FEATURES, MANAGEMENT AND COST OF ILLNESS OF EPILEPSY IN AHMADU BELLO UNIVERSITY TEACHING HOSPITAL (ABUTH), KADUNA: A TWO YEAR RETROSPECTIVE STUDY

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DECLARATION

I declare that the work in the thesis entitled 'Features, management and cost of illness of epilepsy in Ahmadu Bello University Teaching Hospital (ABUTH), Kaduna: A two year retrospective study' has been performed by me in the Department of Pharmacology and Clinical Pharmacy under the supervision of Dr. (Mrs) H.O. Kwanashie, Dr. J.A. Anuka and Dr. T.L. Sheikh. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this thesis was previously presented for another degree or diploma at any university.

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CERTIFICATION

This thesis entitled “FEATURES, MANAGEMENT AND COST OF ILLNESS OF EPILEPSY IN AHMADU BELLO UNIVERSITY TEACHING HOSPITAL (ABUTH), KADUNA: A TWO YEAR RETROSPECTIVE STUDY” by OMONIWA, Aduke Elizabeth meets the regulations governing the award of the degree of Master of Science in Pharmacology of Ahmadu Bello University, and is approved for its contribution to knowledge and literacy presentation.

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TO

ALMIGHTY GOD

AND

MY LOVED ONES

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Abstract

Epilepsy is one of the most common neurological disorders worldwide with prevalence varying from place to place. In Nigeria, the prevalence is between 0.53% and 3.7% of the population depending on several factors including the part of the country and its respective development/available healthcare facilities. There is limited information with regards to epilepsy and indeed other mental/neurological disorders in this part of the country. This study was aimed at documenting features (including cost of treatment) and management of epilepsy in Ahmadu Bello University Teaching Hospital (ABUTH) Kaduna.

A retrospective approach using patients' folders and other hospital records of patients attending psychiatry clinic between January 2003 and December 2004 was used. A sample comprising of epileptics who had attended clinic for at least six times during the study period (total being 91), was studied in detail using a structured data based instrument. Data was analysed using Statistical Package for the Social Scientist (SPSS). The five most common mental/neurological disorders identified at ABUTH were schizophrenia (46.6%), depression (13.7%), seizure/epilepsy (12.1%), anxiety (8.0%) and bipolar affective disorder (7.5%).

There were more male epileptics (52.7%) than female (47.3%) and while half of the patients were below 20 years at first contact with orthodox facilities, a larger proportion (74.5%) were less than that age at onset of epilepsy. Most patients were single (71.9%) and only 18.0% had advanced beyond junior secondary school. Common risk factors identified for epilepsy in this study were febrile convulsion (52.6%) and family history (23.7%). Signs and symptoms presented by these patients

included tonic-clonic muscle contraction and relaxation (76.0%), loss of consciousness (67.0%), psychic symptoms (30.8%), frothing in the mouth (19.8%) and urinary incontinence (17.6%). Relevant laboratory investigations included electroencephalography (EEG), computerized tomography (CT) scan and skull X-ray. Generalized seizures accounted for 59.3% of the patients, partial seizures for 25.3% and 15.4% for others (mixed and unclassified). Mental/neurological comorbid disorders were uncommon (23.0%) and included anxiety, psychosis, mental retardation and abnormal behaviour (personality changes). These various indices are similar to what has been reported for other parts of Africa and elsewhere.

Pharmacological management of these patients included the use of antiepileptic drugs (AEDs), adjuncts, drugs to treat comorbid states and drugs to treat side effects. AEDs used were carbamazepine (91.2%), benzodiazepines (49.4%), barbiturates (24.2%), phenytoin (7.7%), sodium valproate (4.4%) and ethosuximide (1.1%). Adjuncts included vitamin B complex, folic acid and pyritinol, while comorbid disorders/diseases were treated with trifluoperazine, thioridazine, chlorpromazine, haloperidol, amitriptyline, imipramine and diverse agents (antihelmintics, antimalarias, antihypertensives, antifungals and vitamin E). Side effects reported included headaches (20.4%), forgetfulness (20.4%), body weakness (18.2%), dizziness (13.6%), restlessness (13.6%), skin rashes (9.1%) and are traceable to the prescribed drugs for these patients. Side effects were managed by withdrawal of the drugs and replacing indicated drugs with those having less side effects and/or administration of drugs to remedy side effects. Overall, there was sound pharmacological basis for the drug treatment of these epileptic patients and the management of side effects although it was observed that the newer, more efficacious but more expensive AEDs were not used at all in these patients. Non-pharmacological management procedures employed were mainly counseling of patients by doctors and psychotherapy conducted by a psychologist.

The rate of attainment of remission increased with increase in years since first contact with orthodox facilities. Non-compliance to therapy and follow-up were the main contributing factors to failure of achieving remission in some of the patients. The type of seizures (generalized and partial) did not have a significant effect on attainment of remission ($p<0.05$). Longer duration (>1 year) between first seizure attack and medical intervention did not affect attainment of remission. Onset of epilepsy before the age of 10 resulted in lower remission rate. An average of ₦30, 986.67 per patient was spent per year on AEDs while the mean cost of total drugs was ₦33, 697.10 per patient per year. Cost increased with increase in clinic attendance, but decreased with increase in years since first contact with orthodox facilities. There was no significant difference in cost of total drugs or AEDs used as regard the type of seizure. The overall cost of treatment of epilepsy as seen here is considered high in relation to Nigeria's very low per capital income and especially as most of these patients (66.7%) were unemployed and were dependant on caregivers.

TABLE OF CONTENTS

LIST OF TABLES

TABLES:

LIST OF FIGURES

FIGURES:

ABBREVIATIONS

AEDs	Antiepileptic drugs
EEG	Electroencephalogram
CT	Computerized Tomography
MRI	Magnetic Resonance Imaging
NDMA	N-methyl-D-aspartate
AMPA	α-Amino-3-hydroxy-5-methyl-4-isoxaazole propionate
GABA	Gamma amino butyric acid

CHAPTER ONE

INTRODUCTION

In most parts of the World, great importance is not accorded to mental health and mental disorders as compared to the attention paid to physical heath (WHO, 2001). Mental health are largely neglected or ignored, thus, the world is suffering from an increasing burden of mental disorders and a widening ‘treatment gap’ (WHO, 2001). Today, 450 million people suffer from mental or behavioural disorder and only small minority of them receive the most basic treatment (WHO, 2005). In developing countries, most individuals with severe mental disorders are left to cope with their private burdens. Mental and behavioural disorders are estimated to account for 12% of the global burden of disease (WHO, 2001). Mental and behavioural disorders are understood as clinically significant conditions characterized by alterations in thinking, mood (emotions), concentration, memory, judgment or behaviour associated with personal distress and/or impaired functioning (Desia and Isaac, 2005). One incident of abnormal behaviour or a short period of abnormal mood does not, of itself, signify the presence of mental or behavioural disorder. In order to categorize it as disorder, such abnormalities must be sustained or reoccurring and they must result in some personal distress or impaired functioning in one or more areas of life (WHO, 2004). There are various types of mental and neurological disorders as well as psychosocial problems such as: psychotic disorders (e.g. schizophrenia), mood disorders (e.g. depressive disorders), neurotic, stress-related and adjustment disorders (e.g. anxiety disorder), neurological disorders (e.g. Alzheimer’s disease, epilepsy and mental retardation), childhood mental disorders (e.g. attention deficit hyperactivity disorder) and substance abuse-related disorders (e.g. alcoholism, drug abuse) (Desia and Isaac, 2005).

Epilepsy is a neurological disorder and is classified under chapter VI of International Classification of Diseases (ICD-10) with other diseases of the nervous system. However; epilepsy was historically seen as a mental disorder and many societies still consider it this way. The management of epilepsy is often the responsibility of mental health professionals because of the high prevalence of this disorder and the relative scarcity of neurologists especially in developing countries (WHO, 2001). It is characterized by recurrent episodes of self limiting abnormal behaviour which may or may not be associated with loss of consciousness and has a tendency to recur. They can occur several times a day to once every few months. In Africa, 80% of sufferers receive no treatment (ILAE/IBE/WHO, 2000; WHO, 2004).

Ancient physicians relied on clinical observation to distinguish between epileptic syndromes and infer their causes. Early pathphysiological theories of epilepsy correctly identified the brain as the site of the problem, but emphasis incorrect causes such as excess of phlegm in the brain. Treatments consist of prescribed diets or occasional surgery such as bloodletting or skull trephination and medical herbs. The unfortunate but widely held view of epilepsy as being due to occult or evil influence gained adherents even in the medical world during ancient times and the later acceptance of Christianity allowed theological interpretation of seizure as well. Magical or religions treatment were more frequently prescribed as a result, practices which persist to this day (Gross, 1992).

In Africa, epilepsy is traditionally looked upon as caused by ancestral spirit or attributed to possession by evil spirits (Reis, 1994). Sociocultural factors and particularly the supposed causes of epilepsy (evil spirits), can explain why patients first seek advice from traditional therapists and healers (Dale and Ben-Tovim, 1984; Danesi and Adetunji, 1994). It is only after several months or years of trails and error that the patient eventually is taken to hospital or rural health facility (Danesi and Adetunji, 1994).

Majority (80%) of people with epilepsy resides in rural area and do not receive modern treatment, or are not even identified (WHO, 2004). Poor infrastructures, insufficient availability of drugs and scarcity of trained medical personnel are all relevant factors for this situation. Some potential causes include the level of health care development, cultural beliefs, economy, distance from health care facilities, supply of anti epileptic drugs and lack of prioritization in national health policies (WHO, 2004).

Epilepsy places a significant burden on communities, especially in developing countries where it may remain largely untreated. Global Burden of Diseases (G.B.D) 2000 estimates the aggregate burden due to epilepsy to be 0.5% of the total disease burden. In addition to physical and mental disability, epilepsy often results in serious psychosocial consequences for individual and the family (ILAE/IBE/WHO, 2000).

The goal of treating patients with epilepsy is a cost-effective approach in terms of maximizing time taking by health care workers in treating such patients and reducing cost of treatment of epilepsy incurred by patients (both direct and indirect costs). The aim of cost effective approach is to eliminate seizures or reduced their numbers and frequencies while avoiding drug interactions and side effects, so as to achieve the best possible quality of life within the patient's financial limits (Garnett, 2000). Up to 70% of people newly diagnosed with epilepsy can be seizure-free if treated with antiepileptic drugs (WHO, 2005). Although in most countries the cost of treatment can be as low as $5 per patient in a year, the vast majority of sufferers remain untreated (WHO, 2005).

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 DEFINITION

Epilepsy can be defined as ‘the occurrence of transient paroxysms of excessive or uncontrolled discharges of neurons, which may be due to a number of different causes leading to seizures’ (WHO, 2004). Epilepsy may also be viewed as the presence of two or more seizures in the absence of an identifiable cause for the seizures (i.e. no intracranial or metabolic abnormality) (Pschirrer, 2004). The actual presentation or manifestation differs among individuals, depending on the location of the origin of the epileptic discharges in the brain and their spread (WHO, 2004).

A person should only be diagnosed as having “epilepsy” if there are recurrent manifestations i.e. there should be at least two or more unprovoked similar episodes at least 24 hours apart. Hence, the first episode of a seizure is called a “single seizure” and not epilepsy. Epilepsy can also be divided into active and inactive epilepsy. Epilepsy is considered “active” if the patient with epilepsy has at least one seizure unprovoked by any immediate identified cause in the preceding two years and is/or has been on antiepileptic drugs for the same, otherwise it is termed “inactive” (WHO, 2004).

Conditions mimicking seizures include pseudoseizures (also called psychogenic or hysterical seizures), syncope, some forms of stroke (e.g. transient ischaemic attacks), some sleep disorders (e.g. sleep apnoea), night terrors, emotional outbursts, breath-holding spells and hypnagogic (hypnic) jerks (WHO, 2004).

2.2 EPIDEMIOLOGY

Incidence may be defined as the number of new cases of a disease in a specific population over a defined period of time, while prevalence is the number of a disease in a defined population at a particular point in time (HTNF, 2002). Epilepsy represents the most common neurological complication affecting approximately 1% of the world's population (Nassiri *et al.*, 2000). Reported annual incidence rates for recurrent seizures vary between 30 and 50 per 100,000 with prevalence rates in most studies put at between 500 and 1000 per 100,000 (Keranen *et al.,* 1988). About 50 million people in the world will have recurrent seizures and this could be underestimated as partial seizures are often not recognized as such in developing countries (WHO, 2004).

In Africa, epilepsy affects 10 million people directly (WHO, 2004) with prevalence rates ranging from 2.2 to 58 per 1000. The lowest rate in Africa was recorded in South Africa (2.2%). Highest rate of over 15% are mainly detected in rural populations. They reflect poor health conditions resulting in several public health-related diseases complicated by epileptogenic changes in the brain (WHO, 2004).

In Nigeria, Africa's most populous country, the prevalence of epilepsy varies from approximately 0.53% to 3.7% of the population depending on the part of the country, its respective development and available health care facilities (www.epilepsyontario.org). The further developed and the better the health care facilities, the lower the prevalence rate (www.epilepsyontario.org). However, approximately 70% of the country's population resides in the rural areas where adequate health facilities are lacking. The age groups accounting for the highest prevalence rates for epilepsy are those in the first twenty years of life and children of about fifteen years of age or younger constitute almost half of Nigeria's total population (www.epilepsyontario.org). A door-to-door survey carried out in Igbo-Ora, Nigeria (with a population of approximately 20,000) shows a prevalence rate of 5.3 cases/1,000 of active epilepsy (Osuntokun *et al.*, 1987). There were 101 (48 males and 53 females) who suffered from active epilepsy with the highest age-specific prevalence ratios occurring in those below age 20. The prevalence ratio of epilepsy in this town which has an effective health care system is similar to that reported in some developed countries and several times lower than figures derived from studies in developing countries (Osuntokun *et al.*, 1987).

2.3 AETIOLOGY OF EPILEPSY

The Aetiology of epilepsy has both genetic and non-genetic components. Genetic causes contribute to a wide diversity of human epilepsies. Genetic causes are solely responsible for some rare forms inherited in a Mendelian pattern as, autosomal dominant or autosomal recessive. Genetic causes also are mainly responsible for some forms of epilepsy such as juvenile myoclonic epilepsy (JME) and childhood absence epilepsy (MC Namara, 2001). Other causes of epilepsy in developing countries include: brain damage (caused by birth complications, infections and parasitic diseases), brain injuries intoxication and tumours, parasitic infections, cysticercosis (e.g. tape worm, schistosomiasis, malaria), tubercular and viral encephalitis (Senanyake, 1993). However, in half of the cases the causes remain unknown (WHO, 2005). Three potential life-threatening conditions may cause chronic epilepsy: vascular malformations, tumors and neuroinfection (Janszky, 2004).

Epilepsy seen very early in life (<5years) are due more to organic disorders e.g. cerebral palsy, congenital abnormalities, birth complication or infections among others (WHO, 2004). Affected children tend to have low remission rate (Sirdharam, 2002). On the other hand, epilepsy in adolescents (>12years) are likely due to hormonal changes or to other factors such as infection, malformations and neurodegenerative disorders (WHO, 2004). They have higher rate of remission (Sirdharam, 2002). Ovarian steroid hormones alter excitability of neurons of the central nervous system (Martha and Morrell, 2002). Estrogen reduces inhibition at the gamma amino butyric acid (GABA-A) receptor, enhances excitation at the glutamate receptor and increases the number of excitatory neuronal synapses. Progesterone enhances GABA mediated inhibition, increases GABA synthesis and increases the number of GABA-A receptors (Martha and Morrell, 2002). In animal models of epilepsy, estrogen increases and progesterone decreases the likelihood that a seizure will occur (Martha and Morrell, 2002). Women with epilepsy may experience changes in seizures at puberty, during the menstrual cycle and at menopause (Herzog, 1991; Martha and Morrell, 2002). These seizure patterns are believed to be associated with changes in estrogen and progesterone levels (Morrell, 1998). Past studies indicated that androgen deficiency is usually common among men with epilepsy and it possibly exacerbates seizure frequency. However, improved seizure control has been achieved with adjunctive use of the aromatase inhibitor testolactone or antiestrogen clomiphene (Herzog, 1991). Observations of hormone-seizure relationships have led to interest in hormonal therapies (Martha and Morrell, 2002).

High dose progesterone (provided as natural progesterone suppositories or lozenges or as intramuscular medroxyprogesterone) has shown some success (Herzog, 1999).

The high incidence in developing countries are, to a significant degree, attributable to symptomatic epilepsies caused by a host of parasitic and infectious diseases that are largely absent in industrialized countries. Malaria may indirectly lead to epilepsy through febrile seizures just as malnutrition and under resourced health care at the pre- and postnatal levels (Robert *et al.,* 2001; WHO, 2004). For example in the study of infants with epilepsy in Nigeria, 48% of cases were identified as being caused by birth asphyxia, infections or hypoglycemia (Robert *et al.,* 2001). Febrile convulsions, head trauma, meningo-encephalitis, perinatal causes and cerebral palsy are the major causes of seizures in Nigeria (Ogunniyi *et al.,* 1987; WHO, 2004).

In some patients there are certain precipitating or triggering factors for epilepsy, for example loss of sleep, flickering lights and alcohol abuse (WHO, 2004). However, because epilepsy is a medical condition with recurrent unprovoked seizures, repeated seizures due to alcohol withdrawal are usually not considered as epilepsy (Cavazos *et al.,* 2004).

2.4 EPILEPTOGENESIS AND PATHOPHYSIOLOGY OF EPILEPSY

The pathology of epilepsy differs according to the classification into partial and generalized epilepsies.

For partial epilepsies reduction of inhibiting synaptic activity, or enhancement of excitatory synaptic activity might be expected to be a seizure trigger (Porter and

Meldrum, 1998). The neurotransmitters mediating the bulk of synaptic transmission in the mammalian brain are amino acids, gamma-amino butyric acid (GABA) and glutamate being the principal inhibitory and excitatory neurotransmitters respectively. Studies disclosed that micro injection of antagonists of the GABA receptor or of agonists of different glutamates–receptor subtypes, triggers seizures in experimental animals *in vivo*. (Namara, 2001; Porter and Meldrum, 1998). These glutamate-receptor sub-types are N-methyl-D-aspartate (NMDA), α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionate (AMPA) and kainic acid. An electrophysiological analysis of individual neurons during a partial seizure demonstrates that the neurons undergo depolarization and fire action potentials at high frequencies. This pattern of neural firing is characteristic of a seizure. This selective inhibition of this pattern of firing would be expected to reduce seizures with minimal unwanted effects.

Generalized epilepsies in contrast to partial seizures arise from reciprocal firing of nerves in the brain stem (Mc Namara, 2001). The striking synchrony in appearance of generalized seizure discharges in wide spread areas of neocortex led to the idea that a structure in the thalamus and/ or brain stem synchronized these seizure discharges (Mc Namara, 2001). The EEG hallmark of an absence seizure is generalized spike and wave discharges at a frequency of 3 per seconds. A comparison of EEG and intercellular recording reveals that the EEG spikes are associated with the firing of action potentials. One intrinsic property of thalamic neurons that is a pivotally involved in the generation of the 3 per second spike and wave is a particular form of voltage–regulated Ca^{2+} current, the low threshold ("T") current. Burst of action potential in thalamic neurons are mediated by activation of the T current. The T current plays an amplifying role in thalamic oscillations, one oscillation being the 3 per second spike and wave of the absence seizure. When an abnormal epileptic focus reaches seizure threshold, the discharge spreads to adjacent cells with resultant seizure activities. The seizures activities clinically manifest themselves in various ways which include altered personality and behaviour, tonic, clonic and resting phases (Porter and Meldrum1998; Mc Namara, 2001). Within the ictal phase, (convulsive phase) several physiological disturbances occur, which predominantly affect the respiratory system. The excessive muscular activities, which involve the whole system in a typical grand mal seizure, are associated with hypoxia, lactic acidemia and acidosis. These disturbances are short lived after the resting phase (Aguwa *et al.,* 1996).

2.5 DIAGNOSIS OF EPILEPSY

It is essential that, patients with episodes of epilepsy should be accompanied by a witness who can describe the episodes in detail. More often than not, epilepsy can be diagnosed on the basis of reports of patients and eyewitnesses (MOH, 1999). No laboratory test can replace a clear description provided by the eyewitness (WHO, 2004). When epilepsy is diagnosed, it is necessary to document the type of epilepsy, determine the cause and evaluate the intelligence and social background of the patient. This information is extremely useful for further patient management (MOH, 1999). Investigations of underlying causes include EEG and neuro imaging tests such as computerized tomography (CT) scan or brain magnetic resonance imaging (MRI) (WHO, 2004). If the patient has a normal neurological examination and is back to the usual baseline (e.g. cognitive, motor), the preferred study is a brain MRI because it has a much higher resolution to detect subtle abnormalities that cannot be detected by CT scan (Cavazos *et al.,* 2004).

EEG strengthens the diagnosis and provides some help in determining the prognosis of interictal epileptiform discharges or focal abnormalities. EEG is said to be specific to epilepsy in 30-50% of the cases. EEG should take place within 24 hours postictally. If the EEG shows no epileptiform potential, a sleep-EEG is required (Janszky, 2004). Although the criterion standard for diagnosis and the classification of epileptic seizures include the interpretation of a sleep-deprived EEG, the clinical history remains the cornerstone for the diagnosis of epileptic seizures (Cavazos *et al.*, 2004).

Video-EEG monitoring might be needed to establish a definitive diagnosis of seizures with impairment of consciousness. This test can rule out an epileptic aetiology with a high degree of confidence if the patient has a demonstrable impairment of consciousness during the seizure in question (Cavazos *et al.*, 2004). Video-EEG monitoring is also used to characterize the seizure types and epileptic syndrome for optimization of pharmacologic treatment as well as presurgical workup (Cavazos *et al.*, 2004). Lumbar puncture for cerebrospinal fluid (CSF) examination has a role in patients in whom meningitis or encephalitis is suspected (Cavazos *et al.*, 2004). Recent studies have shown that a high serum prolactin level within 100minutes of seizure is a suggestion that a generalized or complex partial seizure has occurred (Pritchard, 1991; Banerjee *et al.*, 2004)

2.6 CLASSIFICATION OF EPILEPTIC SEIZURES

Epilepsy may be classified based on type of seizure and epileptic syndrome. Both are summarized in Table below.

The three broad classes are partial, generalized and unclassified. Other details are as shown in Table 2.1 as described by ILAE (1989) and quoted by MOH (1999).

