In addition, some of the missing variables were minor and would not have significantly affected the objectives of the study. Exclusion of the participants with missing variables would have drastically reduced our sample size and further limited the usefulness of the study.

CHAPTER 8

CONCLUSION

It is clear from our study and other literature that the EEG adds value in the diagnostic work-up of child and adolescent psychiatric patients and guide further management. Most participants in our study were referred for an EEG to confirm epilepsy or because of poor response to psychiatric medication.

The number of patients with diagnoses of ADHD and anxiety disorder who were referred for EEG underscores the difficulty that clinicians often encounter in making definitive diagnoses in children and adolescents, owing to symptom overlap, commonality of symptoms in certain conditions; for instance, behavioural manifestations of epilepsy presenting as attention difficulties and anxiety occurring as part of epilepsy. The EEG is therefore without a doubt a very useful for clinicians and minimally invasive making it an ideal investigative tool in the paediatric population.

Studies have found the prevalence of EEG abnormalities to be higher in ADHD than in the general population. Studies on the prevalence of EEG abnormalities in paediatric patients with anxiety are lacking. Although we could not compare our findings with other studies, epileptiform abnormalities in children are recognised with centro-temporal spikes accounting for most of the abnormalities. It is therefore recommended that clinicians maintain a high index of suspicion in children presenting with symptoms that might suggest an epileptic process, in which case an EEG might be indicated.

Most studies have demonstrated that EEGs done with a low index of suspicion of an underlying medical condition causing psychiatric symptoms, have an unsurprisingly low clinical yield. It is therefore recommended that clinicians should therefore take this into consideration when deciding on whether the EEG will help clarify diagnoses. It is important to carefully select patients in whom the EEG will help confirm or clarify diagnoses or have an impact on further

management. To increase diagnostic yield, techniques like sleep deprivation should be considered, especially where there is a high suspicion of an underlying ictal process.

Our study and those by other authors clearly demonstrate that although the EEG is mainly used in assessment of epilepsy, it is a valuable tool in the psychiatric setting. The current study showed that in our setting a negative EEG can also guide management by clarifying diagnoses in cases where there is confusion between patients' symptoms which is common in the paediatric population. Despite some short-comings of this study, the value of the EEG in a psychiatric setting cannot be overlooked. It is especially useful in the paediatric population where there is frequently a complexity of symptoms, comorbidity, and lack of clarity of psychiatric diagnoses.

We have not found published studies on EEG studies in the South African paediatric population within the psychiatric setting. As far as we know this is the first such study done in South Africa, therefore, more studies in other locations would be useful to assess if our findings could be duplicated or indeed challenged.

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APPENDIX 1

EEG referral form

TARA, THE H MOROSS CENTRE

WP/41

REQUEST FOR E.E.G. - Complete in duplicate
- Send Patient's file

E.E.G. NO._____

PATIENT_____ DATE_____ TIME:_____ RH/LH

AGE:_____HOSP. NO.:_____ HOURS SLEPT:_____ _____

OCCUPATION:_____ WARD:_____ REFERRED BY:_____

LAST MEAL:_____ PROVISIONAL DIAGNOSIS:_____

MEDICATION: USUAL _____

TODAY:_____

HISTORY:(Brief)

_____

_____

_____

_____

_____

_____

_____

E.E.G. REPORT:

_____

_____

_____

_____

_____

_____

_____

_____

_____

/JV
16.2.95.(TH/659)

APPENDIX 2

Data Sheet

Identifying number: _____

Demographic details

Gender:

Female	1
Male	2
Unknown	3

Age: 

Education:

Primary School	1
High School	2
Special education	3
Unknown	4

Clinical details

Psychiatric diagnosis:

	Before EEG	After EEG
Attention deficit hyperactivity disorder	1	15
Oppositional defiant disorder	2	16
Conduct disorder	3	17
Anxiety disorder	4	18
Major depressive disorder	5	19
Bipolar disorder	6	20
Mood disorder d/t another medical condition	7	21
Substance use disorder (specify):	8	22
Intellectual disability	9	23
Autism spectrum disorder	10	24
Schizophrenia	11	25
Psychotic disorder d/t another medical condition	12	26
Other psychotic disorders	13	27
Other (specify):	14	28

History of seizures:

Yes	1
No	2
Uncertain	3
Not stated	4

History of traumatic brain injury:

Yes	1
No	2
Uncertain	3
Not stated	4

Other comorbid medical conditions:

Yes	1
No	2
Unknown	3

List of comorbid medical conditions:

_____

_____

Substance use:

Alcohol	1
Cannabis	2
Cocaine	3
Heroin	4
Methamphetamine	5
Other (specify):	6

Medication:

	Before EEG	After EEG
Methylphenidate	1	10
First generation antipsychotic	2	11
Second generation antipsychotics	3	12
Anti-epileptic	4	13
Lithium	5	14
Antidepressant	6	15
Benzodiazepine	7	16
Antiretrovirals	8	17
Other (specify)	9	18

Reason for referral for an EEG:

To confirm epilepsy	1
New onset seizures	2
Poor response to antiepileptic treatment	3
Poor response to psychiatric treatment	4
Other (specify)	5

EEG result:

Normal	1
Abnormal	
- Generalised epileptiform activity	2
- Focal epileptiform activity	3
- Diffuse slowing	4
- Diffuse excess activity	5
- Non-specific abnormal changes	6
- Other (specify)	7

Clinical Impact of EEG:

Confirmation of diagnosis	1
Change in diagnosis	2
Change in management	3
Other (specify)	4

APPENDIX 3

Approval letter from research site

GAUTENG PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

DEPARTMENT OF HEALTH
TARA the H. Moross Centre
Private Bag X7
RANDBURG 2125
(011) 535-3000
(011) 535-3026

30 August 2017

For attention: **Dr Madigoe and Dr Otieno**

Dear Doctors

Re: Application by Dr O. Mahuma to do research at Tara Hospital

I hereby request permission for Dr O. Mahuma to conduct research at Tara Hospital. Dr Mahuma is a registrar in the Department of Psychiatry and is currently enrolled as a Masters student at the University of the Witwatersrand. Her supervisors are Dr T. Madigoe and Dr N. Tema. It is a retrospective record review and the title is: **The Clinical Utility of EEG studies in children and adolescents at Tara Hospital**. She will apply to the Human Research Ethics Committee at the University of Witwatersrand for ethics approval. No concerns were raised by the Tara Research Committee.

.....

Date: 01.09.2017

Dr Ronelle Price-Hughes

Chairperson- Tara Research Committee

Recommended/Not Recommended (pending ethics)

.....

Dr Thebe Madigoe

Clinical Head

Date: 1/9/17

Approved/Not Approved (pending ethics clearance)

.....

Dr Florence Otieno

CEO

Date: 4/9/17

Commence only once the ethics clearance certificate is presented to the hospital

APPENDIX 4

HREC ethics clearance certificate

R14/49 Dr Othelia Omphemetse Mahuma

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M171040

NAME: (Principal Investigator)	Dr Othelia Omphemetse Mahuma
DEPARTMENT:	Psychiatry Tara Hospital
PROJECT TITLE:	The Clinical Utility of Electroencephalographic Studies in Children and Adolescents at Tara Hospital
DATE CONSIDERED:	27/10/2017
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Dr N. Tema and Dr T. Madigoe
APPROVED BY:	Professor P. Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	30/10/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 301, Third Floor, Faculty of Health Sciences, Phillip Tobias Building, 29 Princess of Wales Terrace, Parktown, 2193, University of the Witwatersrand. I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to resubmit the application to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. In this case, the study was initially reviewed in October and will therefore be due in the month of October each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature Date

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

APPENDIX 5

Plagiarism declaration

PLAGIARISM DECLARATION TO BE SIGNED BY ALL HIGHER DEGREE STUDENTS

SENATE PLAGIARISM POLICY: APPENDIX ONE

I Othelia Omphemetse Mahuma (Student number: 9401612v) am a student registered for the degree of MMed Psychiatry in the academic year 2018.

I hereby declare the following:

- I am aware that plagiarism (the use of someone else's work without their permission and/or without acknowledging the original source) is wrong.
- I confirm that the work submitted for assessment for the above degree is my own unaided work except where I have explicitly indicated otherwise.
- I have followed the required conventions in referencing the thoughts and ideas of others.
- I understand that the University of the Witwatersrand may take disciplinary action against me if there is a belief that this is not my own unaided work or that I have failed to acknowledge the source of the ideas or words in my writing.
- I have included as an appendix a report from "Turnitin" (or other approved plagiarism detection) software indicating the level of plagiarism in my research document.

Signature: _____ Date: _____

1

APPENDIX 6

Turnitin report

Turnitin Originality Report

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