Table 2.1: **Classification of Epileptic Seizures by Type of Seizure**

Seizure Type	Features
1. Partial seizure	
A. Simple partial	Consciousness not impaired
i. With motor signs	Focal motor without march, focal with march, versive and postural phonatory
ii. With somatosensory or special symptoms	Visual, auditory, olfactory, gustatory and vertiginous
iii. With autonomic symptoms or sign	Epigastory aura, pallor, sweating, flushing, piloerection and papillary dilation
iv. With psychic symptoms	Dysphasic, dysmnesic, cognitive e.g. dreamy states distortion of time, affective (e.g. fear, anger), illusions and structured hallucination
B. Complex partial	Loss of consciousness
i. Simple partial	Simple partial followed by impairment of consciousness with/withouit automatism
ii. Complex partial	Simple partial followed by impairment of consciousness with automatism
C. Partial evolving to 2º generalized seizure	
i. Simple partial evolving to 2º generalized seizure	
ii. Complex partial evolving to 2º generalized seizure	
iii. Simple partial evolving to complex evolving to generalized seizure	
2. Generalized seizure	
A. Absence seizure	
i. Absence seizure	Impairment of consciousness only, mild clonic, atonic, tonic component and autonomic components
ii. Atypical Absence seizure	With change in tone that is more pronounced than in above (i) and with onset and/ or cessation that is not abrupt
B. Myoclonic	Sudden brief shock like muscle contraction, may be one limb or bilateral
C. Clonic	Clonic jerks, repetitive rhythmic flexing and stretching of limbs
D. Tonic	Sustained muscle contraction. May involved loss of consciousness
E. Tonic-clonic	Alternate contraction and relaxation of the muscles, loss of consciousness
F. Atonic	
3. Unclassified epileptic seizures	Usually used when an adequate description is not available.

Epileptic syndrome can be idiopathic (i.e. of unknown cause), symptomatic (believed to be the consequence of a known disorder of the central nervous system), or cryptogenic (i.e. thought to be symptomatic but with no known aetiology). Examples of cryptogenic or symptomatic epileptic syndrome includes infantile spasm (west syndrome), Lennox-Gastaut syndrome and epilepsy with myoclonic absence. Where a syndrome diagnosis can be made, it can provide useful information with regards to: onset, prognosis and choice of treatment (The Extra pharmacopoeia, 1993; MOH, 1999)

Table 2.2 shows a classification of epileptic seizure based on syndrome.
Ideally, physicians would classify the seizures of their patients using the classification for seizure types and if possible, make a syndromatic diagnosis (Cavazos *et al.*, 2004).

Table 2.2: **Classification of Epileptic Seizure by Epileptic Syndrome**

S/No	Class/ Syndrome	Sub-class	Examples
1	Localization- related (partial)	Idiopathic	Rolandic and Occipital epilepsies
2	Generalized	Symptomatic	Temporal lobe epilepsy (TLE), Benign occipital epilepsy
		Idiopathic (age–related)	Juvenile absence epilepsy, Juvenile myoclonic epilepsy, epilepsy with seizure precipitated specific modes of activation
		Idiopathic or Symptomatic	Epilepsy with myoclonic absence
		Symptomatic	Inherited metabolic or congenital disorder
3	Epilepsies undetermined whether focal or generalized		
4	Situational-related		Seizure occurring when there is an acute metabolic disturbance

2.7 FEATURES OF EPILEPSY

2.7.1 Partial Seizures

Partial seizures are divided into two groups, simple partial seizures where consciousness is maintained and complex partial seizures where there is an impairment of consciousness.

Simple partial seizures

In simple partial seizures, some patients may experience either motor or sensory phenomena. Such seizures arise from a specific area of the brain, with the patient being fully or partly aware of the event. In motor seizures, the focus is in the primary motor cortex. There are twitchings, starting in a distal part of the extremity, or in the face. The twitching may remain localized, or spread up the whole extremity and even become completely generalized to involve the whole body. Sensory seizures have their focus in the post-central gyrus (primary sensory cortex). There might be feelings of tingling, pins and needles, cold or heat, or numbness of the limb. Sometimes there may be strange feelings with visual signs, or hearing or smelling sensations. Autonomic seizures are associated with foci in the temporal lobe. There may be: a sensation rising from the epigastrium to the throat, palpitations, sweating or flushing. The psychic symptoms may consist of changes in mood, memory, or thought (thinking). There may be distorted perceptions (time, space, or person) or problems with language. Structured hallucinations could occur (music, scenes). These simple partial seizures are usually only recognized as epileptic seizures when they develop into generalized seizures (Porter and Meldrum, 1998; WHO, 2004).

Complex partial seizures

Here the patient has impaired consciousness, but not complete loss of consciousness. He is slightly aware of what is going on, but he cannot respond to anything, neither can he change his behaviour during an attack. The seizure usually starts with an aura which can be of many types such as; a strange feeling in the stomach rising up to the throat and head, or a sensation of light, smell, sound or taste or with changes in perception, e.g. of time (time seems to pass too slowly or too fast), of light sound or space. The surroundings may suddenly seem completely strange and different in scale (things seem larger or smaller than usual). These feelings can cause the patient a great deal of anxiety. Sometimes the seizure occurs with hallucinations or with psychomotor symptoms such as automatisms e.g. pulling at the clothes, chewing, lip smacking, or repeated aimless movements. These automatisms may become very complex, the patient is able to perform difficult tasks, or travel somewhere, but later not remember having done such a thing when he suddenly regains full consciousness, he may find himself in a completely different place. During such an automatism the patient may become aggressive and violent when restrained. There is slow recovery after a complex partial seizure, with a period of confusion. After the attack, there is complete amnesia regarding the attack. These seizures were previously called ‘psychomotor seizures’ and as the localization of the abnormal discharge is often in the temporal lobe, the epilepsy is often called ‘temporal lobe epilepsy’. However, the focus could also be in the frontal lobe (WHO, 2004).

During his lifetime, a patient does not necessarily have only one type of seizure. The type may change over the years, depending on the age and maturation of the brain.

Moreover, one patient may have a combination of different seizure types (The Extrapharmacopeia, 1993; WHO, 2004).

2.7.2 Generalized Seizures

Generalized seizures consist of many different seizure types, of which the primary generalized tonic-clonic seizure (GTCS) is the most common (WHO, 2004).

Tonic-clonic seizure

In a generalized tonic-clonic seizure, the patient loses consciousness, falls down, sometimes with a scream and develops a generalized stiffness (the tonic phase). Breathing stops, as all the muscles of the trunk are in spasm and the patient becomes cyanotic. The head is retracted, the arms flexed and the legs extended. After a while, this tonic phase is followed by the clonic phase, when the muscles alternately contract and relax, resulting in clonic movements. During this jerking, the patient might bite his tongue, urinate, or sometimes defeacate. The clonic phase may last several minutes. When all the jerking stops and the patient regain consciousness, he may feel very tired and confused and also have a headache. He has no memory of what happened. Often, he falls into a deep sleep. The frequency of the seizure may vary from once a day to once a month or once a year, or even once every few years. Generalized tonic -clonic seizures can also occur due to secondary generalization in partial epilepsies (WHO, 2004).

Clonic seizures

These seizures are generalized seizures, where the tonic component is not present, only repetitive clonic jerks (clonic jerks are repetitive rhythmic flexing and stretching of limbs). When the frequency of jerks diminishes, the amplitude of the jerks does not usually diminish (WHO, 2004).

Tonic seizures

Tonic seizures are sudden sustained muscle contractions, fixing the limbs in some strained position. There is immediate loss of consciousness. Often there is deviation of eyes and head towards one side, sometimes rotation of the whole body (WHO, 2004).

Absence seizures

These are short periods of loss of consciousness lasting only a few seconds (not more than half a minute) and occur mostly in children. They are of sudden onset, there are usually no, or only minimal motor manifestations. There is a blank stare, brief upward rotation of the eyes and an interruption of ongoing activity. The child is unresponsive when spoken to. It is suddenly over and the child continues what he/she was doing before the seizure. The child has no memory of these seizures. Absence seizures are easily provoked by hyperventilation (Porter and Meldrum, 1998; WHO, 2004).

Myoclonic seizures

These seizures consist of sudden, brief shock-like muscle contractions, either occurring in one limb, or more widespread and bilateral. They may be single jerks, or jerks repeated over longer periods (Porter and Meldrum, 1998).

Infantile spasms

Patients have flexor spasms of the head, bending of the knees and flexion with abduction of the arms. They occur in the first year of life and are very difficult to treat (Porter and Meldrum, 1998).

2.8 PHASES OF A SEIZURE

Four components of a seizure can be distinguished. Not all seizure types will have all these stages. The presence or absence and the nature of these stages are important for diagnosing the seizure type.

2.8.1 Prodormal Phase **-** This phase is not a seizure in itself. It can be seen in generalized seizure but it is more common in focal epilepsy particularly temporal lobe epilepsy. (Eric *et al.,* 2000). It begins a few hours or even days before the actual seizure and should not be confused with the aura. Prodromal symptoms include: headache, irritability, insomnia, bad temper, depression or increased activity (WHO, 2004).

2.8.2 Aura **–** Auras are focal seizures or the initial focal onset of a seizure and are associated with definable abnormal electrical discharges. Auras are abrupt in onset, last for seconds to minutes, may progress to a generalized seizure or may terminate as the seizure ends (Eric *et al.,* 2000). The type of clinical phenomenon depends on the site of origin and can include motor, sensory, autonomic, perceptual, cognitive and affective abnormalities (Eric *et al.,* 2000). The feelings of the aura are often vague and indescribable, leading to extreme fear, strange epigastric sensations, dreamlike experiences, unpleasant smells, etc. The patient remembers the aura very well, although he/she will not always be able to recount it, he/she can affirm the presence of it, as it happens before consciousness is lost (WHO, 2004).

2.8.3 Seizure (Ictus) - In most seizures there is a loss of consciousness and the patient is therefore not able to give any information about the actual ictus.

Therefore physicians usually depend on eye witnesses who had seen the actual seizure. The patient has no memory of the seizure.

2.8.4 Post-ictal Phase **-** This phase may be absent, brief or may last several hours and sometimes even days. There is usually a deep sleep and waking up with headache, tiredness, irritability, vomiting, confusion, muscular aches or ataxia. Transient paralysis of a part of the body, known as Todd's paresis may occur for a few hours or days. Altered speech or aphasia may occur when the dominant hemisphere of the brain has been involved. Altered behaviour and emotional outbursts may occur and if patients are restrained or interfered with, they may become violent (WHO, 2004).

2.9 SEX AND AGE VARIATION

Globally, in a population of 6 billion, 37 million (0.62%) were reported to be with epilepsy (Colin *et al.,* 2002). Incidence in Males (19.3 million) is higher than in Females (17.9 million) (Colin *et al.,* 2002). Roccg *et al.,* (2001) studies conducted in three Sicilian municipalities' showed that prevalence of active epilepsy (per 1,000 populations) was 3.3 over all, 3.5 for men and 3.2 for women. However, there was no difference in incidence between males and females in study which was carried out is South East England (Dominic *et al.,* 2002). According to Roccg *et al.,* (2001), age specific patterns for active epilepsy differed by sex, with higher figures for men at younger ages (5-19 years) and older ages (50-99 years). Majority of epileptic patients (70%) have their first attack before the age of 20 (Aguwa *et al.,* 1996). However, many epidemiological studies have shown that incidence and prevalence rates of epilepsy increase sharply after the age of 60 years (annual incidence rates being 100-

150 per 100,000) and the patients aged 60 years and above account for about one quarter of all incidence cases (Tapani, 2002).

2.10 MARITAL AND SOCIAL ECONOMIC STATUS

In many African ethnic groups, people with epilepsy cannot marry because of the stigma attached to the illness (WHO, 2004). Frequent seizure is known to have a determining effect on education, employment and marital life (Berhanu *et al.,* 2004).

The long-term study of prognosis of childhood onset epilepsy in Japan showed that the majority of these patients have lower levels of educational background as well as employment and marital status compared to the general population (Tsuji, 2004). From the study conducted by Wada *et al.,* (2004) in Japan, 13 men and 16 women epileptic patients experienced divorce. Epilepsy was the cause of divorce in 7 out of the 29, only one informed the spouse about the disease before marriage. However, in Linsten *et al.,* (2003), studies conducted on newly diagnosed unprovoked epileptic seizure in adult age, reveal that most leisure time activities, marital status and holding driver's license were not affected.

2.11 PSYCHIATRIC COMORBIDITY IN EPILEPSY

Several psychiatric disorders have been shown to be more common in persons with epilepsy compared to the general population (Gilliam *et al.,* 2003). Psychiatric comorbity can be intraictus (anxiety) or interictus (e.g. personality disorder). Depression appears to be the most common psychiatric comorbidity (Edeh *et al.,* 1987; Eric *et al.,* 2000). Some publications show that depression and anxiety are prevalent in epilepsy and many people with recurrent seizures may have one or both psychiatric disorders (Mendez *et al.*, 1988; Baker *et al.*, 1996; Hermann *et al.*, 2000). Bipolar disorder and cognitive disorders also occur at an increased rate in epilepsy compared with the general population (Sigita, 2003). There is evidence of an association between epilepsy and autism, as documented by presence of seizure in approximately 30% of children with autism (Sigita, 2003). Epilepsy is also frequently associated with other behavioural conditions, such as sleep disturbances (Sigita, 2003). Where they co-exist, the simultaneous treatment of the epileptic and the psychiatric disorder (depressions, psychosis and psychogenic non-epileptic attacks) is necessary for sufficient anticonvulsant management (Schmutz *et al.*, 2001). Personality disorder is one on the comorbid mental disorder that occur in epileptic patients the causes are multiple; social limitation imposed on the epileptic person during childhood and adolescence, self-consciousness and reaction to people (Gelder *et al.*, 1999). Patients with epilepsy resulting from a brain damage experiences personality disorder more than others (Gelder *et al.*, 1999).

2.12 DRUGS USED IN TREATMENT OF EPILEPSY

Choices of drugs in treating epilepsy depend on seizure type as shown in Table 2.3.

In simple and complex partial seizures: carbamazepine, phenytoin and valporate are used. The following new drugs are also effective: gabapentine, lamotrigine, levetiracetam, tiagabine, topiramate and zonisamide. In partial and generalized seizure, carbamazepine, phenobarbitone, phenytoin, valporate, primidone, gabapentine, lamotrigine, levetiracetam, tiagabine, topiramate and zonisamide are employed. Ethosuximide is the drug of choice for absence seizure while valporate is effective in ethosuximide resistant patients. Lamotrigine is one of the new drugs that are also employed. In myoclonic seizure, valporate is the drug of choice in myoclonic symdrome, however some patients also respond to clonazepam. In case of generalized tonic-clonic seizures, carbamazepine, phenobarbitone, phenytoin, valporate, primidone. Lamotrigine and topiramate are the new drugs that are also employed. In atonic seizure, it is often refractory to all available medication. Some reports suggest that valporate as well as lamotrigine may be useful. In infantile spasm, it treatment is limited to improvement of seizure control rather than feature of the disorder, such as retardation. Drugs used include; corticotrophin, prednisone and benzodiazepine (clonazepam and nitrazepam) (Porter and Meldrum, 1998; Mc Namara, 2001).

2.12.1 Antiseizure Barbiturate

Phenobarbitone

Phenobarbitone was the first effective organic antiseizure agent. It has relatively low-toxicity. It is inexpensive and is still one of the most effective and widely used drugs for this purpose (Mc Namara, 2001). Its mechanism of action involves potentiation of synaptic inhibition through an action on the GABA receptor. Phenobarbital increases the GABA receptors mediated current by increasing the duration of bursts of GABA receptor-mediated current without changing the frequency of burst. At levels exceeding therapeutic concentrations, phenobarbitone also limits sustained repetitive firings of brain neuron. This may underlie some of the antiseizure effects of higher concentration of phenobarbitone achieved during therapy of status epilepticus (Aguwa *et al.,* 1996).

Side effects produced are sedation, irritability and hyperactivity in children and agitation and confusion in the elderly (Mc Namara, 2001).

Deoxybarbiturates (Primidone)

Primidone is a congener of phenobarbitone in which the carbonyl oxygen of the urea moiety is replaced by two hydrogen atoms (Mc Namara, 2001). The antiseizure effects of primidone are attributed to both the drug and its active metabolites, principally phenobarbitone (Aguwa *et al.*, 1996).

The common side effects are: sedations, vertigo, dizziness, nausea, vomiting, ataxia, diplopia and nystagmus. Patients may experience an acute feeling of intoxication immediately following the administration of primidone (Mc Namara, 2001).

2.12.2 Iminostibenes (Carbamazepine)

Carbamazepine is related chemically to the tricyclic antidepressants. Its mechanism of action like that of phenytoin is via blockade of the sodium channels at therapeutic concentrations and inhibition of high-frequency repetitive firing in neurons in culture (Porter and Meldrum, 1998). It also acts presynaptically to decrease synaptic transmission (Porter and Meldrum, 1998). Carbamazepine is relatively a safe drug, though common adverse effects include: nausea vomiting, diplopia and ataxia. Hypersensitivity reactions also occur and these include the following: dermatitis, eosinophilia, lymphadenopathy and splenomegaly (Aguwa *et al.*, 1996). At higher doses, dizziness, drowsiness and hematological toxicity such as; aplastic anaemia, agronulocytosis (Aguwa *et al.*, 1996; Mc Namara, 2001) are seen. Acute intoxication with carbamazepine can result in a stupor or coma, hyperirritability, convulsions and respiratory depression (Mc Namara, 2001).

2.12.3 Succinimides (Ethosuximide)

Succinimides evolved from a systematic search for effective agents less toxic than the oxazolidinediones for the treatment of absence seizures. Ethosuximide is a typical example of drug that reduces low-threshold Ca^{2+}currents (T currents) in thalamic neurons. The T type calcium currents are thought to provide a pacemaker current, in thalamic neurons responsible for generating the rhythmic cortical discharge of an absence seizure attack. Inhibition of this current could therefore account for the specific therapeutic action of the drug (Porter and Meldrum, 1998). The most common dose-related adverse effects are gastric distress, including pain, nausea and vomiting. Other dose related adverse effects include transient lethargy or fatigue and much less commonly, headaches, dizziness, hiccup and euphoria (Porter and Meldrum, 1998).

2.12.4 Valporic acid (Sodium Valproate)

Valporic acid is effective against absence as well as partial and generalized tonic-clonic seizures. It inhibits sustained high frequency repetitive firing of neurons in culture at therapeutic concentration. The action is similar to that of phenytoin and carbamazepine and it appears to be mediated by a prolonged recovery of voltage activated Na^{+} channels from inactivation (Mc Namara, 2001). Another potential mechanism that may contribute to valproate's antiseizure actions involves metabolism of GABA. At high concentration, it increases the amount of the GABA by blocking the conversion of GABA to succinic semialdehyde (Porter and Meldrum, 1998). The more prominent neurological side effects include: tremor, somnolence and behavioral changes, memory, concentration, perception, decision making are not greatly affected.

Fine tremor that is dose dependent resolves on it own as therapy continues (Aguwa *et al.,* 1996).

2.12.5 Oxazolidinediones (Trimethadione)

Trimethadione and paramethadione are the two common examples of this group. Trimethadione is no longer the clinical agent of choice (Mc Namara, 2001). The oxazolidinediones increase the seizure threshold for seizure following repetitive thalamic stimulation. Both the drug and it active metabolite (dimethadione) has the same effect on thalamic Ca^{2+} currents as ethosuximide (reducing the T-type calcium current) (Porter and Meldrum, 1998). The most common undesired effects of trimethadione are: sedation and hemeralopia (blurring of vision in bright light). Less common but more serious untoward effects include: exfoliate dermatitis and other skin rashes, blood dyscrasias and hepatitis (Mc Namara, 2001).

2.12.6 Benzodiazepines (Diazepam)

At therapeutic concentrations diazepam and other active benzodiazepines augment the inhibitory effects produced by stimulating various GABAergic pathways and enhance GABA-induced changes in membrane potential. Molecular cloning and study of recombinant receptors have demonstrated that the benzodiazepine receptor is an integral part of the $GABA_A$ receptor. The acute toxicity of the benzodiazepines is low, relative to usual clinical dosage. Cardiovascular and respiratory depression may occur after the intravenous administration of diazepam, clonazepam or lorazepam, particularly if other antiseizure agents or central depressants have been administered previously (Mc Namara, 2001). The principal side effects of long-term oral therapy with clonazepam are drowsiness and lethargy. These occur in about 50% of patients initially but tend to subside with continued administration (Mc Namara, 2001).

2.12.7 Gabapentin

Gabapentin is one of the new antiseizure drugs. The chemical structure of gabapentin is a GABA molecule, covalently bond to a lipophitic cyclohexane ring. Gabapentin was designed to be a centrally active GABA agonist, with a high lipid solubility aimed at facilitating its transfer across the blood-brain barrier (Mc Namara, 2001). Gabapentin appears to act by promoting release of GABA by an unknown mechanism. It has a relatively poor efficacy (Wallace, 2001). The most common adverse effects of gabapentin are: somnolence, dizziness, ataxia and fatigue. These effects usually are mild to moderate in severity but resolve within two weeks of onset during continued treatment. Gabapentin is well tolerated (Mc Namara, 2001).

2.12.8 Lamotrigine

Lamotrigine is a phenyltriazine derivative. It has a wide spectrum of action, hence, should be prescribed with care (Wallace, 2001). It acts by blocking sustained repetitive firing of spinal cord neurons and delays the recovery from inactivation of recombinant Na^+ channels. These results are similar to those observed with phenytoin and carbamazepine (Mc Namara, 2001). Most common adverse effects seen in lamotrigine are: dizziness, ataxia, blurred vision or diplopia, nausea, vomiting and rash (hypersensitivity reaction) (Porter and Meldrum, 1998).

2.12.9 ϒ - Vinyl GABA (Vigabatrin)

ϒ-vinyl GABA (Vigabatrin) is arguably the first antiseizure drug developed on the basis of insights into synaptic mechanisms controlling seizure threshold. The goal was to pharmacologically enhance GABA-mediated synaptic inhibition through an irreversible inhibition of the degradable enzyme GABA transminase (Mc Namara, 2001).

Vigabatrin irreversibly inhibits GABA aminotransferase (GABA-T), the enzyme responsible for degradation of GABA. It therefore increases the amount of GABA at synaptic site, thereby enhancing inhibitory effects (Porter and Meldrum, 1998). The principal unwanted effects are psychiatric disorders, including depression and psychosis in a small number of patients (Mc Namara, 2001).

2.12.10 Felbamate

Felbamate is a dicarbamate. Clinically relevant concentrations of felbamate inhibit NMDA-evoked responses and potentiate GABA-evoked responses in whole-cell, voltage clamp recordings of cultured rat hippocampal neurons. This dual action on excitatory and inhibitory transmitter responses may contribute to the wide spectrum of action of the drug in seizure models (McNamara, 2001). An active control, randomized double-blind protocol, demonstrated the efficacy of felbamate in patients with poorly controlled partial and generalized seizures (Mc Namara, 2001).

2.12.11 Levetiracetam

Levetiracetam is a pyrrolidine, the racemically pure s-enatiomer of ∝–ethyl-2-oxo-pyrrolidineacetamide. A double blind, placebo-controlled trial of adults with refractory partial seizures demonstrated that addition of levetiracetam to other antiseizure medications was superior to placebo (Mc Namara, 2001). The mechanism by which it exerts antiseizure effects is unknown. A stereo selective binding site has been identified in rat brain membranes, but the molecular identity of this site remains obscure (Mc Namara, 2001). The drug is well tolerated, the most frequently reported adverse effects being somnolence, asthenia and dizziness (Mc Namara, 2001).

2.12.12 Tiagabine

Tiagabine is a derivative of nipecotic acid. It inhibits the GABA transporter, GAT – 1and thereby reduces GABA uptake into neurons and glia. In CA1 neurons of the hippocampus, tiagabine increases the duration of inhibitory synaptic currents, findings consistent with prolonging the effect of GABA at inhibitory synapses through reducing its re-uptake by GAT-1 (Mc Namara, 2001). Studies had established tiagabine’s efficacy as add-on therapy of refractory partial seizures with or without secondary generalization. Its efficacy in monotherapy for this indication has not yet been established (Mc Namara, 2001).

Toxicity/Side effects including Dizziness, somnolence and tremor, appear to be mild to moderate in severity and appear shortly after drug initiation (Mc Namara, 2001).

2.12.13 Topiramate

Topiramate is a sulfamate-substituted monosaccharide (Porter and Meldrum, 1998). It acts by reducing voltage-gated Na^+ current in cerebellar granule cells and may also act on the inactivated state of the channel in a manner similar to that of phenytoin (Porter and Meldrum, 1998). Topiramate also enhances post synaptic $GABA_A$-receptor currents and also limits activation of the AMPA-kainate-subtype(s) of glutamate receptor. Topiramate is also a weak carbonic anhydrase inhibitor (Mc Namara, 2001). The drug is well tolerated, the most common adverse effects are somnolences, fatigue, weight loss and nervousness (Mc Namara, 2001). However, Wallace (2001) showed that topiramate is widely used but poorly tolerated.

2.12.14 Zonisamide

Zonisamide is a sulfonamide derivative. It is effective against partial and generalized tonic-clonic seizures (Porter and Meldrum, 1998). It inhibits the T-type Ca^{2+} currents. In addition, it inhibits the sustained, repetitive firing of spinal neurons, presumably by prolonging the inactivated state of voltage-gated Na^{+} channels in a manner similar to actions of phenytoin and carbamazepine (Mc Namara, 2001). Zonisamide is well tolerated. However, some adverse effect seen are; ataxia, anorexia, nervousness and fatigue (Mc Namara, 2001).

2.12.15 Paraldehyde

It is a polymer of acetaldehyde, though it is best regarded as a polymer of cyclic structure. It had been used for the treatments of convulsions (including status epilepticus) in children (Charney *et al.,* 2001).

Sulphonylureas has been used in treatment of epilepsy.

Table 2.3: **Seizure Type and Drugs of Choice**

Seizure Type	Conventional Antiseizure Drugs	Recently Developed Antiseizure Drugs
Partial seizures		
Partial seizure (Simple and Complex)	Carbamazepine, phenytoin and valproate	Gabapentine, levetiracetam, tiagabine, lamotrigine, topiramate and zonisamide
Partial with secondarily generalized seizure	Carbamazepine, phenytoin, valproate, primidone, phenobarbitone (not in absence seizure)	Gabapentine, levetiracetam, tiagabine, lamotrigine, topiramate and zonisamide
Generalized seizures		
Absence seizure	Ethosuximide and valproate	Lamotrigine
Myoclonic seizure	Valproate and clonazepam	
Generalized tonic-clonic seizure	Carbamazepine, phenobarbitone, phenytoin, valproate and primidone	Lamotrigine and topiramate
Atonic seizure	Refractory to medication but valproate may be useful	Lamotrigine
Infantile spasm	Corticotrophin, prednisolone and benzodiazepines (nitrazepam and clonazepam)	

Seizure types and drugs of choice as described by Mc Namara, 2001.

Table 2.4 below shows the first and second line drugs for different types of seizures and drug to be avoided to prevent worsen of seizure in patients.

Table 2.4: **Drug options (First and Second line) by Seizure Type**

Seizure type	First-line drugs	Second-line drugs	Other drugs that may be considered	Drugs to be avoided (may worsen seizures)
Generalised tonic–clonic	Carbamazepine[a] Lamotrigine Sodium valproate Topiramate[a]	Clobazam Levetiracetam Oxcarbazepine[a]	Acetazolamide Clonazepam Phenobarbital[a] Phenytoin[a] Primidone[a,b]	Tiagabine Vigabatrin
Absence	Ethosuximide Lamotrigine Sodium valproate	Clobazam Clonazepam Topiramate[a]		Carbamazepine[a] Gabapentin Oxcarbazepine[a] Tiagabine Vigabatrin
Myoclonic	Sodium valproate Clobazam (Topiramate[a,])	Clonazepam Lamotrigine Levetiracetam Piracetam Topiramate[a]		Carbamazepine[a] Gabapentin Oxcarbazepine[a] Tiagabine Vigabatrin
Tonic	Lamotrigine Sodium valproate	Clobazam Clonazepam Levetiracetam Topiramate[a]	Acetazolamide Phenobarbitone[a] Phenytoin[a] Primidone[a,b]	Carbamazepine[a] Oxcarbazepine[a]
Atonic	Lamotrigine Sodium valproate	Clobazam Clonazepam Levetiracetam Topiramate[a]	Acetazolamide Phenobarbitone[a] Primidone[a,b]	Carbamazepine[a] Oxcarbazepine[a] Phenytoin[a]
Focal with/without secondary generalisation	Carbamazepine[a] Lamotrigine[b] Oxcarbazepine[a] Sodium valproate Topiramate[a]	Clobazam Gabapentin Levetiracetam Phenytoin[a] Tiagabine	Acetazolamide Clonazepam Phenobarbitone[a] Primidone[a,b]	

[a] Hepatic enzyme-inducing AED.

[b] Should rarely be initiated – if a barbiturate is required, Phenobarbitone is preferred.

[c] In children, for severe myoclonic epilepsy of infancy.

Source: www.rcgp.org.uk/nccpc/docs/appendix%

2.13 NON – PHARMACOLOGICAL THERAPIES

2.13.1 Ketogenic Diet

Ketogenic Diet was developed in the 1920s as a treatment for intractable childhood seizures at a time when few antiepileptic drugs were available (Kinsman *et al.*, 1992). It is an alternative therapy used to control intractable seizures. It involves consuming a calorie – restricted diet in which the fat, carbohydrate and protein ratio ranges from 2:1 to 5:1. Recent prospective studies in children demonstrate that about 5% of children will continue on the diet for at least a year with 40 – 50% of those starting the diet having a >50% reduction in seizures after 12 months (Vining, 1999). Its side effects, both cognitive and allergic, appear fewer than most available medications and it is also cheaper than most new anticonvulsants (Swink *et al.*, 1997).

Though the mechanisms are not well understood, ketogenic diet appears to rely on a fundamental change in the brain's metabolism, from that of a glucose-based energy substrate to a ketone-based substrate. This change in some fashion is critical to the maintenance of seizure threshold (Swink *et al.*, 1997). Understanding how ketogenic diet works (how the changing of a glucose substrate to a ketone body substrate possess anticonvulsant properly), then perhaps a medication could be developed that would be a major departure in the study of the neuroscience of epilepsy (Swnk *et al.*, 1997). There are no reports on the use of this method in Africa (WHO, 2004).

The Atknis diet with less restriction therapy that also creates ketosis and can lower the number of seizures (Kossoff, 2004) was recently used. Dietary therapy may become even more valuable in the therapy of epilepsy, when the mechanisms underlying their successes are better understood (Kossoff, 2004).

2.13.2 Surgery

Despite the new advancements in antiepileptic drug development, thousands of people with epilepsy will remain intractable to medication. For a considerable proportion of these people, epilepsy surgery is a consideration for better control of their seizures. In some places, surgery is now a standard practice for patients with medication refractory epilepsy (Tatum *et al.,* 2000). Surgical procedures related to the outcome of epilepsy surgery, surgical procedures were classified as “curative” (which included temporal and extratemporal resections, hemispherotomy and stereotactic radiosurgery) and as “palliative” (which mainly include callosotomy and multiple subpial transections) (Guenot, 2004). Results from past studies suggest that temporal lobe surgery is an efficient treatment of drug refractory temporal lobe epilepsy (Guenot, 2004).

As technology and drug availability increases in the new millennium, it is important for the primary care physicians to be aware of epilepsy surgery as a means of treating patients with antiepileptic drug refractory epilepsy (Tatum *et al.,* 2000).

2.13.3 Vagus Nerve Stimulation

Vagus nerve stimulation was approved in 1997 as a novel adjunctive therapy that has recently become commercially available for intractable epilepsy (Benbadis *et al.,* 2001). It has been indicated as add-on treatment for seizures of partial onset with or without secondary generalization in patients of about 12 years of age or older (Uthman, 2000). The vagus nerve stimulation system is composed of a battery generator that delivers regular intermittent electrical stimuli, programmed via menu driven software and an interrogating wand. The generator is implanted in the left upper chest and connected to the left cervical vagus nerve, via a pair of semi-circular helical electrodes wound around the vagus nerve and wires tunneled under the skin. Surgery is normally completed within 2 hours under general anesthesia and the patient can go home within a few hours post operatively (Uthman, 2000).

Two multicentre active-control parallel, double-blind protocols showed a statistically significant reduction in partial onset seizure with reasonable and well-tolerated side effect (Ramsay *et al.,* 1994). Adverse events related to vagus nerve stimulation include; voice alteration, throat pain and tingling sensation in the throat during stimulation. Coughing during stimulation occurred normally when therapy was initiated and shortness of breath occurred mainly during exertion. It is effective when compared to novel potent anti-epilepsy drugs, however it does not replace resection epilepsy surgery in selected patients in whom chances of seizure-free results are high (70 – 90%) (Uthman, 2000). Vagus stimulation has not been used in Africa (WHO, 2004).

2.13.4 Psychotherapy

Psychotherapy refers to planned and structured psychological interventions aimed at influencing behaviour, mood and emotional pattern of reaction to different stimuli through verbal and non-verbal psychological means. This includes: behavioural therapy (which consist of the application of scientific based psychological principle, to the solution of clinical problem), cognitive therapy (aimed at changing thought pattern and behaviour through the practice of new ways of thinking and acting), interpersonal therapy, relaxation techniques and supportive therapy (counseling based on doctor –patient relationship) (WHO, 2001). Psychotherapy cannot cure epilepsy, but it can help patients that are tensed up or emotionally upset. It can also help patients to understand their problem, to have a positive attitude and control stress as some parents of children with epilepsy are extremely worried and required psychotherapy themselves (WHO, 2004).

2.14 COST OF EPILEPSY TREATMENT

2.14.1 Definitions

Cost is defined as the value of the resources consumed by a programme or drug therapy of interest (Sanchez, 1999). The term "cost" refers to the economic value of resources consumed or not produced because of an illness (Begley *et al.*, 2002). Calculation of the cost of epilepsy involves estimating direct and indirect cost attributable to epilepsy (Hodgson *et al.*, 1982). Direct cost reflects resources consumed when healthcare and other social services are used to prevent, diagnose, treat or rehabilitate persons with epilepsy. Such cost may include: hospital inpatient and outpatient services, care by physicians and other practitioners, drugs, rehabilitation services and other non-medical items such as home, car, special equipment and so on. Indirect cost is defined as cost productivity due to morbidity and mortality including unemployment, under employment and decreased household work (Kotsopoulos *et al.,* 2001; Bagley *et al.,* 2002).

2.14.2 Types of Cost of Illness Study

Cost studies vary in terms of their time perspective and population considered. Prevalence-based cost studies estimated the cost over a short period (usually a year) for all prevalent cases, regardless of the evolutionary stage of the disorder. The short term of this study makes it most appropriate for estimating the cost of saving a new technology or programme that has short-term benefits for all patients. However, the study may underestimate cost because, newly diagnosed patients, who represent the highest fraction of the cost of epilepsy, are less frequently represented (Begley *et al.,* 2002).

Incidence-based studies, consider the cost of epilepsy for new cases from the time of diagnosis to a definite end point, such as cure or death. This study is more appropriate when evaluating the cost saving of interventions that prevent new cases, or prevent or reduce the future course of a disease. However, incidence-based study is more difficult than the prevalence-based study (Begley, *et al.*, 2002). A more thorough incidence study of estimate of the costs would require a few years of follow-up, because incidence studies by Cockerell, (1994) and De Zelacourt *et al.,* (2000), showed that the costs are largest during the first year after the onset of epilepsy and tends to decrease substantially in the second year.

There are two broad estimate approaches that are used in cost of illness research in epilepsy: bottom-up individual related and top- down service related.

Bottom-up individual related estimate includes: cost estimate of the number and type of health-care and social services consumed by individual patients and derived from observational studies of patients samples, or alternatively, based on hypothetical information provided by expert panels and related literature (Begley *et al.,* 2002). This approach is preferred when more precise estimates of individual costs are required and when the examination of the variation in cost across individual is desired (Begley *et al.,* 2002). One of the strengths of this approach is that it is well described, making it possible to explain differences in cost based on factors such as the seizure frequency, temporal stage of disease and healthcare setting (Forsgren *et al.,* 2005). One of the weaknesses of such a study is that it is often difficult to distinguish the epilepsy-specific costs from cost for co-morbidities (Forsgen *et al.,* 2005).

In the top-down type of study, estimates are based on surveys of funds received by providers of care and a portion is attributed to epilepsy. This approach is preferred for estimates of high prevalence illnesses that are well represented in national surveys (Begley *et al*., 2002).

2.14.3 Cost Items

Studies vary in terms of the cost items that are included. Most studies include the well-known epilepsy related items of healthcare consumption i.e. physician's visits, hospitalizations, diagnostic investigations, laboratory tests and treatments (antiepileptic drugs, surgical procedures, etc). Some studies also account for non-healthcare items such as transportation cost, residential care and unpaid care or services provided by patients' relatives (Begley *et al.*, 2002).

It has been recommended that studies should focus on the most comprehensive set of cost items. When it is necessary to rank the cost measures that are included, studies should focus on those items that are most likely to have a significant effect on cost (Begley *et al*., 2002).

2.15 AIM AND OBJECTIVES

The aim of this study was to evaluate the management of epilepsy at the ABUTH, Kaduna. The specific objectives included:

1. To ascertain the prevalence of epilepsy relative to other mental and neurological disorders.
2. To document the types and features of seizure disorders as presented by the patients including age, risk factors, comorbidities and effect on education.
3. To assess pharmacological management of epilepsy; rationale for drugs used, handling of drugs' side effects and non-pharmacological management.
4. To evaluate treatment outcome and some factors affecting attainment of at least one year remission.
5. To determine the cost of treatment of epilepsy in this part of the country.

Pharmacoeconomics studies have been conducted in different part of the world especially in United Kingdom (Heaney *et al.,* 2000) and the United States (Begley *et al.,* 2001). The economic burden due to epilepsy has not been adequately examined in developing countries. Cost estimates are very important in health care, planning and delivery of services (Thomas *et al.*, 2001). Included in this study is the cost of treatment of epilepsy which has never been carried out to the best of my knowledge in this part of the country (ABUTH, Kaduna).

Treatment outcome, based on remission of seizure attack in the patients, effect of epilepsy in patients' social life (education and marital) were also assessed. Epilepsy in Nigeria also has social problems just as in other part of the globe. Many patients lose their job, friends and even withdraw from social activities because of the fear of having a seizure in public (Danesi *et al.*, 1981).

ABUTH was chosen as the study center because it is a tertiary institution that enjoyed referrals from different part of the country, therefore assess to different ethnic group in Nigeria is possible. The preliminary study conducted shows proper documentation of their (ABUTH) medical reports which is necessary for this retrospective study. Past studies in Africa indicated that duration of epilepsy (time interval between first attack and medical intervention), type of seizure and age of onset do not influence seizure control (Cockerell *et al.*, 1995; Berhanu and Prevett, 2004). Is this true of Nigerian epileptics? This study was aimed at verifying the effect of the above named factors in the attainment of at least one year remission in Nigerian epileptics attending ABUTH, Kaduna.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 FEATURE AND MANAGEMENT

A retrospective approach using selected patients' folders and medical records from the Ahmadu Bello University Teaching Hospital (ABUTH) Psychiatry Department was employed for data collection. The data collection was in three stages.

STAGE ONE

The hospital unit numbers of patients in ABUTH who attended clinics between January 2003 and December 2004 were collected using the out patient clinic department register which usually held twice a week, Mondays and Thursdays. This accounted for the total number of mental and neurological disorder patients who attended clinics within this period under study. The data was analyzed using Statistical Package for the Social Scientist (SPSS version 11.0) and the frequency of attendance was determined. Case notes of patients who attended clinic at least six times during this period (January 2003 to December 2004) were sorted out for the second stage of data collection. This sampling criterion was based on the fact that stable patients were usually given a minimum of sixteen weeks interval between appointments (personal communication from clinician).

STAGE TWO

This stage of data collection involved collection of patients' social demographical data, categories of diagnosis and specific diagnosis, using the patient's folders. This data was also analyzed using SPSS and data was sorted out based on specific diagnosis. The frequency and percentage of each specific diagnosis were determined.

From this stage; patients diagnosed for epilepsy were filtered out for the third stage of data collection. Out of the 92 patients diagnosed of epilepsy, one patient's folder was not available for the third stage of data collection.

STAGE THREE

A researcher's-administered structured data forms (Appendix) was used for the rest of the information needed for this retrospective study. Data collected were analyzed using SPSS. Frequencies of the socio demographical data (residence, sex, age at first contact with orthodox facilities, age of onset of epilepsy, years since contact with orthodox facilities, number of times accompanied, occupation, etc) were determined. Others included: risk factors (and age at which it occurred), alternative therapy ever used, duration between first seizure attack and medical intervention, signs and symptoms, diagnosis and laboratory investigation. The following data were also collected, family history of mental or neurological disorder, drugs used, side effects of drug(s) used, overall clinic attendance, clinic attendance in relation to residence and general treatment outcome.

Epileptic patients, who had attended clinics for more than one year, were used to determine patients who were free of seizure for at least one year. Within this group, those that attained one year remission were compared to those who had not (as of their last clinic visit). Clinic attendances, adherence to therapy, age of onset of epilepsy, duration between first attack and medical intervention and seizure type were factors taken into consideration. Patients who had missed clinic appointment at least for 4 months were regarded as clinic defaulters. Those who had at least one record of poor adherence between 2003 and 2004 were said to have poor adherence to therapy.

Chi square test was used to compare the various patient groups and statistical significance at 95% confidence interval was calculated.

3.2 COST OF ILLNESS

Prevalence studies using bottom-up method was chosen because it is typically based on the recruitment of patients with "active" epilepsy (i.e. epilepsy with recurring seizures and/or under current treatment). In this study, patients (within the epilepsy patients' population) who had attended clinics regularly between the period of November 2003 and October 2004 (1 year follow-up) and did not default any of their clinic attendance were used. The cost item evaluated in this study is basically the cost of drugs used by patients during this period.

CHAPTER FOUR

4.0 RESULTS

4.1 CLINIC AND PATIENT ATTENDANCE

The result of the first stage of data collection is shown in Table 4.1. In 2003, the number of clinics held was 100 and total number of attendance was 6,460. In 2004, the number of clinics held was 97 and total number of attendance was 6,959. The average numbers of patient attendance per clinic are 64.6 and 71.7 in 2003 and 2004 respectively. Of the total number of attendances in 2003 and 2004 (being 13,419) 875 patients attended clinics at least six times.

Table 4.1: **Clinics and Patient Attendance in 2003 and 2004**

Month	Number of Clinics Held in 2003	2003 Total Attendance	Number of Clinics Held in 2004	2004 Total Attendance
January	9	495	8	561
February	8	450	8	567
March	9	508	9	702
April	7	385	8	666
May	8	457	9	667
June	9	507	8	543
July	9	568	9	679
August	8	551	9	635
September	9	710	9	768
October	9	682	6	410
November	7	579	8	552
December	8	568	6	209
Total	100	6460	97	6959

4.2 DIAGNOSIS OF MENTAL AND NEUROLOGICAL DISORDERS

Of the 875 patients, 60 patients' folders (6.9%) could not be located at the time of data collection while no specific diagnosis was recorded for 54 others (6.2%). The five most common mental and neurological disorders were schizophrenia (46.6%), depression (13.7%), seizure disorder/epilepsy (12.1%), anxiety disorders (8.0%) and bipolar affective disorder (7.5%). Details are shown in Table 4.2.

Table 4.2: **Categories of Diagnosis in Total Patient Population**

Categories of Diagnosis	Number of patients	% Total Mental cases categorized
Schizophrenia	355	46.6
Depression	104	13.7
Seizure Disorders/epilepsy	92	12.1
Anxiety Disorder	61	8.0
Bipolar Affective Disorder	57	7.5
Mania	27	3.5
Organic brain Disorders	26	3.4
Substance Abused Disorders	16	2.1
Somatization	16	2.0
Dementia	2	0.3
Personality Disorder	1	0.1
Mental Retardation	1	0.1
Autistic Disorder	1	0.1
Developmental Disorder	1	0.1
Insomnia	1	0.1
Total	761	100.0

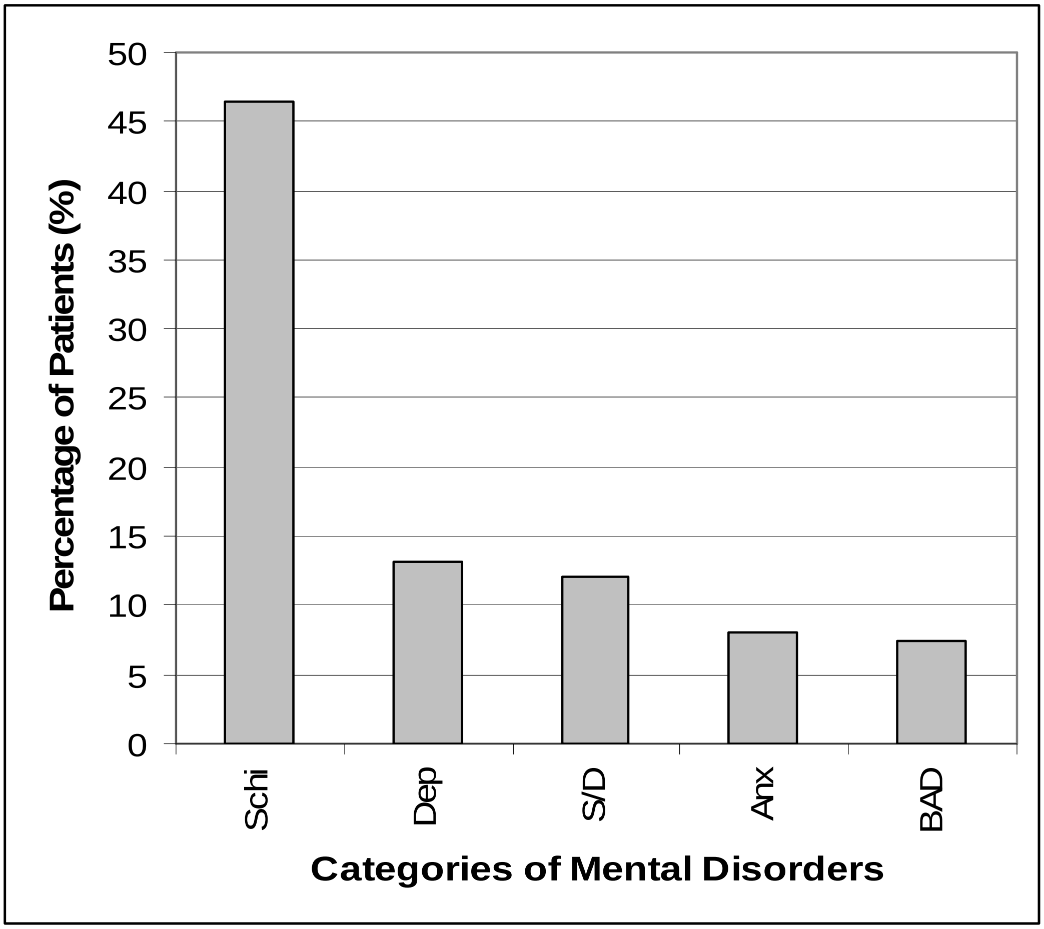

Fig 4.1: Most Common Mental Disorders

KEY:

Schi	Schizophrenia
Dep	Depression
S/D	Seizure Disorder/Epilepsy
Anx	Anxiety
BAD	Bipolar Affective Disorder

4.3 SEX DISTRIBUTION

Of the 92 diagnosed epileptics, one folder was empty (devoid of notes) and could not be used further.

Table 4.3 illustrates sex distribution of epileptic patients seen at ABUTH. There were more epileptic male (52.7%) than female (47.3%).

Table 4.3: **Sex Distribution of Epileptic Patients as seen at ABUTH**

Sex	Number of patients	Percent
Male	48	52.7
Female	43	47.3
Total	91	100.0

4.4 AGE DISTRIBUTION

Age distribution of epileptic patients is illustrated on fig 4.2 and fig 4.3. Half of the patients were less than 20 years of age (50.6%) at first contact with orthodox facilities (fig 4.2).

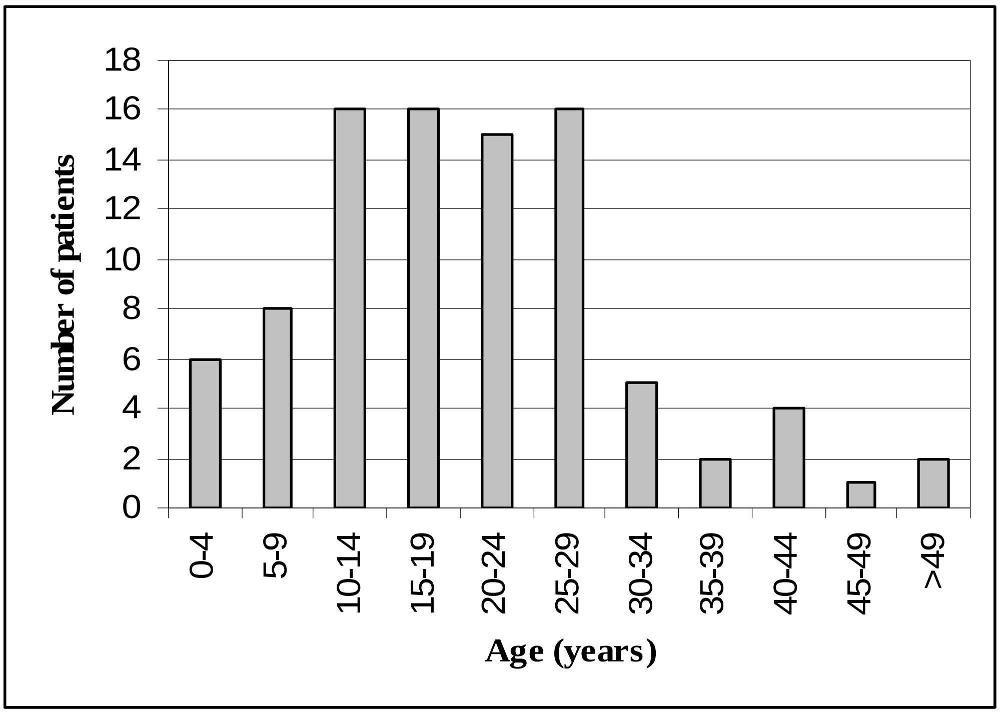

Fig 4.2: Age at First Contact with Orthodox Facilities of the Epileptic Patients

Thirty-six patients had no record of age of onset of epilepsy. Majority (74.6%) of the epileptic patients were less than 20 years of age at the time of onset of epileptic disorder (fig 4.3).

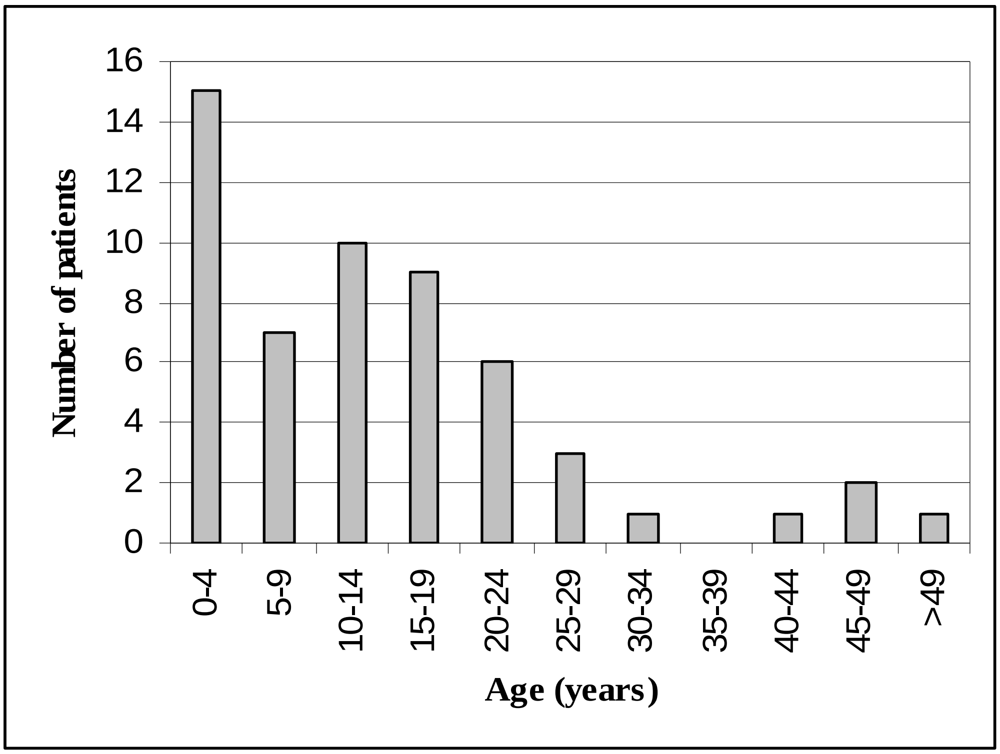

Fig 4.3: Age of Onset of Epilepsy in the Epileptic Patients

4.5 OTHER SOCIO-DEMOGRAPHICAL FEATURES

Years since contact with orthodox facilities means years since patient registered in the psychiatric clinic of ABUTH, Kaduna. Fifty one (56%) of the patients were less than 2 years since contact with orthodox facilities, twenty (22%) each were 2-4 and >4years since contact with orthodox facilities. Years since contact with orthodox facilities of the patients in general ranged from 0.41- 29.83years, with mean of 3.41 years.

The table below (Table 4.4) illustrates frequency distribution of tribal origin of epileptic patients. 49.45% of the Patients were Hausa/Fulani.

Table 4.4: **Frequency Distribution of Tribal Origin of Epileptic Patients seen at ABUTH**

Tribe	Number of Patients	Percent	Cumulative Percent
Hausa/Fulani	45	49.5	49.5
Yoruba	14	15.4	64.9
Ibo	6	6.6	71.5
Others	26	28.6	100.0
Total	91	100.0	

Geopolitical Zone of Epileptic Patients

Patients' geopolitical zones were derived from state of origin (not shown). Forty two of the 66 recorded (63.6%) were from the North West. Fifteen (22.7%) from North Central, 4 (6.1%) from North East, 3 (4.5%) from South East. The least percentage (3.0%) was recorded for South West.

Residence and Religion of Epileptic Patients

Seventy three (80.2%) of the patients reside in Kaduna metropolis, while 18 (19.8%) reside outside Kaduna metropolis. Four patients had no record against religion. Thirty three (37.9%) were Christians while 54 (62.1%0 were Muslims.

4.6 MARITAL STATUS, EDUCATION AND OCCUPATION

Sixty four (71.9%), 23 (25.8%), 2 (2.2%) of the patients are single, married and divorcee respectively, while the marital status of 2 patients were not record. Details are shown in Table 4.5.

Table 4.5: **Marital Status of the Epileptic Patients as seen at ABUTH**

Marital status	Number of Patients	Percent	Cumulative Percent
Single	64	71.9	71.9
Married	23	25.8	97.7
Divorced	2	2.2	99.9
Total	89	100.0	

Fifty-two of the 91 patients (57.1%) had no record for education. Details of level of education are shown in Table 4.6.

Table 4.6: **Level of Education of the Epileptic Patients as seen at ABUTH**

Level of Education	Number of Patients	Percent	Cumulative Percent
Never been to school	5	12.8	12.8
Koranic school	3	7.7	20.5
Nursery school	4	10.2	30.7
Primary school uncompleted	8	20.5	51.2
Primary school completed	6	15.4	66.6
JSS completed	6	15.4	825.0
SSCE	3	7.7	89.7
Graduate	4	10.2	99.9
Total	39	99.9	

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Table 4.7 illustrates occupation of epileptic patients. Six patients had no occupation record. More than two-third (81.1%) the epileptic patients were unemployed. They were; children (42.3%), students (21.2%), housewives (12.9%) and applicants (4.7%).

Table 4.7: **Occupation of Epileptic Patients as seen at ABUTH**

Occupation	Number of Patients	Percent	Cumulative Percent
Child	36	42.3	42.3
Student	18	21.2	63.5
Housewife	11	12.9	76.4
Privately Employed	10	11.8	88.2
Applicants	4	4.7	92.9
Civil Servant	4	4.7	97.6
Force	1	1.2	98.8
Prisoner	1	1.2	100.0
Total	85	100.0	

4.7 RISK FACTORS FOR EPILEPSY

Risk factors are factors that predispose an individual to epilepsy. Risk factor was not indicated for 53 of the 91 patients. Of those indicated, 9 (23.7%) had family history of epilepsy, while febrile convulsion occurred in 20 (52.6%) patients. Available details in Table 4.8.

Table 4.8: **Risk Factors for Seizure Disorder as seen at ABUTH**

Risk factors	Number of Patients	Percent	Cumulative Percent
Febrile convulsion	20	52.6	52.6
Family history	9	23.7	76.3
Road Traffic Accident	3	7.9	84.2
Head Injury	2	5.3	89.5
Infection (Measles, encephalitis)	1	2.6	92.1
Neonatal Jaundice	1	2.6	94.7
Parturition	2	5.3	100.0
Total	38	100.0	

Table 4.9 illustrates age at which risk factors occurred in epileptic patients. Twenty patients experienced febrile convulsion prior to the development of epilepsy, however, 10 of them had febrile convulsion between 7 months to 6 years of their life, while jaundice and infection occurred in two patients (one each) between the ages of 1-3 years.

Table 4.9: **Cross Tabulation of Risk Factors and Age at which They Occurred**

Risk factors	Age at which Risk Factors Occurred					Total
	7-11 months	1-3 years	20-24 years	30-39 years	Not recorded	
Febrile convulsion	3	7	-	-	5	15
Road Traffic Accident	-	-	1	1	1	3
Head Injury	-	-	-	-	2	2
Parturition	-	-	1	-	1	2
Infection (Measles, encephalitis)		1	-	-	-	1
Neonatal Jaundice	-	1	-	-	-	1
Total	3	9	2	1	9	24

Family history was not recorded for 37 out of the 91 patients. Out of 54 patients who had records of family history, 39 patients had no history of mental and neurological disorders. The most common family history of mental or neurological disorder is epilepsy. Available details in Table 4.10.

Table 4.10: **Family History Showing Specific Mental and Neurological Disorder of Epileptic Patients as seen at ABUTH**

Family history of mental/neurological disorders	Number of Patients	Percent	Cumulative Percent
No family history	39	72.2	72.2
Epilepsy	9	16.7	88.9
Psychosis	2	3.7	92.6
Febrile convulsion	2	3.7	96.3
Mental illness not specified	2	3.7	100.0
Total	54	100.0	

4.8 DIAGNOSIS OF EPILEPSY

Diagnosis of epilepsy by sex is illustrated in Table 4.11. More than half of the patients (58.2%) were diagnosed as having generalized tonic-clonic seizure disorder.

Table 4.11: **Specific Diagnosis of Epilepsy by Sex as seen at ABUTH**

Class of seizure	Specific Diagnosis	Male		Female		Total	
		Number of patients	%	Number of patients	%	Number of patients	%
Generalized	Tonic–clonic	27	29.7	26	28.6	53	58.3
Partial	Complex partial	10	11.0	10	11.0	20	22.0
Unclassified	Seizure disorder	5	5.5	5	5.5	10	11.0
Mixed seizure	Mixed seizure	2	2.2	0	0.0	2	2.2
Partial	Simple partial	1	1.1	1	1.1	2	2.2
Unclassified	Post traumatic seizure	1	1.1	0	0.0	1	1.1
Status epilepticus	Status epilepticus	1	1.1	0	0.0	1	1.1
Generalized	Myoclonic seizure	0	0.0	1	1.1	1	1.1
Partial	Partial motor seizure	1	1.1	0	0.0	1	1.1
Total		48	52.8	43	47.3	91	100.1

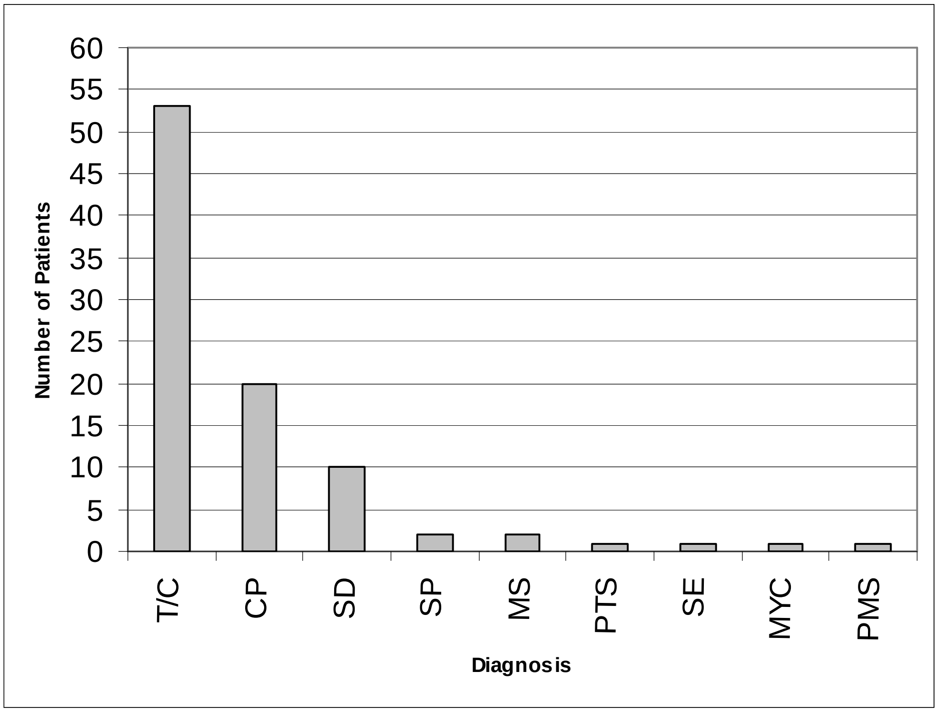

Fig 4.4: Specific Diagnosis of Type of Seizure

Key:

T/C	Tonic-clonic seizure
SD	Seizure disorder (Unclassified)
CP	Complex partial seizure
SP	Simple partial
MS	Mixed seizure
PTS	Post-traumatic seizure
SE	Status epilepticus
MYC	Myoclonic seizure
PMS	Partial motor seizure

Specific diagnosis by age is illustrated in Table 4.12. Most of those diagnosed of seizures disorder (unclassified) were teenagers, 8 patients (40.0%) diagnosed of complex partial were in their twenties. Most of those diagnosed of tonic-clonic seizures (Grand mal) were teenagers and those in their twenties.

Table 4.12: **Specific Diagnosis of Epilepsy by Age as seen at ABUTH**

Diagnosis	Age at first contact with orthodox facilities (years)											Total
	0-4	5-9	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	>49	
Tonic-clonic	4	4	11	10	12	8	1		2		1	53
Complex partial	1	1	1	3	2	6	4	1	1			20
Seizure Disorder (unclassified)		3	4	1						1	1	10
Mixed seizure				2								2
Simple partial	1								1			2
Myoclonic seizure						1						1
Post-traumatic					1							1
Status epilepticus								1				1
Partial motor						1						1
Total	6	8	16	16	15	16	5	2	4	1	2	91

Family's history of mental disorders was not documented for 37 patients. Available details in Table 4.13. Out of 54 patients whose family's history of mental/neurological disorder was documented, fifteen patients had record of family mental/neurological disorder. Seven out of the 15 patients with history of mental/neurological disorder were diagnosed of generalized tonic- clonic seizures, family history of mental/neurological disorder was not recorded for patients diagnosed of simple partial, post traumatic seizure and partial motor seizure.

Table 4.13: **Cross tabulation of Specific Diagnosis and Family History of Mental Disorder of Epileptic Patients as seen at ABUTH**

Specific Diagnosis	Family History of mental/neurological disorder			Total
	Yes	No	Not recorded	
Generalized Tonic- clonic	7	28	18	53
Complex partial	5	5	10	20
Seizure disorder	-	5	5	10
Mixed seizure	1	1	-	2
Simple partial seizure	-	-	2	2
Myoclonic seizure	1	-	-	1
Post traumatic seizure	-	-	1	1
Status epilepticus	1	-	-	1
Partial motor seizure	-	-	1	1
Total	15	39	37	91

Out of the 91 patients used for this study, 55 patients had no record of effect of epilepsy on their education. Of those that had records, 69.5% of them were either withdrawn from school or slow in learning or can not even go to school because of epilepsy. Details are shown in Table 4.14.

Table 4.14: **Effect of Epilepsy on Education as seen at ABUTH**

Effect of Epilepsy on Education	Number of Patients	Percent	Cumulative Percent
Dropped out because of embarrassment	1	2.8	2.8
Cannot go to school	4	11.1	13.9
Slow learning	3	8.3	22.2
Withdrawn from nursery school	2	5.6	27.8
Withdrawn from primary school	6	16.7	44.5
Withdrawn from secondary school	7	19.4	63.9
Withdrawn from tertiary	2	5.6	69.5
Doing well in school	11	30.6	100.1
Total	36	100.1	

Illustrated on Table 4.15 is effect of epilepsy on marital life of the patients. Eighteen years was considered as marriageable age. Thirty nine patients (42.9%) were below marriageable age. Two patients were divorced as a result of epilepsy, 25 (27.5%) were above the marriageable age at onset of epilepsy. However, one of these 25 patients is unable to marry because of epilepsy.

Table 4.15: **Effect of Epilepsy on Marital life as seen at ABUTH**

Effect on marital life	Marital status			Total
	Single	Married	Divorced	
Below marriageable age	39	-	-	39
Remained single	25	-	-	25
Not affected	-	16	-	16
Not recorded	1	8	-	9
Divorced	-	-	2	2
Total	65	24	2	91

The record of 49 patients did not contain information on the duration between first attacks of seizure and medical intervention. Available details in Table 4.16. Nine patients (21.5%) sought for medical intervention in less than a year of their first seizure attack and 11 patients (26.2%) did after 4 or more years of first seizure attack.

Table 4.16: **Duration between First Seizure Attack and Medical Intervention as seen at ABUTH**

Duration	Number of Patients	Percent	Cumulative Percent
< 1year	9	21.4	21.4
1-2 years	12	28.6	50.0
3-4 years	10	23.8	73.9
> 4 years	11	26.2	100.0
Total	42	100.0	

Frequency of seizure attack before seeking medical treatment was recorded for only 26 patients, while others (65 patients) were not recorded. Details are in Table 4.17.

Table 4.17: **Frequency of Seizure Attack before First Medical Intervention as seen at ABUTH**

Frequency of Seizure	Number of Patients	Percent	Cumulative Percent
Only Once	1	3.8	3.8
Only twice	2	7.7	11.5
Once a month	3	11.5	23.0
2-3 times per month	2	7.7	30.7
4-7 times per month	1	3.8	34.5
8-12 times per month	4	15.4	49.9
60-90 times per month	10	38.5	88.4
Recurrent not specific	3	11.5	99.9
Total	26	100.0	

The period of seizure attack was not recorded for 60 patients (65.9%) of the 91 epileptic patients. Of those recorded, seizures usually occurred at night in 21 or 67.7% of the patients. Available details in Table 4.18.

Table 4.18: **Period/Time Seizures Usually Occurred in Epileptic Patients as seen at ABUTH**

Period/Time	Number of Patients	Percent	Cumulative Percent
Night	21	67.7	67.7
During sleep	1	3.2	70.9
Day or Night	9	29.0	99.9
Total	31	99.9	

Sign and symptoms presented by epileptic patients are illustrated in Table 4.19. More than two-third of the patients (67.0%), presented with tonic-clonic muscle contraction and relaxation and loss of consciousness during attacks.

Table 4.19: **Signs and Symptoms Presented by Epileptic Patients as seen at ABUTH**

Major Sign and Symptoms	Number of patients	% of patients
Tonic-clonic (contraction and relaxation of muscles)	70	76.9
Loss of consciousness	61	67.0
Psychic symptoms	28	30.8
Foaming in the mouth	18	19.8
Urinary incontinence	16	17.6
Abnormal/bizarre behaviour	13	14.3
Hallucination	11	12.1
Abnormal body movement	11	12.1
Headaches	11	12.1
Jerking movement of upper limbs	8	8.8
Lip smacking/biting	8	8.8
Dizziness	7	7.7
Memory impairment	7	7.7
Poor concentration	6	6.6
Insomnia	6	6.6
Fecal incontinence	4	4.4

Table 4.20 illustrates laboratory investigations carried out on epileptic patients. The most common laboratory investigations carried out on these patients included serum urea & electrolyte, full blood count (FBC) & differential, stool microscopy, general haematological investigations, culture and sensitivity of urine.

Table 4.20: **Laboratory Investigations carried out on Epileptic patients at ABUTH**

Investigation	Numbers of patients	% of Patients
Serum Urea & Electrolyte	34	37.4
Full Blood Count and Differential	34	37.4
General Haematological Investigation	27	29.7
Stool Microscopy	26	28.6
Culture & Sensitive of urine	14	15.4
Urine Microscopy	12	13.2
Serum Glucose	12	13.2
Malaria Parasite	8	8.8
Electroencephalography (EEG)	7	7.7
Urinalysis	7	7.7
Skull X-ray	6	6.6
Fundoscopy	3	3.3
Liver function Test	2	2.2
Computerized Tomography (CT) Scan	2	2.2

Comorbid mental or neurological disorder was not recorded for 4 patients (2 patients for both sexes), 67 patients (77.0%) have no comorbid mental disorder (38 males and 29 females). Twenty patients (23.0%) presented with comorbid mental or neurological disorders (8 males and 12 females). Available details on Table 4.21.

Table 4.21: **Cross tabulation of Mental or Neurological Comorbid disorders and Sex of Epileptic Patients as seen at ABUTH**

Mental or Neurological comorbid disorders	Sex		Total
	Male	Female	
Anxiety	1	3	4
Psychosis	2	2	4
Abnormal Behaviour	1	1	2
Mental retardation	1	1	2
Dissociative disorder	-	1	1
Cerebral palsy	-	1	1
Neurological abnormality	1	-	1
Multiple somatic complain	-	1	1
Depression	-	1	1
Amnesia	1	-	1
Affective disorder	1	-	1
Total	8	12	20

4.9 DRUG TREATMENT OF EPILEPSY

Table 4.22 illustrates antiepileptic drugs prescribed for epileptic patients between 2003 and 2004. Carbamazepine was the most commonly prescribed drugs (91.2%) for the epileptic patients in ABUTH under the period of study.

Table 4.22: **Anti Epileptic Drugs Prescribed for Epileptic Patients in ABUTH, Kaduna between 2003 and 2004**

Drug prescribed	Pharmacological basis for Their Use	Class/Subclass	Number of patients using drug	% of patients using drug
Carbamazepine	Antiepileptic	Iminostibenes	83	91.2
Diazepam	Antiepileptic	Benzodiazepines	31	34.1
Phenobarbitone	Antiepileptic	Barbiturate	18	19.8
Clonazepam	Antiepileptic	Benzodiazepines	14	15.4
Phenytoin	Antiepileptic	Hydantoin	7	7.7
Primidone	Antiepileptic	Deoxy Barbiturate	4	4.4
Sodium Valproate	Antiepileptic	Valporic acid	4	4.4
Ethusuximide	Antiepileptic	Succinimides	1	1.1
Nitrazepam	Antiepileptic	Benzodiazepine	1	1.1

Illustrated in Table 4.23 are other drugs prescribed for epileptic patients between 2003 and 2004. Vitamin B complex was frequently prescribed as a nutritional supplements and its effect on the nerves.

Table 4.23: **Other Drugs Prescribed for Epileptic Patients in ABUTH, Kaduna between 2003 and 2004**

Drug prescribed	Pharmacological basis for Their Use	Class/Subclass	Number of patients using drug	% of patients using drug
Vitamin B complex	For the nerves	Fat soluble Vitamin	53	58.2
Folic acid	Folate deficiency	Vitamin B	30	33.0
Amitriptyline	Antidepressant	Tricyclic Antidepressant	21	23.1
Trifluoperazine	Antipsychotic	Phenothiazine	20	22.0
Pyritinol	Cerebrovascular & mental disorders	An inootropic agent	17	18.7
Benzhexol	For extrapyramidal effects	Antiparkinsonism	16	17.6
Haloperidol	Antipsychotic	Butyrophenone	9	9.9
Thioridazine	Antipsychotic	Phenothiazine	6	6.6
Imipramine	Antidepressant	Tricyclic Antidepressant	5	5.5
Chlorpromazine	Antipsychotic	Phenothiazine	2	2.2

Side effects were recorded for forty four patients. 20.5% of them experienced headaches and forgetfulness. Available details in Table 4.24.

Table 4.24: **Common Drug Side Effects Recorded among Epileptic Patients Treated at ABUTH**

Side Effects	Number of Patients	Percent of Patients
Forgetfulness	9	20.5
Headaches	9	20.5
Body weakness	8	18.2
Restlessness	6	13.6
Dizziness	6	13.6
Insomnia	5	11.4
Skin rashes	4	9.1
Pruritius	3	6.8
Poor appetite	3	6.8
Excessive sleep	3	6.8
Epigastric pain	3	6.8
Depression	3	6.8
Fear	3	6.8
Dystonia	3	6.8
Diarrhoea	2	4.5
Lack of Libido	2	4.5

The following side effects (not shown in table 4.24) occurred in some patients: agitation, blurred vision, drowsiness, dyspepsia, lethargy, nausea and vomiting, palpitation, breathlessness, spasm of the right side of the neck, abdominal pain, vertigo, erectile dysfunction, waist pain, periorbital swelling, nightmares, bedwetting, swelling of feet, thought interruption, disorientation and abnormal behaviour.

Side effects of drugs presented in these patients were adequately managed through the following ways. Patients were counseled and usually such counseling is geared towards continuing their medication and that the side effects would subside with time. Other patients' drugs were discontinued and another drug was prescribed for such patients; however, drugs were also prescribed for them to remedy the side effects. Eight patients had their drugs discontinued and an alternative drug was prescribed. Drugs were prescribed for 21 patients to remedy their side effects. Doses of drugs were reduced for 4 patients and one patient had the frequency of drugs administration increased while another had it reduced.

4.10 NON-PHARMACOLOGICAL MANAGEMENT

Non-pharmacological management carried on the epileptic patients included, counseling, psychotherapy and group counseling.

Non-pharmacological management was not recorded for 20 patients. Counseling of patients by their Doctors either to comply with treatment or follow-up is the most common form of non pharmacological management noted in this study (84.5% of the patients). Psychotherapy was carried out on 12 (16.9%) patients. Only one patient underwent group counseling. However, physiotherapy was recommended for a patient as a result of comorbid disease (patient was experiencing weakness of flexor of his right hand for more than three days).

Use of alternative therapy prior to medical intervention was not recorded for 57 (62.6%) patients. Sixteen of 34 (47.1%) who had record of use of alternative therapy had never used alternative therapy. However, seventeen (50.0%) had used traditional medicine while one (2.9%) had underwent religion intervention.

4.11 TREATMENT OUTCOME AND REMISSION

Remission indicates patients that were free of seizure. Six of 22 (30.0%) patients who had been on treatment for more than 4years attained more than 2 years remission, while 10 (50.0%) of them still experienced seizures within 6 months of their last clinic visit. Five of 51 (9.8%) patients out of those that were <2years since contact with orthodox facilities attained at least one year remission. Details available in Table 4.25

Table 4. 25: **Cross Tabulation of General Treatment Outcome and Years since Contact with Orthodox Facilities**

Years since contact with orthodox facilities	General Treatment outcome				Total
	Free of Seizure in > 2years	Free of Seizure between 1-2 years	Seizure between 6month-1year	Seizure in <6months	
<2 years	-	5 (9.8%)	14 (27.4%)	32 (62.7%)	51
2-4 years	4 (20.0%)	5 (25.0%)	5 (25.0%)	6 (30.0%)	20
>4years	6 (30.0%)	2 (10.0%)	2 (10.0%)	10 (50.0%)	20
Total	10	12	21	48	91

Patients who had missed clinic appointment at least for 4 months were regarded as clinic defaulters. Out of 91 patients enrolled in this study, only 39 (42.9%) did not default in their clinic attendance, while 52 (57.1%) were defaulted on clinic attendance. Details are shown in Table 4.26.

Table 4. 26: **Cross Tabulation of Years since Contact with Orthodox Facilities and Clinic Attendance**

Years since contact with orthodox facilities	Clinic Attendance		Total
	Did not Default Clinics	Defaulted Clinics	
<2 years	18 (35.3%)	33 (64.7%)	51(100.0%)
2-4 years	9 (45.0%)	11 (55.0%)	20 (100.0%)
>4years	12 (60.0%)	8 (40.0%)	20 (100.0%)
Total	39 (42.9%)	52 (57.1%)	91

Out of 73 patients that resided in Kaduna metropolis, 32 (43.8%) did not default clinics, while 41 defaulted (56.2%). Out of 18 patients that resided outside Kaduna metropolis, 7 (38.9%) did not default clinic attendance, while 11 (61.1%) defaulted clinic attendance. Available details in Table 4.27.

Table 4. 27: **Cross Tabulation of Residence and Clinics Attendance of Epileptic Patients seen at ABUTH**

Residence	Clinic Attendance		Total
	Did not Default Clinics	Clinics Defaulters	
Within Kaduna	32 (43.8%)	41 (56.2%)	73 (100.0%)
Outside Kaduna	7 (38.9%)	11 (61.1%)	18 (100.0%)
Total	39	52	91

2 x2 Chi square test with rate correction for continuity is 0.144, p value =0.704

Remission indicates patients that were free of seizure. Out of 39 patients that has been attending clinic for more than 2 year, 17 (43.6%) attained one year remission while 22 (56.4%) were still experiencing seizure within their last year of clinic attendance. Available details in Table 4.28.

Table 4.28: **Cross Tabulation of Years since Contact with Orthodox Facilities and Remission of Epileptic Patients as seen at ABUTH**

Years since contact with orthodox facilities	Remission		Total
	One year remission	Not in remission	
>1-2 years	5 (13.9%)	31 (86.1%)	36 (100.0%)
> 2years	17 (43.6%)	22 (56.4%)	39 (100.0%)
Total	22	53	75

2 x2 Chi square test with rate correction for continuity is 7.96, p value= 0.005

Patients who had at least one record of poor adherence between 2003 and 2004 were said to have poor adherence to therapy. Eighteen (40.9%) of 44 patients that adhered to their therapy attained one year remission, while 26 were not yet free of seizure. Details are shown in Table 4.29.

Table 4.29: **Cross Tabulation of Adherence and Remission of Epileptic Patients as seen at ABUTH**

Adherence	Remission		Total
	One year remission	Not Seizure free	
Good	18 (40.9%)	26 (59.1%)	44 (100.0%)
Poor	4 (12.9%)	27 (87.1%)	31(100.0%)
Total	22	53	75

2 x2 Chi square test with rate correction for continuity is 6.881, p=0.009

Forty five of 64 (70.3%) patients, who had more than one year of registration, were diagnosed as generalized seizure while 19 were diagnosed as having partial seizures. Details are shown in Table 4.30.

Table 4.30: **Cross Tabulation of Type of Seizure and Remission of Epileptic Patients as seen at ABUTH**

Type of seizure	Remission		Total
	One year remission	Not Seizure free	
Generalized	10 (22.2%)	35 (77.8%)	45 (100.0%)
Partial	8 (42.1%)	11 (57.9%)	19 (100.0%)
Total	18	46	64

2 x2 Chi square test with rate correction for continuity is 2.613, p value =0.106

Out of 12 patients who had their first attack of seizure before 10 years of age and had more than one year since contact with orthodox facilities in the clinic, 3 attained one year (or more) remission while 9 of them still experienced seizures within their last year of clinic attendance. Available details are shown in Table 4.31.

Adherence to therapy for each group was considered. Nine (75.0%), 15 (55.5%) and 11 (55.0%) of patients who were <10 years, between 10-20 years and >20 years of age respectively adhered to their medication.

Table 4.31: **Cross Tabulation of Age at Onset and Remission of Epileptic Patients as seen at ABUTH**

Age at onset (years)	Remission		Total
	One year remission	Not Seizure free	
<10	3 (25.0%)	9 (75.0%)	12 (100.0%)
10-20	8 (29.6%)	19 (70.4%)	27 (100.0%)
>20	5 (25.0%)	15 (75.0%)	20 (100.0%)
Total	16	43	59

Patients who sought for medical intervention early (i.e. <1year of first seizure experience), did not attained one year remission as compare to those who had longer duration between their first attack of and medical intervention. Details are shown in Table 4.32.

Table 4.32: **Cross Tabulation of Duration between First Attacks of Seizure and Medical Intervention and Remission**

Duration between first attack of seizure	Remission		Total
	One year remission	Not Seizure free	
< 1 year	-	8	8
1-2 years	4	6	10
3-4 years	1	6	7
≥5 years	2	7	9
Total	7	27	34

Five out of 8 patients who sought for medical intervention in less than a year of their first attack of seizure had record of good adherence to treatment, 3 from this group had at least one record of poor adherence in the past two years of clinic attendance. Sixteen out of 26 patients of those who sought for medical intervention in more than a year after their first attack of seizure had record of good adherence to their therapy, while the remaining patients had record of poor adherence in the past two years of their clinic attendance. Available details are shown in Table 4.33.

Table 4.33: **Cross Tabulation of Duration between First Attacks of Seizure and Medical Intervention and Adherence**

Duration	Adherence		Total
	Good	Poor	
< 1 year	5	3	8
1-2 years	8	2	10
3-4 years	2	5	7
≥5 years	6	3	9
Total	21	13	34

The patients enrolled on the table below (Table 4.34) were patients who had 100% clinic attendance between November 2003 and October 2004.

4.12 COST OF EPILEPSY

Table 4.34: **Social Demographical Data, Diagnosis and Cost of Drugs (Both AEDs and all Drugs Used) for 12 Months Therapy**

S/N	Mental Comorbid	Non mental Comorbid	Diagnosis	Age @first contact with orthodox facilities	Occupation	No of Clinics Attended	Years Since first contact with orthodox facilities	Total Cost of Drugs used (~~N~~)	Total Cost of AEDS used (~~N~~)
1		Round worm	G. Tonic-clonic	20	Student	6	1.19	24,200	18,270
2		Round worm	G. Tonic-clonic	11.5	Child	10	1.20	14,016	13,300
3			G. Tonic-clonic	17	Student	10	1.27	33,484	33,033
4			Seizure disorder	48	Petty trader	6	1.28	28,835	25,550
5			TLE	26	Petty trader	7	1.31	34,733	34,569
6	Mental subno.	Malaria	TLE	26	Housewife	11	1.38	37,971	37,501
7			Myoclonic	25	Housewife	13	1.46	43,300	41,274
8			G. Tonic-clonic	40	Farmer	12	1.54	57,105	50,454
9		Malaria	Seizure disorder	8	Students	8	1.98	35,788	33,705
10		Diabetes	G. Tonic-clonic	50	Policeman	6	2.17	25,550	25,550
11		Malaria	G. Tonic-clonic	12	Student	6	2.32	12,775	12,775
12	Amnesia		Mixed seizure	18	Student	9	2.37	45,690	39,239
13	MSC		Complex partial	39	Not recorded	7	2.39	40,643	37,960
14			G. Tonic-clonic	15	Student	4	2.58	15,890	15,890
15	Mental retard.		G. Tonic-clonic	11	Child	9	2.58	27,515	25,550
16			G. Tonic-clonic	12	Student	8	2.60	27,736	25,500
17	Cerebral palsy		G. Tonic-clonic	23	Not recorded	11	2.71	59,331	55,172
18			TLE	26	Student	6	3.27	41,809	37,960
19			G. Tonic-clonic	22	Unemployed	5	3.31	39,428	38,115
20	Anxiety	*Taenia spp*	Complex partial	8	Child	5	3.62	27,160	25,500
21	Anxiety	Malaria	TLE	25	Tailor	6	6.53	7,317	5,390
22		Malaria	G. Tonic-clonic	4	Child	16	7.11	47,696	45,958
23			G. Tonic-clonic	9	Student	5	7.15	11,335	9,415
24			Complex partial	12	Child	16	7.71	31,493	29,402
25			G. Tonic-clonic	21	Student	3	7.92	37,960	37,960
26			TLE	26	Unemployed	12	8.21	42,834	32,096
27	Psychosis		Status epilepticus	39	Teacher	5	8.96	40,880	25,550
28			G. Tonic-clonic	24	Farmer	6	11.51	2,256	2,160
29	Psychosis	Hypertension	G. Tonic-clonic	26	Civil servant	12	16.95	40,673	39,900
30			TLE	28	Student	10	29.83	75,510	74,902

Key: Mental subnormality, TLE ______Temporal Lobe Epilepsy. MSC______Multiple somatic complain G. Tonic-clonic _Generalized Tonic –clonic seizure
Mental retard_____Mental retardation AEDs___antiepileptic Drugs

Thirty eight patients had been accompanied to the clinic all through their clinic attendance, while 2 patients were never accompanied. Details are shown in Table 4.35.

Table 4.35: **Cross Tabulation of Years since Contact with Orthodox Facilities and Percentage of Time Accompanied**

Years since contact with orthodox facilities	Percentage of Time Accompanied					Total
	100	70-99	50-69	1-49	0	
<2 years	27	4	2	18	-	51
2-4 years	8	2	1	8	1	20
>4years	3	2	3	11	1	20
Total	38	8	6	37	2	91

CHAPTER FIVE

5.0 DISCUSSION

5.1 CLINIC AND PATIENTS ATTENDANCE

Results from general clinic attendance in the Psychiatric Department of ABUTH, Kaduna, shows that there was higher attendance in 2004 (6,959) as compared to 2003 (6,460) despite the fact that more clinics were held in 2003 compared to 2004 (100 and 97 clinics respectively (Table 4.1). This could be due to the fact that new cases of mental/neurological disorder were reported in the clinic and/or that there were fewer clinic defaulters in 2004. However, it was reported that resident doctors all over the country were on strike in December 2004, which could account for the lower number of clinics held in December 2004. Based on 52 week year and twice-weekly clinics, the maximum clinics expected would be 104. The 100 and 97 clinics observed therefore, representing 96.2% and 93.3% is indicative that clinics were held regularly. Regular clinics are critical for patient monitoring and management.

5.2 CATEGORIES OF DIAGNOSIS

Based on the 761 patients whose case records were complete, schizophrenia has the highest prevalence of 46.6%. Patients diagnosed of depression, seizure disorders/epilepsy, anxiety and bipolar affective disorder were 13.7%, 12.1%, 8.0% and 7.5% respectively (Table 4.2). This is not in keeping with the WHO health report (2001) which indicated that, depression is the most common mental and neurological disorders affecting 121 million people worldwide, followed by substance abuse disorders (70 million people have alcohol disorder), 50 million were reported to be affected by epilepsy, 37 million suffer from dementia (Alzheimer's disease being responsible for majority of the cases) and 24 million worldwide were said to suffer from schizophrenia at one point in time. Studies by Vicente *et al.,* (2002) working in Chile shows that agrophobia is the most common mental disorder, followed by depressive disorder, dysthmia and alcohol dependence occurring in 11%, 9%, 8%and 6% respectively. High percentage of schizophrenic patients in this study could be due to the fact that the disorder is usually very visible and demands prompt medical attention as compared of some other mental disorders e.g. depression and alcohol abuse disorder.

5.3 SEX DISTRIBUTION

There were more epileptic male (52.7%) than female (47.3%) (Table 4.3). This is similar to what was obtained in some past studies globally, indicating that there is a higher incidences of epilepsy in male than in female (Aguwa *et al.,* 1986; Roccg *et al.*, 2001; Colin *et al.,* 2002). It is noteworthy however, that Dominic *et al.,* (2002) in a study in South East England obtained similar incidence in male and female epileptic patients.

5.4 AGE DISTRIBUTION

At first contact with orthodox facilities, the ages of the epileptic patients ranged from 2.5 to 61 years with a mean of 20.20years. 15.4% were less than 10 years of age, 35.2% were teenagers, 34.1% were in their twenties, 7.7% were in their thirties and 7.7% were in their forties. From this result 50.6% of these patients were less than 20 years, majority of who were teenagers (Fig 4.2). Only 55 patients had their age of onset of epilepsy recorded. 74.5% of these patients were below 20 years of age at the onset of epilepsy (Fig 4.3). This is in similar to findings in past studies in Nigeria that majority of epileptic patients (70%) were less than 20 years of age at the onset of epilepsy. (Aguwa *et al.*, 1996; Osuntokun *et al.,* 1987; www.epilepsyontario.org).

5.5 OTHER SOCIO-DEMOGRAPHICAL FEATURES

56% of the patients had less than 2 years since contact with orthodox facilities, 22% each had 2-4 and >4years years since contact with orthodox facilities. Years since contact with orthodox facilities of the patients in general, ranged from 0.41 to 29.83 years, with mean of 3.41 years. The inference is that more than half of the 91 epileptic patients (56.0%) were new patients who were not stable.

Those who were more than 2 years since contact with orthodox facilities (44.0%) were fewer in number.

Table 4.4 shows that 49.5%, 15.4% and 6.6% of patients were Hausa/ Fulani, Yoruba and Ibo tribes respectively, while 28.6% comprised of other minority tribes. High prevalence in Hausa/ Fulani people (49.5%) could be explained by the fact that the study was carried out in a Hausa/Fulani populated area. The geopolitical zones of 66 patients were derived from recorded state of origin. North West had the highest number of 42 patients (63.6%) out of which 25 (37.9%) are from Kaduna state. Fifteen patients (22.7%) were from North Central, 4 (6.1%) from North East, 3 (4.5%) from South East and 2 (3.0%) from South West. States of origin of 25 of the patients were not recorded. 80.2% of the patients reside in Kaduna metropolis, while 18 reside outside Kaduna (9.9% in Zaria, 2.2% in Rigachukwu, 1.1% each in Kafancha, Kano, Makera, Nasara village, Kagoro, Auchan town and Niger state).

The religion of 4 patients was not recorded. 37.9% and 62.1% of the patients were Christians and Moslems respectively. Religion is unlikely factor in the prevalence of epilepsy; Islam being the predominant religion in the Northern Nigeria.

5.6 MARITAL STATUS, EDUCATION AND OCCUPATION

Sixty four (71.9%), 23 (25.8%), 2 (2.2%) were single, married and divorced respectively, while marital status of 2 patients were not recorded (Table 4.5). The high population of teenagers (41 patients) could account for high percentage of singles and is therefore not surprising. Fifty two patients' (57.1%) educational level was not recorded. Of those recorded, about half (51.2%) were below primary school certificate level and 18.0% only had advanced beyond junior secondary school (Table 4.6). This is in agreement with the patients' age and marital status which supported that most were children/teenagers and were single. Six patients had no occupation record. More than two-third (81.1%) of the epileptic patients were unemployed. They are; children (36 or 42.3%), students (18 or

21.2%), housewives (11 or 12.9%) and applicants (4 or 4.7%). Table 4.7 shows details of patients' occupations. The most salient point being that 81.1% (69 out of 85) had no income. Again, these tallies with age, marital and education status.

5.7 RISK FACTORS FOR EPILEPSY

Risk factors frequently exist for diseases and epilepsy is no exception. Risk factors were recorded in only 38 of the patients and these are, febrile convulsion which occurred in 20 patients (52.6%) and family history in 9 patients (23.7%). Other risk factors identified were road traffic accident, head injury, infections (measles, encephalitis) and neonatal jaundice. However, one patient started experiencing seizures after parturition (Table 4.8). This data is similar to other reports that febrile convulsions, head trauma, meningo-encephalitis, perinatal causes, cerebral palsy were the major causes of seizures in Nigeria (Ogunniyi *et al.*, 1987; WHO, 2004). Careful appraisal of Table 4.9 shows that 15 patients experienced febrile convulsions prior to development of epilepsy, of which 10 had febrile convulsion between 6months and 3years of age. According to WHO (2004), children less than 6 years old are prone to convulsion when they have high fever. In addition, the risk of developing chronic epilepsy following febrile convulsion is 2-7% and prolonged febrile convulsion has been shown to be associated with the development of temporal lobe epilepsy later in life (MOH, 1999).

Specific mental and neurological disorders identified with respect to family history as a risk factor are shown in table 4.10. Out of 54 patients whose family history of mental /neurological disorder was documented, 39 have no family history of mental or neurological disorder, 15 patients had specified mental or neurological disorder as shown in the table 4.13. Disorders that occurred in family varied from epilepsy (9), febrile convulsion (2) and psychosis (2). While the specific mental /neurological disorder occurring in the family or in family member was not recorded for two patients. Relationship with the family member varies from siblings (10), fathers (2), Uncles (2) and Cousin (1). This implies that there could be genetic predisposition of epilepsy in some patients (WHO, 2004).

5.8 DIAGNOSIS OF EPILEPSY

Generalized seizures accounted for 54 patients (59.3%) while partial seizures accounted for 23 patients (25.3%). Other types of seizures encountered in this study accounted for the remaining 15.4% (Table 4.11). Equal numbers (27) of generalized seizures were present in both sexes. Partial seizures were present in 12 males and 11 females. The lower percentage of partial seizures (25.3%) reported in this study is similar to other studies conducted in Africa. Studies in Saharan Africa had found clinical evidence of partial seizure in up to 38% (Berhanu *et al.*, 2002), while studies in Central Ethiopia found that 20% of epileptic patients had seizure of partial onset when clinical criteria alone were used (Berhanu *et al.*, 2002). However, high proportion was found for partial seizure (53.3%) in a community-based study in Nigeria (Ogunniyi *et al.,* 1998).

From Table 4.11; more than half of the patients (53 or 58.2%) were diagnosed as having generalized tonic-clonic seizure disorder, 20 or 22.0% were diagnosed of complex partial seizure. Other diagnoses included mixed seizure disorder, simple partial seizure, each of which were 2 or 2.2%; while myoclonic, post traumatic, partial motor seizures and status epilepticus were diagnosed in one patient each (i.e. 1.1% for each of the seizure type). Ten or 11.0% of the patients were simply diagnosed as having seizure disorder (i.e. unclassified). Table 4.12 shows that 5 patients (50.0%) of those diagnosed of seizures disorder (unclassified) were teenagers, 8 patients (40.0%) diagnosed of complex partial were in their twenties. Of 53 diagnosed of tonic-clonic seizures (Grand mal) 41.5% were teenagers and 37.7% were in their twenties. These tallies with the general age prevalence of epilepsy in this study.

Seven out of the 15 patients with history of mental/neurological disorder were diagnosed of generalized tonic-clonic seizures, 5 accounted for complex seizures while status epilepticus, myoclonic and mixed seizure were diagnosed in one patient each, however, family history of mental/neurological disorder was not recorded for patients diagnosed of simple partial, post traumatic seizure and partial motor seizure (Table 4.13).

Table 4.14 shows how epilepsy negatively affects education. Effect of epilepsy on academics was not recorded for 55 patients. Although, 14 patients of this 55 were neither student nor pupil. Of those recorded, 11 patients (30.6%) were doing well academically, while 18 patients (61.10%) dropped out of school because of epilepsy, 4 patients (11.1%) can not go to school because of epilepsy and 3 patients (8.3%) were slow in learning when compared to their siblings who were not epileptic. Past studies indicated that children with epilepsy had a lower performance at school than other pupils, including those suffering from other chronic diseases that affected their attendance at school (Robert *et al.*, 2001). Past studies also showed that frequent seizure have a determining effect on not only education but marital life (Berhanu and Prevett, 2004; Tsuji, 2004; Wada *et al.*, 2004**).** Out of 23 married patients, two patients were divorced as a result of the epilepsy. Though, 16 patients' marital lives were not affected by epilepsy. Twenty five (27.5%) of the 91 patients were above the marriageable age. However one of these 25 patients was unable to marry because of epilepsy (Table 4.15).

Duration between first seizure attack and medical intervention was recorded for only 42 patients. Nine patients (21.4%) sought for medical intervention in less than a year of onset of epileptic seizure, 12 patients (28.6%) in 1-2 years, 10 patie 96 [illegible].8%) in 3-4 years and 11 patients (26.2%) after 4 or more years after first seizure attack (Tab [illegible]). Thus, a total of 78.6% of the patients for whom record was available presented themselves for medical intervention one year or more after the first seizure attack. The possible effect of this delayed treatment on prognosis of the disorder is further discussed on page 107.

Frequency of seizure attack before seeking medical treatment was recorded for only 26 patients, while others (65 patients) were not recorded (Table 4.17). Though, this number was rather small for any statistical analysis, it does appear that a large proportion of those indicated (38.5%) experienced seizures at the rate of 60-90 episodes in one month. In addition, another 15.4% experienced 8-12 episodes per month. Sridharam (2002) reported that patients with high frequency of seizures prior to treatment have lower rate of attainment of remission. Wilner's (2005) studies revealed that the more the number of seizures a patient had in 3 months before starting treatment, the higher the chances of having a refractory seizure.

Although period of seizure attack was not recorded for 60 patients (65.9%), seizures usually occurred in 21 (67.7%) of the patients at night, 9 (29.0%) had seizures either during the day or night, while 3.2% had seizure during sleep (Table 4.18). Literature revealed that seizure can occur during the day (diurnal), during the night (nocturnal) or during the day and night (diffuse) (Gibberd and Bateson, 1974; D'Alessandro *et al.*, 2004).

Sign and symptoms: as illustrated on Table 4.19, more than two-third of the patients (70 or 76.9%) presented with tonic-clonic sustained muscle and relaxation, 61 or 67.0% experienced loss of consciousness during attacks. Twenty eight patients (30.8%) experienced psychic symptoms (which included; slightly elevated mood, social withdrawal, emotional upset, irrational talks, inappropriate speech, talkativeness, restlessness, paranoid ideas, insight impairment, grandiose ideas, excessive worries, excessive laughter, aggressiveness and mute to conversation). Frothing in the mouth during attacks was experienced in 8 (19.8%) of the patients, urinary incontinence and abnormal/bizarre behaviour in 16 (17.6%) and 13 (14.3%) patients respectively.

Eleven patients (12.1%) each presented with hallucination (auditory, visual and olfactory), headaches and abnormal body movement. Jerking movement of the upper limbs and biting of lips were presented by 8 patients (8.7%) each. Other presentation includes memory impairment and dizziness 7 (7.7%) each, insomnia 6 (6.6%) and fecal incontinence 4 (4.4%).

The following symptoms were also reported, though they are less frequent: clonic jerking of the thumb, fear (of being alone or of darkness), jerking movement of the head and neck, impaired consciousness lasting for 30 second to 2 minutes, sweating, neglect of self care. Others includes; tremor on right side of the body, post ictal sleep, rapid movement of eyes and upper limb, sluggishness, unclear speech, forgetfulness, automatism (unusual repetitiveness), inability to take care of self properly and inability to raise upper limb above shoulders.

The signs and symptoms presented by these patients are typical of seizure disorders as described by previous workers (Porter and Meldrum, 1998; Eric *et al*, 2000; WHO, 2004). However, few of them may be as a result of the comorbid disorders such as; anxiety, psychosis (including organic psychosis), abnormal behaviour, mental retardation, dissociative disorder, cerebral palsy, neurological abnormality, multiple somatic complaints, depression, mental subnormality, amnesia and psychiatric disorder. Details of comorbid disorders will be discussed later.

Diagnoses were arrived at based on history (from patients and an eye witness of seizure attacks) and also clinical presentations by the patients coupled with clinical investigations (Table 4.20). Most common laboratory investigations carried out included: serum urea & electrolyte, full blood count & differential, general haematological investigations, stool microscopy, culture and sensitivity of urine, urine microscope, serum glucose, malaria parasite test, urinalysis, fundoscopy and liver function test. These may or may not be related to the epilepsy diagnosis but were carried out to be sure the seizure experienced by the patient is not as a result of infections, brain intoxication and/or metabolic abnormalities. However, Electroencephalogram (EEG), Computerized Tomography (CT) scan, skull x-ray which were performed also in some patients were obviously relevant. EEG and CT scan provide some help in determining the prognosis of interictal epileptiform discharges and focal abnormalities. Skull X-ray also helps to detect abnormality in the brain (Cavazos *et al.,* 2004; WHO, 2004). Some of the laboratory investigations are actually carried out to monitor adverse effects of some of the drugs administered to these patients. For example phenytoin causes megaloblastic anaemia while Succinimides causes bone marrow depression.

The most commonly reported comorbid mental or neurological disorder was anxiety, which was presented by 4 patients (1 male and 3 female) (Table 4.21). This is in support of past studies that, anxiety is prevalent among people with epilepsy (Gilliam *et al.,* 2003). Other comorbid disorders included; psychosis (including organic psychosis) presented in 4 patients, abnormal behaviour (personality changes) and mental retardation, presented in 2 patients each. Dissociative disorder, cerebral palsy, neurological abnormality, multiple somatic complaints, depression, mental subnormality, affective disorder and amnesia which were also presented.

5.9 DRUG TREATMENT OF EPILEPSY

5.9.1 Antiepileptics and Other Drugs Used

Drugs which were prescribed between 2003 and 2004 at ABUTH Psychiatric Department Kaduna for the management of epilepsy and comorbid disorders, including the number of patients that used each drug (Table 4.22 and Table 4.23). Most of the drugs were antiseizures/ antiepileptics drugs (AEDs) such as; carbamazepine, benzodiazepines (diazepam, clonazepam and nitrazepam) barbiturate (phenobarbitone and primidone), phenytoin, sodium valproate and ethusuximide.

Among all the drugs prescribed, carbamazepine was the most commonly prescribed drug, used by 91.2% of the patients (Table 4.22), followed by the benzodiazepines (total 49.4%) while barbiturates were used by 24.2% of the patients. It is noteworthy that phenobarbitone is generally regarded as the most commonly prescribed antiepileptic drug (AED) in developing countries on accounts of its being 5, 15 and 20 times cheaper than phenytoin, carbamazepine and sodium valporate respectively, (Robert *et al*., 2001; WHO, 2004). None of the newer AEDs such as; gabapentin, lamotrigine, levetiracetam, tiagabine, topiramate and zonisamide were used in these patients.

Overall, the use of the above drugs was based on sound pharmacological rationale either as first line or second line drugs for the seizure type presented by each patient. For example, carbamazepine, used in 91.20% of all the patients is a drug of choice in both generalize and partial seizures except in myoclonic seizures. The benzodiazepines used in 49.4% are also generally effective in both classes of seizures in addition to myoclonic seizures. The latter drugs are also preferred to the barbiturates (used in 24.2%) on account of their greater efficacy and lesser side effects. It was not surprising therefore, that although barbiturates are cheaper and frequently prescribed as front line drugs in epilepsy, discerning clinicians could opt for the benzodiazepines. Phenytoin and sodium valporate were rarely used (7.7% and 4.4% respectively) possibly on account of their relatively high cost. Ethosuximide, indicated specifically for absence seizures, again was hardly used in these patients, probably because absence seizure was not diagnosed in any of the patients.

Antipsychotic drugs such as Phenothiazine (trifluoperazine, thioridazine and chlorpromazine) and haloperidol were also used for associated psychic symptoms or comorbid psychosis presented by the patients (Table 4.23). Antidepressants used were amitriptyline and imipramine, both of which were tricyclic antidepressants. Other drugs prescribed were employed either to treat side effects (e.g. benzhexol) or as adjuncts/nutritional supplements (e.g. vitamin B complex, neurobion, pyritinol and folic acid). Other comorbid diseases or infection observed among the epileptic patients (not included in table) were appropriately treated with antihelmitics, antimalarias, antihypertensives, antibiotics, antifungals and vitamin E). There was also good pharmacological basis for the uses of these other drugs. Only 25 patients (out of 91 patients) were on one AED (excluding adjuncts and therapy used for comorbid disease or mental disorders), others were on two or more AEDs.

Overall, polypharmacy was more the norm than exception creating possibilities for drug-drug interactions and poor compliance. It was observed however that there were no records of drug-drug interactions. Though only four and one patient was diagnosed of anxiety and depression, amitriptyline and imipramine were prescribed for 21 and 5 patients respectively. There was no obvious rationale for prescription of these drugs in patients that were not diagnosed nor had anxiety or depression as side effect of the AEDs used.

5.9.2 SIDE EFFECTS EXPERIENCED AND THEIR MANAGEMENT

Forty four (48.4%) of the epileptic patients did not report side-effects. However, Common side effects experienced by the patients were: forgetfulness, headaches, body weakness, restlessness, dizziness, insomnia, skin rashes, pruritus, poor appetite, excessive sleep, epigastric pain, depression, dystonia, diarrhea and lack of libido (Table 4.24). Forgetfulness experienced by 20.4% of the patients is an important side effect of barbiturates as is body weakness and dizziness. Headache also experienced by 20.4% of the patients is a common side effect of the drugs used. Skin rashes, dizziness, breathlessness, nausea and vomiting are known side effects of carbamazepine. While drowsiness and lethargy are the principal side effects of long-time use of benzodiazepine. Thus, the side effects profile observed in this study were easily attributable to the drugs employed.

Side effects were managed for the majority of patients although there was no record of management of side effect(s) for 11 patients, several approaches were adopted in the side-effect management as detailed below.

Patients were counseled and usually such counseling is geared towards continuing their medication (which was essential) and that the side effects would subside with time. Other patients' drugs were discontinued and another drug was prescribed for such patients; however, drugs were also prescribed for them to remedy the side effects (e.g. carbamazepine caused skin rashes in a patient and was withdrawn. In this patient carbamazepine was replaced with primidone and prednisolone was prescribed to remedy the side effect). Eight patients had their drugs discontinued and an alternative drug was used (carbamazepine was replaced with primidone and clonazepam in a patient experiencing breathlessness as it side effect). Drugs were prescribed for 21 patients to remedy their side effects (e.g. benzhexol used for extrapyramidal side effects). Doses of drugs were reduced for 4 patients (e.g. trifluoperazine dose was reduced in a patient because of extrapyramidal effect) and one patient had the frequency of drugs administration increased (e.g. benzhexol dose was increased for a patient who experienced a server dystonic reaction) while another had it reduced (e.g. carbamazepine frequency was reduced as patient was experiencing dizziness and general body weakness).

Overall, there was sound pharmacological basis for the drug treatment of these epileptic patients and the management of side effects although it was observed that the newer, more efficacious but more expensive AEDs were not used at all in these patients.

5.10 NON-PHARMACOLOGICAL MANAGEMENT

Counseling of patients by their doctors either to comply with treatment or follow-up is the most common form of non-pharmacological management noted in this study being carried out in 84.5% of 71 patients who underwent non-pharmacological management. Psychotherapy was used in 12 patients (16.9%), while group counseling was carried out in 1 patient (1.4%). Group counseling is a counseling method in which patients with similar psychological problems were brought together by the health care team and the psychologist to discuss their problems and how such can be tackled. This method is beneficial to the patients as they shared from each other's experiences.

Half (50.0%) of those who had record as regards use of alternative therapy had used traditional medicine, while one patient 2.9% had sought for religious intervention. This also support pasts studies that most epileptic patients in Africa visit traditional healers first before visiting the hospital (Berhanu *et al.*, 2002; WHO, 2004). This study did not ascertain whether the use of these alternative therapies continued after commencement of treatment at ABUTH.

5.11 TREATMENT OUTCOME AND REMISSION

Considering overall outcome of treatment in all the patients (Table 4.25), among the 51 patients who were attending clinics for less than 2years, 32 (62.7%) experienced seizures in less than 6months of their last clinic attendance in 2004, while 14 patients (27.4%) had seizures between 6 month and one year of their last clinic attendance. However, 5 (59.8%) attained one year remission. Among the 20 patients who had been attending clinics for 2-4 years, 4 (20.0%) has been free of seizure for more than 2 years, 5 (25.0%) were free of seizure for 1-2 years, 5 (25.0%) had seizures between 6 month and one year of their last clinic attendance, while 6 (30.0%) still experienced seizures in the proceeding 6 month as at their last clinic attendance. Among the 20 patients who has been attending clinic for four years and above, 6 (30.0%) were free of seizure for more than 2years, 2 (10.0%) were free of seizure for 1-2 years, 2 (10.0%) were free in the past 6 month and year, while 10 patients (50.0%) among this group still experienced seizure in the past 6 months of their last clinic attendance (Table 4.25). Thus, the longer the years since contact with orthodox facilities the more likely the patients were free of seizures confirming that management of their epilepsy was yielding positive results.

As shown on Table 4.26, 39 patients (42.9%) out of 91 used for this study did not default their clinics attendance. However 52 patients (57.1%) defaulted. Thus, overall, more patients defaulted. 64.7% of patients who were <2 years since contact with orthodox facilities, 55.0% of those with 2-4 years since contact with orthodox facilities and 40.0% of those with >4 years since contact with orthodox facilities were clinic defaulters. This implies that patients who were <2 years since contact with orthodox facilities defaulted clinic appointment more than the other groups. This group also tended to have a poorer treatment outcome with 90.1% having seizures within the last year of their clinic attendance.

Out of 73 patients that resided in Kaduna metropolis, 32 (43.8%) did not default clinic in 2003 and 2004, while 41 defaulted clinics (56.16%). Out of 18 patients that reside outside Kaduna metropolis, 7 (38.9%) did not default clinic attendance, while 11 (61%) defaulted clinic attendance (see Table 4.27). Though, the percentage of those clinic defaulters that reside outside Kaduna metropolis was higher than those residing within, the difference is not statistically significant ($p< 0.05$).

Data for Patients who had registered for more than a year were extracted for further analysis on attainment of at least one-year remission as of their last day of clinic appointments.

Table 4.28 shows that out of 39 patients that has been attending clinic for more than 2 years, 17 patients (43.6%) attained one year remission while 22 (56.4%) were still experiencing seizures within their last year of clinic attendance. On the other hand, patients who were 1- 2 years since contact with orthodox facilities and who attained at least one year remission were less in number i.e. 5 patients (13.9%), 31 patients (86.1%) are not free of seizures within their last year clinic visit. There is a significant different ($p< 0.05$) in attainment of at least one year remission in these two groups. This implies that the longer a patient is on therapy the better the outcome. This is similar to findings of Specchio *et al.,* (2002) studies which revealed that there was decrease in seizure frequency and better prognosis as treatment continued, with exception in patients who had other mental disorders.

From Table 4.29, 18 (40.9%) out of 44 patients that adhered to their therapy attained one year remission, while 26 (59.1%) are not yet free of seizure. However, only 4 patients (12.9%) out of 31 that had at least one record of poor adherence (between 2003 and 2004) attained one year remission, while the remaining 27 (87.1%) had not attained at least one year remission as of their last clinic visit. The difference in the two groups' attainment of remission is statistically significant ($p< 0.05$). Therefore failure to adhere to therapy has a significant effect on attainment of remission in these patients. This is similar to what is obtained in the literature. Poor compliance to prescribed medications is considered to be the main cause of unsuccessful drug treatment for epilepsy (French, 1994; Gledhill, 1997; Gomes and Maia, 1998; Mitchell *et al*., 2000). Patients that failed to comply with therapy experienced increase in number and severity of seizures (Leppik, 1990; Hargrave and Remler, 1996; WHO, 2004).

Considering attainment of remission in relation to class of seizure, out of 64 patients who had more than one year since contact with orthodox facilities, 45 were diagnosed of generalized seizure while 19 were diagnosed of partial seizure (Table 4.30). However, there is no significant difference ($p< 0.05$) in the attainment of at least one year remission in these two classes of seizures (generalize and partial). Care was also taken to ensure that adherence to treatment for both groups was similar (i.e. there was no significant difference in their adherence). This suggests that the type of seizure did not affect attainment of remission in epileptic patients in as much as the patient adhered to therapy. This result also is similar to results obtained in past studies in Ethiopia (Berhanu and Prevett, 2004) and United Kingdom, where a prospective population based study showed that seizure type had no significant effect on the chances of achieving remission (Cockerell *et al.*, 1995). Although, Deker (2002) and Sirdharam (2002) studies have a contrary view, partial seizure was observed to have lower rate of remission as compared to generalized seizure.

Out of 47 patients who had their first attack of seizure before 10 years of age and have more than one year since contact with orthodox facilities in the clinic, 3 (25.0%) attained of remission while 9 (75.0%) of them still experienced seizures within their last year of clinic appointments. Out of 27 patients who had their first seizure attack between 10-20 years of age and have more than one year since contact with orthodox facilities in the clinic, 8 (29.6%) attained remission, while 19 (70.4%) still experienced seizures within their last year of clinic appointment. Out of 20 patients who had their first seizure attack above 20 years of age and have more than one year since contact with orthodox facilities in the clinic, 5 (25.0%) attained remission, while 15 (75.0%) still experienced seizures within their last year of clinic appointment (Table 4.31). Poor adherence to treatment could accounts for low remission rate among patient between 10-20 years and > 20 years. However, there was no better rate of remission of seizure in patients that were less than 10 years despite the fact that majority of them adhered to their treatment (75.0%) as compared to those between 10-20 and >20 years of age (55.5% and 55.0% respectively). Therefore, early presentation of seizure could probably have effect on attainment of remission. This finding is similar to what was obtained in the study conducted in Ethiopia, where age of onset before 15years and frequency prior treatment were both associated with failure to become free of seizures (Berhanu and Prevett, 2004). Sirdharam (2002) also reported that the lower the age of onset, the lower the rate of attainment of remission.

From Table 4.32, seven patients (26.9%) out of 26 who sought for medical intervention after at least one year of first seizure attack attained one year remission, while the remaining patients in this group (19) still experienced seizure(s) in the past one year of their clinic attendance. None of the 8 patients who sought for medical intervention early (i.e. <1 year of first seizure experience), on the contrary, attained one year remission. When adherence to therapy was considered for the two groups of patients, 5 (62.5%) patients from the latter had record of good adherence to treatment, 3 (37.5%) from this group had at least one record of poor adherence to therapy in the past two years of clinic attendance. However, table 4.33 demonstrated that 16 (61.5%) out of 26 patients in the former group had record of good adherence to their therapy, while the remaining patients (38.5%) had record of poor adherence to therapy in the past two years of their clinic attendance. From this result, there was no difference in the percentage of patients that had record of good adherence to therapy in the two groups (62.5% of the latter group and 61.5% of the former group). Therefore, remission was not attained in patients that sought for medical attention not later than one year after their first seizure attack. From these result, patient who have been having epilepsy long before medical treatment still have a chance of better prognosis of their epilepsy as seen in patients who have been having seizure for more than one year before seeking medical intervention. This is similar to finding from other studies that duration of epilepsy did not influence seizure control (Berhanu and Prevett, 2004). However, Sridharam, (2002) and Deker (2002) findings indicated that the shorter the duration of seizure prior treatment the higher the rate of attaining remission.

5.12 COST OF EPILEPSY TREATMENT IN ABUTH KADUNA

The patients whose data were tabulated on table 4.34 are patients who were regular in the clinic between November 2003 and October 2004 (100.0% clinic attendance). This criteria was used in order to assess the amount they spent on their drug during one year course of treatment as required for cost prevalence studies. The minimum years since contact with orthodox facilities were 1.19 and maximum of 29.83 years. The minimum clinic attendance was 3 and maximum was 16 for patients that had 7.92 and 7.71 years since contact with orthodox facilities respectively.

Two patient's occupation was not recorded. Eight were either self employed or civil servants. Twenty out of thirty patients (66.7%) were unemployed (students, child, housewives or applicants) who depend on their parents or husband for financial support. This implies that the cost of treatment of their illness is the sole responsibilities of their care givers (dependants). The minimum cost of AEDs is ₦2, 160 and maximum was ₦74, 902, the mean annual cost for AEDs is ₦30, 986.67 (Table 4.41). However, minimum total cost of drugs including drugs used for comorbid disorder/disease and also drugs use as adjunct is ₦2, 256 and maximum was ₦75, 510. The mean cost of total drugs taking by these patients was ₦33, 697.10. Based on National Per Capital Income (which is ₦4.46 according to 2004 Central Bank of Nigeria bulletin), the patients or their caregiver were really affected financially. It is noteworthy that cost items which includes; direct cost (hospital in-patients and out-patients services, care by physician and other practitioners, rehabilitation services, transport fare, special equipments used, home services and others) and indirect cost such as; unemployment (patients or caregivers), underemployment, lost of productivity of patients and caregivers, decrease household work and soon on were not used in this study.

Regression analysis of clinic attendance and cost of AEDs drugs showed that cost increased with increased clinic attendance. This could be due to the fact that patients attending clinic frequently (less stable patients) drug(s) doses and frequencies may have been increased and therefore resulting in increased in their drug cost. Drug cost decreased with decreased in clinic attendance. These could be as a result of good treatment outcome and therefore decreased in frequencies and doses of drug(s) prescribed for such patients. The strength of this relationship is however not strong when the data was compared using correlation (0.454). One way analysis of variance shows no significant difference in cost of either total drugs (F=0.449, sig. =0.838) or cost of AEDs used (F=0.348, sig. = 0.904) and type of seizure diagnosed for patient. Regression analysis revealed a negative relationship between years since contact with orthodox facilities and cost of AEDs. The strength of this relationship was however weak when the same data were compared using correlation (0.358). This result indicated that cost of AEDs used by the patients reduced with increased in years since contact with orthodox facilities. This finding is similar to that of Berto *et al.,* (2000) and Forsgren *et al.,* (2005) studies which showed that direct cost (which include cost of drug) reduces with increase in year of clinic attendance/commencement of AEDs. Berto *et al.,* (2000) also indicated that, direct cost accounted for 87.6% of total cost in which drug cost is 10.5%. This implies that drug cost accounted for a small percentage of the cost of illness. This therefore means the cost of illness of epilepsy in

Nigeria (if other cost items listed above were include will be very high and the impact on the economy state of the patients, caregiver and the country as a whole can not be over emphasized.

Thirty eight patients had been accompanied to the clinic all through their clinic attendance, while 2 patients were never accompanied. Fourteen patients were accompanied for more than half of the times (50-99%) of their clinic attendance (see Table 4.42). The effect of lost of time of productivity and cost of transport fare for both the patients and person(s) who accompanied them cannot be over emphasized.

5.13 CONCLUSION

From this study, epilepsy affect marital life (27.5% were above marriageable age but were yet to marry while 2.2% were divorce because of epilepsy) and education of the patients (epilepsy have a negative effect on education of 69.5% of the patients) in ABUTH, Kaduna as it does in other part of the world. There were more male epileptics than female and while half of the patients were 20 years old at first contact with orthodox facilities, larger proportions were less than that age at the onset of epilepsy. Most patients were single. Common risk factors identified for epilepsy in this study were febrile convulsion and family history of epilepsy.

Sign and symptoms presented by these patients included; tonic-clonic muscle contraction and relaxation, loss of consciousness, psychic symptoms frothing in the mouth and urinary incontinence. Diagnosis was made based on information from eye witness of attack, patients and clinical examinations. EEG and CT scan was performed on 7 and 2 patients respectively. MRI (magnetic resonance imaging) was not used for any of the patients because the machine is not available in this hospital. Generalized tonic–clonic seizure was most commonly reported in this hospital. This could be associated with the fact that the features are easily noticeable and need for medical attention was obvious to the patients or their relatives.

Although phenobarbitone is regarded as the first line drug in epilepsy in this part of the world, carbamazepine was the most commonly prescribed drug in this study centre, probably because of the side effects (e.g. cognitive impairment) notable with phenobarbitone therapy (as majority of the patients are children or/and in their active age) and carbamazepine being a drug of choice for both partial and generalized seizures. Side effects of drugs used by patients were less frequently reported and those reported were adequately managed. Psychotherapy was also employed in this hospital and was done by the psychologist. Counseling of patients and their families by doctors was also employed. More females reported comorbid disorder than their male counterparts. Anxiety disorder is the most common comorbid mental disorder in epileptic patients used for this study.

In generally, patients that had less than 2 years since contact with orthodox facilities defaulted clinics attendance than others who had more than 2 years since contact with orthodox facilities. Patients who reside outside Kaduna metropolis had higher percentage of those that defaulted clinic attendance when compared to those residing within the Kaduna metropolis, though the difference is not statistically significant. This study revealed that, the longer patients stay on therapy, the better their outcome (this was found to be significant, $p < 0.05$). Non-adherence to follow-up and therapy has a significant effect on the outcome of treatment in term of attaining one year remission.

From the results obtained from this study, that the type of seizure (Generalized or Partial) does not have a significant effect on attainment of one year remission of seizures. Long duration of epilepsy before seeking medical intervention also did not affect attainment of remission in as much as the patient adhered to therapy. However, this study revealed that early presentation of epilepsy (in <10 years of age) have lower remission rate.

The cost of AEDs used by the patients per year was high in relation to Nigeria per capital income as most of these patients (66.7%) were unemployed. Cost increased with increased in clinic attendance, but decreased with increased in years since contact with orthodox facilities. There was no significant difference in cost of drugs (either total drugs or AEDs) as regards the type of seizure.

Results from this retrospective study (which involved the use of medical records and patients' folder) are indicative of proper record keeping in the psychiatry department of ABUTH, Kaduna.

5.14 RECOMMENDATION

According to WHO, 2004, Nigeria with a population of 113.8 million is served by 11 neurologists, 5 neurosurgeons and 110 psychiatrists. Equipments available for proper diagnoses of epilepsy include; 10 EEG machine, 9 CT scan and 1 MRI. This indicates that there is need for training of more specialists (Neurologists and Neurosurgeons) and also provision of medical equipments in our hospitals especially the tertiary health institutions. This will go a long way in achieving proper, accurate diagnosis of seizure type and epileptic syndrome, as this is important in the choice of AEDs, response to treatment and prognosis of epilepsy.

Effective actions for the prevention of epilepsy should include; adequate prenatal and postnatal care, safe delivery, control of fever in children, control of parasitic and infectious diseases and prevention of brain injury (e.g. control of blood pressure and the use of safety belt and helmets).

In process of supportive therapy for patients and family members by clinician, emphasis should be laid on the need to continue treatment and follow-up except by physician's directives. This is because cessation of seizures does not mean that treatment is no longer necessary as there is possibility of seizure relapse.

To aid adherence to drug therapy, the following are recommended:

- Drugs (especially AEDs) should be made available in the hospitals and pharmacy records should be individualized for each patient.
- Patient who usually missed their drugs can be counseled to use clock/other reminders and be scheduled for more frequent clinic appointment.
- The use of monotherapy (75-80% of people with epilepsy can be managed easily by monotherapy) where possible, will enhance adherence and it will also reduce cost of treatment of epilepsy.

Suggestion for further studies

Cost of epilepsy in Nigeria which will include various cost items (mentioned earlier in Chapter Two) is essential so that the economic impact of epilepsy can be made known to the government, in order that commitment to resources for epilepsy treatment can be gained from government and international health organizations.

REFERENCES

Aguwa, C. N. and Ochei, U. (1996). Epilepsy. *In: Therapeutic Basis of Clinical Pharmacy in the Tropics.* Aguwa C. N. T. (2nd Edition). Optimal Publisher, Enugu, Nigeria. PP. 333-347.

Banerjee, S., Paul, P. and Talab, V.J. (2004). Serum prolactin in seizure disorder. *Indian pediat*., **41**(8):827-31.

Baker, G.A., Jacoby, A. and Chadwick, D.W. (1996). The associations of psychopathology in epilepsy: a community study. *Epilepsy Res*, 25:29–39.

Begley, C.E., Lairson, D.R., Reynolds, T.F. and Coan, S. (2001). Early treatment cost in epilepsy and how it varies with seizure types and frequency. *Epilepsy-Res*, **47**(3): 205-15

Begley, C.E., Ettore, B., Beran, R.G., Heaney, D., Langfitt, J.T., Pachlatko. C., Silfvenius.H., Sperling. R.M. and Wiebe, S. (2002). ILEA Commission on the Burden of Epilepsy Sub commission on the Economic Burden of Epilepsy: Final Report 1998-2001. *Epilepsia*, **43**(6): 668-673.

Benbadis, S.R. and Tatum, W.O. (2001). Advances in the treatment of epilepsy. *Am – Fa – fam – physician*, **1:64**(1): 91-8.

Berhanu, S., Alemu, S., Asmara, J.and Prevett, M. (2002).Primary care treatment of epilepsy in Rural Ethiopia. *The Ethiopia Journal of Health Development*, **16**(3):235-240.

Berhanu, S. and Prevett, M. (2004). Treatment of epilepsy in rural Ethiopia: 2-year follow-up. *The Ethiopia Journal of Health Development*, **18**(1): 31 – 34.

Berto, P., Tinuper, P. and Viaggi, S. (2000). Cost of illness of epilepsy in Italy. Data from a multicentre observational study. *Pharmacoeconomic*, **17**(2): 197-208

Cavazos, J.E., Lum, F and Spitz, M. (2004). Seizures and Epilepsy: Overview and Classification. eMedicine.com, Inc.

Charney, D.S., Mihic. J.S. and Harris, R.A. (2001).Hypnotics and sedatives. *In* Goodman and Gilman's *The Pharmacological Basis of Therapeutics*. (10th edition). McGraw Hill, Medical Publishing Division, New York. Pg 399-427.

Cockerell, O. C., Hart. Y. M., Sander, J. W.and Shorvon, S. D. (1994). The Cost of Epilepsy in The United Kingdom: an estimation based on the results of two population – based studies. *Epilepsy Res*, 18:249-260.

Cockerell, O.C., Johnson, A.L., Sander, J.W., Hart, Y.M. and Shorvon, S. D. (1995). Remission of epilepsy: results from national general practice studies of epilepsy. *Lancet*, 346: 140-44.

Chisholm, D., Sekar, K., Kumar, K., Kishore, K., Saeed, X., James, S., Mubbasbar. M. and Murthy, R. S. (2000). Integration of mental healthcare into primary care: Demonstration cost-outcome study in India and Pakistan. *British Journal of Psycliatry*, 176: 581 – 588.

Colin, D.M., Clandia, S.D., Chalapati, R., Mie, I., Niels, T., Christians, B., Alan, D.L. and Christopher, J.L.M. (2000). *Global Burden of Disease*: Version 2 method and results Commission of Classification and Terminology of the International League against Epilepsy. (1981) *Epilepsia*, 22: 489-501

D'Alessandro, R., Guarino, M., Greco, G., Bassein, L. and Elilia, R. (2004). Risk of seizure while awake in pure sleep epilepsies: A prospective study. *Neurology*, 62:254-257.

Dale, J.R and Ben-Tovim, D.I (1984). Modern or traditional? A study of treatment preference for neuropsychiatric disorders. *Br. J. Psych*, 145:187-192.

Danesi, M.A and Adetunji, J.B. (1994). Use of alternative medicine by patients with epilepsy: a survey of 265 epilepsy patients in a developing country. *Epilepsia*, 35:344-351.

Danesi, M.A., Odusote, K.A., Robert, O.O. and Adu, E.O. (1981). Social problems of adolescent and adult epileptics in developing countries as seen in Lagos, Nigeria. *Epilepsia*, **22**(6): 689-96.

Desai, N.G. and Isaac, M. (2005). Mental health in South-East Asia: reaching out to the community. Regional health forum WHO South East region. Volume 5, number 1.

Deker, P.A. (2002). Epilepsy: A manual for medical and clinical officers in Africa. *WHO publication*.1-133.

De Zelicourt, M., Buteau L., Fagnani, F. and Jallon, P. (2000). The contributing factors to medical cost of epilepsy: an estimation based on a French prospective cohort study of patients with newly diagnosed epilepsy seizures (the CAROLE STUDY). Active coordination of the longitudinal observational network in epilepsy. *Seizure*, 9: 88-95.

Dominic, C.H., MacDonald, B.K., Everitt, A., Stevenson, S., Leonardi, G.S., Wilkinson, P. and Sander, W.J. (2002). Socioeconomic variation in incidence of epilepsy: prospective community based study in South East England. *BMJ*, Volume 325

Edeh, J. and Toone, B. (1987). Relationship between interictal psychopathology and the type of epilepsy: results of a survey in general practice. *Br J Psychiatry*, 151: 95–101

Epilepsy in Africa and the African American Community. Complied by: Puja Sahni http://www.epilepsyontario.org/

Eric, D., Caine, M.D., Jeffrey, M and Lyness, M..D. (2000). Delirium, dementia and amenstic and other cognitive disorders. *In* Kaplan and Sdock' Comprehensive textbook of psychiatry. 7th edition. Lippincott Williams and wikins, Philadelphia, U.S.A. pp 854- 923.

French, J. (1994). Long-term therapeutic management of epilepsy. *Annals of internal Medicine*, 120: 411-422.

Forsgren, E., Beghil and Ekman, M. (2005). Cost of epilepsy in Europe. *European Journal of Neurology*, **12**(1): 54 – 58.

Garnett,W. R.(2000). Antiepileptic Drug Treatment: Outcome and Adherence. *Pharmacothereapy*, **20**(8 pt 2): 1915-1995.

Gelder, M., Mayou, R. and Geddes, J. (1999). Psychiatry. 2nd edition. Oxford university press, New York. pp 184-201.

Gibberd, F.B. and Bateson, M.C. (1974). Sleep epilepsy. Its pattern and prognosis. *BMJ*, 2: 403-405.

Gilliam, F., Hecimovic, H. and Shelinea, Y. (2003). Psychiatric comorbidity, health and function in epilepsy. *Epilepsy & Behavior*, 4: 26–S3.

Gledhill, R.F. (1997). In the shadow of epilepsy. *Lancet*, 350:811.

Gross,R.A.(1992). A brief history of epilepsy and its therapy in the Western hemisphere. *Epilepsy-Res*, **12**(2): 65-74.

Guenot, M. (2004). Surgical treatment of epilepsy: Outcome of various surgical procedure in adult and children (in process citation) *Rev Neuro.*, **160**(1): 241-50.

Hargrave, R. and Remler, M.P. (1996). Non-adherence. Journal of the National Medical Association. 56: 708-713.

Heaney, D.C., Shorvon, S.D., Sander, J.W., Boon, P., Komarek, V., Marvsic, P., Dravet, C., Perucca, E., Majowski, J., Lima, J.L., Arroyo, S., Tomsom, T., Ried, S., Van-Dam-Selaar., Eskazan, C., Peeters, P., Carita, P., Ijong-a-Hung, I., Myon, E. and Taieb, C. (2000). Cost minimization analysis of antiepileptic drugs in newly diagnosed epilepsy in 12 European countries. *Epilepsia*, **41**(5): 37-44.

Hermann, B.P., Seidenberg, M. and Bell, B. (2000). Psychiatric comorbidity in chronic epilepsy: identifications, consequences and treatment of major depression. *Epilepsia*, **41**(2): 31–41.

Herzog, A.G. (1991). Reproductive endocrine considerations and hormonal therapy for men with epilepsy. Epilepsia, **32**(6): 34-7.

Herzog, A.G. (1999). Progesterone therapy in women with epilepsy: a 3-year follow-up. *Neurology*, 52: 1917-8.

Hodgson T. A. and Meiners, M. R. (1982). The cost of illness methodology: a guide to current practices and procedures. Milbank mem fund Q: 60: 49-61.

HTNF.(2002).AllergyGlossary.http/www.hom.ch/library/theme/allergy/Glossary/technic.htm

ILAE/IBE/WHO (2000). Global campaign Against Epilepsy. Geneva, World Health Organization

Janszky, J. (2004). Diagnosis of epilepsy. *Ideggyogy S z*, **57**(5-6): 157-63.

Keranen, T. and Rickkinen, P. (1988). Severe epilepsy: Diagnostic and epidemiological aspects. *Acta – Neurol-Scand*, 117: 7-14.

Kinsman, S.L., Vining, E.P., Quaskey, S.A., Mellits, D. and Freeman, J.M. (1992).

Efficacy of the ketogenic diet for intractable seizure disorder: review of 58 cases. *Epilepsia*, **33**(6): 1132-6.

Kossoff, E.H. (2004). More fat and fewer seizures: dietary therapies for epilepsy. *Lancet-Neurol.*, **3**(7): 415-20.

Kotsopoulos, I.A., Evers, S.M., Ament, A.J. and de Krom, M.C. (2001). Estimating the costs of epilepsy: an international comparison of epilepsy cost studies. *Epilepsia*, **42**(5): 634-40.

Leppik, I.E. (1990). How to get patients with epilepsy to take their medication. The problem of non-adherence. *Postgraduate medicine*, 80: 253-256.

Linsten, H., Stenlind, H. and Forsgen, L. (2003). Leisure time and social activity after a newly diagnosed unprovoked epileptic seizure in adult age. A population based case-referent study. *Acta – Neurol Scan*, **107**(2): 125 – 33.

Martha, J. and Morrell, M.D. (2002).Epilepsy in women. *American Family Physician,* **66**(8): 1489-1494.

Morrell, M.J (2002). Effect of epilepsy on women's reproductive health. *Epilepsia*, **39**(8) 32-7.

Mc Namara, J. O. (2001). Drugs effective in the therapy of the epilepsies. *In* Goodman and Gilman's *The Pharmacological Basis of Therapeutics*. (10thedition). McGraw Hill, Medical Publishing Division, New York. pp 521-547.

Meerding, W. J., Bonneux, L., Polder J.J., Koopmanschap, M.A. and Maas P.J. (1998). Demographic and epidermiological determinants of healthcare costs in the Netherlands: Cost of illness study. *British Medical Journal*, 317: 11 – 115.

Mendez, M.F., Cummings, J.L and Benson, D.F. (1986). Depression in epilepsy: significance and phenomenology. *Arch Neurol*, 43:766–70.

MOH Clinical practice guidelines (1999). Diagnosis and Management of Epilepsy in Adults.

Nassiri, R. and Stelmasiak, Z. (2000). Pharmacotherapy of epilepsy. *Neur o-Neurochin – Pol*, **34**(8): 47-58.

Osuntokun, B.O., Adeuja, A.O.G. and Nottidge, V. A. (1987). Prevalence of the epilepsies in Nigerian Africans: a community-based study. *Epilepsia*, 28: 272-279.

Ogunniyi, A., Oluwole, O.S. and Osuntokun, B.O. (1998). Two–year remission in Nigerian epileptics. *East Afr Med J*, **75**(7): 392-5.

Porter, R.J. and Meldrum, B.C. (1998). Antiepileptic Drugs. *In*: Katzung, B. G. *Basic and Clinical Pharmacology* (7th Edition). Appleton and Lange, Simon and Schuster Company, United States of America, pp. 386-408.

Pritchard, P.B. (1991).The Effect of Seizure on Hormone. *Epilepsia*, **32**(6): 44-50

Pschirrer, E.R. (2004). Seizure disorders in preganancy. *Obstet. Gynecol. Clin North Am-jun*, **31**(2): 373-84.

Ramsay, R.E., Uthman, B.M., Augustinsson, L.E., Upton, A.R., Naritoku, D., Willis, J., Trief,T., Barolat, G.J. and Werickie, J.F. (1994). Vagus stimulation for treatment of partial seizures: 2 safety, side effects and tolerability. First international vagus nerve stimulation study group.

Reis, R. (1994). Evil in the body, disorder of the brain; interpretations of epilepsy and the treatment gap in Swaziland. *Trop Geogr Med*, 46: S40-3.

Rice, D. P., Kelman, S., Miller, L.S. and Dummeyer, S. (1990). The Economic costs of alcohol and drug abuse and mental illness: 1985. Rockville M. D. alcohol, Drug abuse and Mental Health Administration. *ADM*, 90 – 1694.

Robert, S.A., Lhatoo, S.D. and Sander J.W.A. (2001). The Treatment of epilepsy in developing countries: Where do we go from here? *Bulletin of World Health Organisation*, 79: 344-351.

Roccg, W.A., Savettieri, G., Aderson, D.W., Meneglini, F., Grigoletto, F., Morgante, A., Reggio, G., Salem, F., Patti, F. and Perri, R.D. (2001). Door to door prevalence survey of epilepsy in three Sicillian municipalities. *Neuro epidemiology*, 20: 237-241.

Sanchez, L.A. (1999). Pharmacoeconominic: Principles. *In*: Dipiro, T.T, Talbert, L.R, Yee, C.G, Matzke, G.R, Well, B.G. and Posey, L.M. *Pharmacotherapy. A Pathophysiologic Approach* (4th Edition). Mc Graw-Hil,l Medical Publishing division, New York, pp. 1-11.

Senanayake, N. and Roman, G.C. (1993). Epidermiology of epilepsy in developing countries. *Bulletin of World Health Organization*, 71: 247-258.

Schmutz, M. and Toygar (2001). Psychiatric and psychotherapeutic aspects in epileptology. *Ther Umsch*, **58**(11): 668-70.

Sigita Plioplys, M.D. (2003). Psychiatric comorbidity in youth with epilepsy. *The child's Doctor. fall.*

Specchio, L.M., Tramacere, L., LaNeve, A. and Beghi, E. (2002). Discontinuing antiepileptic drugs in patients who are seizure free on monotherapy. *J Neurol Neurosurg Psychiatry*, **72**(1): 22-5.

Sridharam, R. (2002). Epidemiology of Epilepsy. *Current Science*, **82**(6): 664-670.

Swink, T.D; Vining, E.P. and Freman, J. (1999). Ketogenic Diet. *Adv-Pediatr.*, 44: 297-329.

Tapani, K. (2002). Evidence based treatment of newly diagnosed epilepsy in elderly. www.uku.fi/neuro/epilepsy, 2002speaker.pdf.

Tatum,W.O., Benbadis, S.R. and Vale, F .L. (2000). The neurosurgical treatment of epilepsy. *Arch-Fam-Med,* **9**(10): 1142-7.

The Extra Pharmacopoeia. (1993). (30th edition).The Pharmaceutical Press, London.pp 292-1578.

Thomas, S.V. (2000). Money matters in epilepsy. *Neurol. India,* **48**(4): 322-9.

Tsuji, S. (2004). Social Aspects of Epilepsy; marriage, pregnancy, antiepileptic drug withdrawal and against social stigma. *Rinshoshin Keigaku,* **44**(11): 865-7.

Uthman, B.M. (2000). Vagus nerve Stimulation for seizures. *Arch-Med-Res*., **31**(3): 300-3.

Vicente, B., Rioseco, P., Saldivia, S., Kohn, R. and Torres, S. (2002). Chilean study on the prevalence of psychiatric disorders. *Rev Med Chil.*, **130**(5): 527-36.

Vining, E.P. (1999). Clinical efficacy of the ketogenic diet. *Epilepsy-Res,*. **37**(3): 181-90.

www.rcgp.org.uk/nccpc/docs/appendix% 20b.pdf.

Wada, K., Iwasa, H., Okada, M., Kwata, T., Murakami, T., Kamata, A., Zhu, G., Osanai, T., Kato, T. and Keneko, S. (2004). Marital status of patients with epilepsy with special reference to the influence of epileptic seizures on the patient’s marital life. *Epilepsia,* **45**(8): 33-6.

Wallace, S. J. (2001). Newer antiepileptic drugs: Advantages and disadvantages. *Brain–Dev.*, **23**(5): 277 – 83.

Wilner, A. (2005). Refractory epilepsy can be identified early. Neurology reviews. Com.

World Health Organisation (WHO) (2001). Mental and neurological disorder. Fact sheet NO 265.

WHO. (2001). *The World Health Report*. Mental health: New understanding, new hope.

WHO. (2004). Epilepsy: A manual for physician.

WHO. (2004). Epilepsy in the WHO African region. Bridging the gap. The Global campaign against epilepsy “Out of the shadow’.

WHO. (2005). Mental health. The bare facts.

Code Number: ___________

A P P E N D I X

Department of Pharmacology & Clinical Pharmacy

Ahmadu Bello University, Zaria

SEIZURE DISORDERS PROJECT: PATIENTS' DATA FORM

(To be completed from patients' folders)

June 2005

To whom it may concern:

This research data form relates primarily to drug and non-drug management of seizure disorders/epilepsy in Ahmadu Bello University Teaching Hospital Kaduna. The purpose of the study is to generate baseline data that would, in addition to contributing to literary scholarship, impact on improved care of seizure disorders/epileptic patients, not only in this hospital; but also beyond. All information obtained, either retrospectively or prospectively, will be treated confidentially and it will also not be used for any other purpose than that stated above. The relevant hospital authority is aware of this study and has given its approval. The data will be reported as a thesis for an M. Sc degree in Pharmacology and publication in academic journals/presentations.

Ms Aduke E. Omoniwa (M.Sc candidate)
Dr(Mrs) Helen O. Kwanashie, Dr J.A. Anuka and Dr Taiwo L. Sheikh (Supervisors)

PART I: GENERAL INFORMATION AND SOCIO-DEMOGRAPHIC DATA

1. Unit number: ___________________

2. Number of clinic attendance within the last 10 years

i.	2004 ______	v.	2000 ______	ix.	1996 ______
ii.	2003 ______	vi.	1999 ______	x.	1995 ______
iii.	2002 ______	vii.	1998 ______		
iv.	2001 ______	viii.	1997 ______		

3. Calculate frequency of visits – i.e. average number of clinic visits per year (using only years in which clinic was attended): _______

4. Sex i. Male ☐ ii. Female ☐ iii. Not recorded ☐

5. Age at first registration (in years) _______________

6. Availability of initial medical record (e.g. clerking).

i. Available ii. Not available

7. Age at first presentation/episode (in years)____________

8. Date of first registration: ____________. Therefore, calculate years since first registration.

____

9. Nationality: i. Nigerian ☐ ii. Non-Nigerian ☐ Specify country: _____________

10. Geopolitical zone (of origin, not of residence)

i. South West ☐
ii. South East ☐
iii. South South ☐
iv. North Central ☐
v. North East ☐
vi. North West ☐
vii. Not Applicable (i.e. if non-Nigerian) ☐

11. State of origin

i.	Abia ☐	xiv.	Enugu ☐	xxvii.	Ogun ☐
ii.	Adamawa ☐	xv.	Gombe ☐	xxviii.	Ondo ☐
iii.	Ak/Ibom ☐	xvi.	Imo ☐	xxix.	Osun ☐
iv.	Anambra ☐	xvii.	Jigawa ☐	xxx.	Oyo ☐
v.	Bauchi ☐	xviii.	Kaduna ☐	xxxi.	Plateau ☐
vi.	Bayelsa ☐	xix.	Kano ☐	xxxii.	Rivers ☐
vii.	Benue ☐	xx.	Katsina ☐	xxxiii.	Sokoto ☐
viii.	Borno ☐	xxi.	Kebbi ☐	xxxiv.	Taraba ☐
ix.	Cr/River ☐	xxii.	Kogi ☐	xxxv.	Yobe ☐
x.	Delta ☐	xxiii.	Kwara ☐	xxxvi.	Zamfara ☐
xi.	Ebonyi ☐	xxiv.	Lagos ☐	xxxvii.	FCT ☐
xii.	Edo ☐	xxv.	Nasarawa ☐	xxxviii.	Not Recd ☐
xiii.	Ekiti ☐	xxvi.	Niger ☐	xxxix.	N/A ☐

12. Local Government Area (LGA): ____

13. Place of Origin (Hometown) : ____

Code Number: ___________

14. Ethnicity / Tribe
    i. Hausa ☐   ii. Yoruba ☐   iii. Ibo ☐
    iv. Other ☐   Specify: _____________   v. Not recorded ☐

15. Religion

    i. Christianity ☐   ii. Catholic ☐   iii. Protestant ☐
    vi. Islam ☐   v. Traditional African Religion ☐
    vi. Other ☐   Specify _____________________________

16. Marital Status
    i. Single ☐   ii. Married ☐   iii. Separated ☐
    iv. Divorced ☐   v. Widowed ☐

17. For married men only: Number of wives
    i. 1 ☐   ii. 2 ☐   iii. 3 ☐   iv. 4 ☐
    v. > 4 ☐ Specify _______   vi. Not recorded ☐
    vii. Not applicable (i.e. single men, never married) ☐
    viii. Not applicable (Female) ☐

18. For married women only: Number of times married
    i. 1 ☐   ii.2 ☐   iii. 3 ☐   iv. > 4 ☐ Specify _________
    v. Not recorded☐   vi. Not applicable (i.e. single Women, never married) ☐
    vii .Not applicable (Male) ☐

Code Number: ___________

19. Number of Children
i. 1-2 ☐ ii. 3-4 ☐ iii. 5-6 ☐
iv. 7-8 ☐
v. > 8 ☐ Specify _______ vi. Unknown ☐
vi Not applicable(singles, never married).

20. Recorded next of kin

i. Father ☐
ii. Mother ☐
iii. Brother ☐
iv. Sister ☐
v. Husband ☐
vi. Wife ☐
vii. Uncle ☐
viii. Aunt ☐
ix. Son ☐
x. Daughter ☐
xi. Friend ☐
xii. Other ☐ Specify _____________
xiii. Not recorded ☐

21. Accompanying persons to clinic (indicate number of times in last 1-10 years)
i. Alone (Not accompanied) ☐ ____
ii. Father ☐ ______
iii. Mother ☐______
iv. Brother ☐______
v. Sister ☐______
vi. Husband ☐______
vii. Wife ☐______
viii. Uncle ☐______
ix. Aunt ☐______
x. Son ☐______
xi. Daughter ☐______
xii. Friend ☐______
xiii. Not recorded ☐
xiv. Other ☐______ Specify _____________

22. Calculate percent of times accompanied ______ %

23. Highest educational level attained
i. None (i.e. never been to school) ☐
ii. Koranic school ☐
iii. Pre primary school ☐
iv. Primary school uncompleted ☐

v. Primary school completed ☐
vi. Junior secondary school completed ☐
vii. Senior secondary school completed ☐
viii. Tertiary (i.e. higher education after secondary school) ☐
ix. Postgraduate ☐
x. Adult literacy ☐

Code Number: ___________

xi. Other ☐ Specify ____________________________
xi. Not recorded ☐

24. Main occupation
i. Farmer ☐ ii. Petty trader ☐
iii. Big business man / contractor ☐ iv. Driver / mechanic / artisan / carpenter ☐
v. Civil servant ☐ vi. Teacher / Lecturer ☐
vii. Policeman / Soldier / Navy ☐ viii.Health worker ☐
ix. Doctor ☐ x. Lawyer ☐
xi. Student ☐ xii .Child ☐
xiii House wife ☐ xiv Unemployed ☐
xv. Retiree ☐ xvi. Other Specify ______________
xvii. Unknown

25. Residence
i. Kaduna metropolis ☐ Specify area ____________________________
ii. Within Kaduna State ☐ Specify town ____________________________
iii. Outside Kaduna State ☐ Specify state ____________________________

PART II: MEDICAL HISTORY

26. Specific Diagnosis.
i. Epilepsy unspecified☐ ix Seizure disorder (unspecified) ☐
ii. Complex partial seizure☐ x. Seizure affective disorder☐
iii. Mixed seizure disorder☐ xi. Status epilepticus ☐
vi. Myoclonic seizure☐ xii. Simple partial seizure☐
v. Partial with 2^o generalised tonic-clonic seizure☐ xiii.Temporal lobe Epilepsy☐
vi Partial seizure unspecified☐ xiv.Generalized tonic-clonic ☐
vii. Post traumatic seizure ☐ xv Others ☐ specify________
viii.Psychomotor seizure☐

27. Risk factors
i. Febrile illness ii. Road traffic accident iii. Head injury
Vi .illness (unspecified) v.Birth defect
vi. Unknown
vii. Others, specify ___________________________ viii. Not recorded

28. Age at which above (Q 28) occurred.
i. Neonate ii.2-6 months iii. 7-11month
iv. 1-3 yrs
v. 4-6 yrs vi. 7-9 yrs vii.10-14 yrs viii. 15—19 yrs ix. 20-24 yrs x.25-29 yrs xi. 30-34 yrs
xii. 35-39 yrs
xiii. 40-45 yrs xiv.>45 yrs xv. Not applicable

29. Period/Time at which seizure/disorder is trigger or usually occurs.
i. Day☐ ii. Night☐ iii. Not specific (day or night) ☐ iv Others specify_________☐

30. Other alternative therapy patient had ever used
i None☐ ii. Traditional medicine☐ iii. Religious intervention☐ (deliverance) iv. Others ☐ specify_____ v. Not recorded☐

31. Frequency of seizure before first visit to hospital.
i. Only once ☐ ii. Only twice ☐
iii. Once a month ☐ iv. 2-3 times per month ☐
v. 4-7 times per month ☐ vi. 8-12 times per month☐
vii. 60-90 times per month☐ viii. Recurrent, not specific☐

ix. Not recorded☐

32. Duration (time interval) between first attack of seizure and medical intervention.
i. immediately after the attack ☐ ii .<6months ☐ iii. 6-8 months ☐
vi. 9-11 months ☐ v.1-2yrs vi. 3-4yrs ☐
vii. ≥5yrs ☐ viii. Not recorded ☐
ix specify___________________________

33 Frequency of seizure since attendance of clinic.
i. Twice per day ☐ ii. Twice per week ☐
iii. Once a month ☐ Vi. Twice a month ☐
v. 2-3 times a month☐ vi. Once in 2-3 months ☐
Vii. Once in 5-6 months ☐ viii. Once in 7-8months ☐
ix. Once in a year ☐ x. Once in 2 years ☐
xi. Others, specify _____________☐ xii. No seizures at all ☐

34. Family history of psychiatry disorders I: Disorder
i. Yes ☐ Specify disorder _____________________________
ii. No ☐
iii. Don't know ☐ vi Not recorded ☐

35. Family history of psychiatry disorders II: Relationship
i. Father ☐
ii. Mother ☐
iii. Sibling ☐
iv. Other ☐ Specify relationship _____________________________
v. Not applicable (i.e. 'No' to Q 23) ☐
vi. Not recorded ☐

36 Effect of seizure disorder on performance in school
i. Dropped out because of embarrassments☐
ii. Cannot go to school (formal education) ☐
iii. Slow learning☐
iv. Dropped out at primary school☐
v. Dropped out at secondary school☐
vi. Dropped out at tertiary school☐
vii. Not affected –doing well☐

Code Number: ___________

viii. Not recorded
ix. Not applicable (neither pupil nor student)

37 Effect of disorder on marital life
i. Divorced ii. Separation
iii.Remained single (i.e. unwilling to/or unable to marry due to disorder)
iii vi. Not recorded v. Not applicable (e.g children

39. Signs and symptoms which patient presented with

i. Abdominal fullness ☐
ii. Abrupt onset of impaired consciousness☐
iii. Aches and pains ☐
iv. Anxious mood / worries / pessimistic ☐
v. Brief shock like contraction of muscles ☐
vi. Blurred vision ☐
vii. Cessation of ongoing activities(lasting for 30 seconds) ☐
viii. Chest pain ☐
ix. Clonic jerking of left thumb☐
x. Clonic (relaxation of muscles) ☐
xi. Constipation ☐
xii. Depression ☐
xiii. Diarrhoea ☐
xiv. Dizziness ☐
xv. Dry mouth ☐
xvi. Dysmenorrhoea ☐
xvii. Excessive worries ☐
xviii. Faintness ☐
xix. Fear (non specific) ☐
xx. Fears (of darkness, being alone, strangers, etc) ☐
xxi. Headaches ☐
xxii. Lip smacking, hand wring ☐
xxiii. Impair consciousness lasting 30 seconds to 2 minutes ☐
xxiv. Impotence ☐
xxv. Insomnia ☐
xxvi. Intrusive thoughts ☐
xxvii. Loss of appetite ☐
xxviii. Loss of consciousness
xxix. Memory impairment ☐
xxx. Nausea / vomiting ☐
xxxi. Nightmares ☐
xxxii. Palpitations ☐
xxxiii. Paresthesia of consciousness☐
xxxiv. Preservation of consciousness ☐
xxxv. Poor concentration ☐
xxxvi. Pounding sounds in the head ☐
xxxvii. Psychosomatic complaints ☐
xxxviii. Restlessness ☐
xxxix. Ringing in the ear ☐
xl. Shortness of breadth ☐

xli. Suicidal tendency ☐
xlii. Staring ☐
xliii. Sweating ☐
xliv. Tachycardia ☐
xlv. Tension / cries easily ☐
xlvi. Tonic (sustained contraction) ☐
xlvii. Trembling ☐
xlviii. Urinary incontinence/lost of sphincter control ☐
xlix. Weakness ☐
l. Weight loss ☐

li. Foaming /salivation in the mouth ___________________
lii. ___________________
liii. ___________________
lv ___________________

lvi Not recorded

40. Co-morbid (i.e. co-existing) psychiatry conditions. State onset (in years, relative to 2004 = 1) in space provided, as appropriate.

i. Schizophrenia ☐ ____
ii. Depression ☐ ____
iii. Anxiety ☐ ____
iv. Dementia ☐ ____
v. Drug / substance abuse ☐ ____
vi. Others ☐ Specify ____ ____

41. Co-morbid (i.e. co-existing) non-psychiatry conditions. State onset (in years, relative to 2004 = 1) in space provided, as appropriate.

i. Malaria ☐ ____
ii. Typhoid fever ☐ ____
iii. Diabetes ☐ ____

iv. Hypertension ☐ ____
v. HIV / AIDS ☐ ____
vi. Tuberculosis ☐ ____
vii. Asthma ☐ ____
viii. Others ☐ Specify _____________________ ____

42. Some basic life style information

i. Physical exercise (sports) ☐
ii. Other recreational activities ☐
iii. Alcohol consumption ☐
iv. Tobacco use (e.g. cigarette smoking) ☐
v. Marijuana (Indian hemp) use ☐
vi. Other drug / substance abuse ☐
vii. Multiple sex partners ☐
viii. Other ☐ Specify ____________________________
xi. Unknown (not recorded) ☐

43. Number of times patient was hospitalized for psychiatry illnesses each year of the last 10 years (i.e. from 1995; leave blank if not hospitalized)

i. 2004 ______
ii. 2003 ______
iii. 2002 ______
iv. 2001 ______
v. 2000 ______
vi. 1999 ______
vii. 1998 ______
viii. 1997 ______
ix. 1996 ______
x. 1995 ______

44. Calculate average number of hospitalization per year ___________

45. Number of times patient was hospitalized for psychiatry illnesses in 2003 and 2004

i. 0 ______
ii. 1 ______
iii. 2 ______
iv. 3 ______
v. 4 ______
vi. 5 ______
vii. 6 ______
viii. Other ☐ Specify______

46. Total duration of psychiatry hospitalization in 2003 and 2004
i. < 1 week ☐
ii. 1 – 2 weeks ☐
iii. 3 – 4 weeks ☐
iv. 5 – 6 weeks ☐
v. 7 – 8 weeks ☐
vi. > 8 weeks ☐ Specify ____________________

47. Hospital in which patient was hospitalized in 2003 and 2004
i. ABUTH ☐
ii. Psychiatry Hospital, Barnawa ☐
iii. Other ☐ Specify ____________________________

48. Laboratory investigations carried out
i. Culture and Sensitivity ☐
ii. Electro encephalogram (EEG) ☐
iii. General haematological investigations ☐
iv. HIV Screening test ☐
v. Liver function test ☐
vi. Malaria parasite ☐
vii. Serum glucose ☐
viii. Serum urea electrolyte creatinine ☐
ix. Stool microscopy ☐
x. Urinalysis ☐
xi. Urine microscopy ☐
xii. WBC and differential ☐
xiii. Widal test ☐
xiv. Other ☐ Specify________
xv. None

49. Side effects experienced with drugs used

- i. Anemia ☐
- ii. Agitation ☐
- iii. Ataxia ☐
- iv. Behavioral change ☐
- v. Blurred vision ☐
- vi. Confusion ☐
- vii. Crawling sensation ☐
- viii. Depression ☐
- ix. Dizziness ☐
- x. Drowsiness ☐
- xi. Dry irritating cough ☐
- xii. Dryness of mouth ☐
- xiii. Emotional upset ☐
- xiv. Epigastric pain ☐
- xv. Excessive sleep ☐
- xvi. Forgetfulness ☐
- xvii. Headache ☐
- xviii. Increase in frequency of seizure ☐
- xix. Insomnia / poor sleep ☐
- xx. Irritability ☐
- xxi. Lethargy ☐
- xxii. Nasal congestion ☐
- xxiii. Nausea /vomiting ☐
- xxiv. Palpitations ☐
- xxv. Poor appetite ☐
- xxvi. Pruritus ☐
- xxvii. Restlessness ☐
- xxviii. Sensations in the ear ☐
- xxix. Skin rashes
- xxx. Tremors ☐
- xxxi. Upward rolling of eyes ☐
- xxxii. Weakness ☐
- xxxiii. Others Specify ☐ ________________ ________________ ________________

50. Management of side effects

- i. Discontinuation and use of alternative drugs ☐
- ii. Use of other drugs to remedy side effects ☐
- iii. Reduction in dosage ☐
- iv. Reduction in frequency of administration ☐
- v. Counseling ☐
- vi. None (i.e. no specified management)
- vii. Other ☐ Specify: ______________________

51. Reasons for drug discontinuation

- i. Side effects ☐
- ii. Therapeutic failure ☐
- iii. Non-compliance / adherence ☐
- iv. Prohibitive cost ☐
- v. Other ☐ Specify: ______________________

52. Assessment of drug compliance

i. Poor because of lack of funds ☐ ii. Poor because patient refused to take drugs ☐

iii. Poor but later improved (e.g. after counseling) ☐ iv. Fair ☐

v. Good ☐

53. Individualized pharmacy records for patient

i. Available ☐ ii. Not available ☐

54. Non-pharmacological management. Indicate number of sessions in the spaces provided.

- i. Electro-convulsive therapy (ECT) ☐ _______
- ii. Insight counselling ☐ _______
- iii. Counseling with family ☐ _______
- iv. Group counseling ☐ _______
- v. Psychotherapy ☐ _______
- vi. Cognitive behaviou therapy ☐ _______

vii. Other ☐ Specify: ____________________

55. Was the patient experiencing seizures while attending clinic
   i. Yes, because of poor compliance due to cost of drug(s)ǐ
   ii. Yes, due to febrile illnessǐ
   iii. Yes, even while on treatmentǐ
   iv. Yes, was triggered by exerciseǐ
   v. Yes, when patient misses or ran out of drugsǐ
   vi. Yes, at initial stage of therapy but fit stopped as treatment progressesǐ
   vii. No fit at allǐ
   viii. Others specify___________________

56. Abscondment within the last 10 years: indicate duration in months.

i. 2004 ______	v. 2000 ______	ix. 1996 ______
ii. 2003 ______	vi. 1999 ______	x. 1995 ______
iii. 2002 ______	vii. 1998 ______	
iv. 2001 ______	viii.1997 _____	

57. Reasons for abscondment

   i. lack of funds

ii. Patient feels better

   iii. Others specify_____________________ vi. Not

applicable

58. Relapse within the last 10 years: indicate number of times.

i. 2004 ______
ii. 2003 ______
iii. 2002 ______
iv. 2001 ______
v. 2000 ______
vi. 1999 ______
vii. 1998 ______
viii.1997 _____
ix. 1996 ______
x. 1995 ______

Code Number: ___________

59. Calculate number of relapses per year: ___________

60. Reason for relapse

i. Abscondment ii. Therapeutic failure ii. Prohibitive cost

Vi. Poor compliance v. Not applicable

61. Overall treatment outcome

i. Good ☐
ii. Satisfactory ☐
iii. Poor ☐

62. Additional notes

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This section below is to be filled in by person completing, or overseeing the completion of the questionnaire

Date questionnaire was completed: ___________________________________

Name and signature: ____