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THE ROLE OF COMPUTERIZED TOMOGRAPHY IN THE
DIAGNOSIS AND MANAGEMENT OF EPILEPSY

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DECLARATION

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This dissertation is my own original work and has not been presented for a degree in any other University.

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TERMINOLOGY AND ABBREVIATIONS

- AURA Is the first warning to the patient that an attack is beginning. The type of aura depends on where the attack originates from.
- CT Computerized tomography
- EEG Electroencephalogram. Is a recording using scalp electrodes of the changes of the electric potential of the brain.
- FDG Glucose Fluorodeoxyglucose.

 Glucose combined with fluorine (in F) to form a radionuclide

Functional deficit zone

An area of the brain that functions abnormally in the interictal period.

- GCS Generalized convulsive seizures
- MRI Magnetic Resonance Imaging
- MTS Mesial temporal sclerosis (Ammon's Horn, or Hippocampal sclerosis)
- PCS Partial complex seizures
- PET Positron emission tomography. Is a form of emission computed tomography.
- PSG Partial seizures secondarily generalized
- SPS Simple partial seizures
- SPECT Single photon emission computerized tomography
- °9|B TC HMPAO Technetium 99m Hexamethyl propylene amine oxime
- TLE Temporal lobe epilepsy
- ILAE International League Against Epilepsy.

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DEDICATION

To my beloved parents Mr. Jeremiah Ngobe and Mrs. Esther Wanjiru Ngobe.

SUMMARY

Epilepsy is a common neurological disorder in Kenya, affecting many people especially those below the age of 20 years. Thr> highest number of cases are concentrated below 10 years of age. Other age groups are also affected but to a lessor degree. All epileptic patients who underwent computerized tomography examinations of the brain at Kenyatta National Hospital for convulsive disorders since the installation of the Tomoscan CXQ machine on 11th November 1992 up to 31st July 1996 were retrospectively reviewed. A total of 484 patients were included in the study.

Computerized tomography (CT) was found to be of extreme value in screening and evaluation of seizure patients. In this study 56.4% of the total number of patients were found to have abnormal CT scan findings. CT scan examination is a guick, simple, non invasive method of evaluating patients with intracranial abnormalities. The diagnostic accuracy of CT scan examination is high. The commonest abnormal or positive finding was brain atrophy. Diffuse brain atrophy was more common than focal brain atrophy.

Intracranial tumours were the second commonest abnormal CT scan finding. These were all primary brain tumours.

The predominant type of brain tumours found in the study were gliomas with (55.3%). The main seizure pattern in these tumours was the generalized convulsive type.

The third commonest abnormal CT scan finding were cases of infections. Brain abscesses were the commonest abnormal CT scan finding among cases of infections, with a total of 58.6%.

Trauma cases were fourth in CT scan abnormal findings.

The role of CT scan in the dignosis of head injury patients presenting with convulsions is remarkable. This mode of investigation has virtually replaced cerebral angiography since no false negative or false positive results have been observed.

Other important abnormal CT scan findings were cases of hydrocephalus. Communicating type of hydrocephalus had the highest abnormal findings in this group. Cases of hydrocephalus and cerebral infarcts occupied the 5th position of the CT scan abnormal findings.

Patients with a short duration of seizures (less than 6 months) had the highest CT scan abnormal findings. Patients above 65 years of age also had a high number of abnormal CT scan findings.

The above findings correlated well with findings in other studies, except in the age group below 10 years as described in the text.

CHAPTER 1

INTRODUCTION

Epilepsy is one of the most common neurological disorder affecting mankind (39). It is thought more as a symptom than a disease, and is characterized by recurrent convulsions or seizures. The seizures stem from excessive nerve discharges within the brain and are accompanied by sudden disturbances of function of the body or mind (13) (39). A seizure js defined as a sudden paroxysmal electrical discharge of neurons within the cerebral cortex (49).

More than 2 million people in the United States of America (USA) had epilepsy in 1981. This translated to over \$ 3 billion a year in unemployment, underemployment, increased deaths, medical care services and research, according to the report by the United States Commission for the Control of Epilepsy and its consequences (39).

It was estimated that in 1982, about 700-1500 people per 100,000 suffered from epilepsy throughout the world, according to the then Deputy Director of World Health Organization (WHO), T.A. I.ambo (40).

The data that was available by then suggested a prevalence rate of 4 or 5 times in the developing countries of Africa, Asia

and Latin America compared to that of the industrialized countries (40). The high prevalence rate in the developing countries may be related to poor antenatal and maternal care, prematurity, birth injuries, infantile febrile convulsions, malnutrition, multiple infections and trauma (40,76).

In Kenya it is estimated that about 500,000 people suffer from epilepsy and yet only less than 20% of these patients are properly managed mostly because of ignorance and the stigma attached to this disease, according to Okello the former Director of Medical Services in Kenya (76).

Epilepsy is the second commonest presenting neurological condition at Kenyatta National Hospital after infections according to Kwasa (77) with an average annual prevalence rate of 201 cases while that of infection (all types of meningitis except tuberculous meningitis) has an average annual prevalence rate of 280 cases (77).

The value of computerized tomography (CT) of the brain in detecting structural abnormalities in epileptic patients is well documented (33,36,90).

Computerized tomography of the brain is of extreme value in the screening and definative evaluation of seizure patients (33,36,90).

The highest positive CT findings are in patients with focal seizures secondarily generalized (36). The older patients have a higher number of abnormal CT scans of the brain

particularly, if they are over 65 years of age (36). Patients with a short duration of seizures (less than 6 months) also have a high probability for CT scan abnormalities (33, 36). Individuals with positive neurological findings (pxclusive of mentation changes) show CT abnormalities of the brain (33). Computerized tomography of the brain is more accurate than radionuclide scanning in the detection of intracranial abnormalities (33).

Computerized tomography of the brain has been available in Kenyatta National Hospital since 11th November 1992, whpn a Philips Tomoscan CXQ a 3rd generation CT scanner machine was installed. Since then many patients with convulsive disorders have been scanned to check for any intracranial pathology. This study is therefore intended to look into the contribution of this important diagnostic modality in the diagnosis of patients with convulsive disorders, since its installation. The study will also examine the possibilities of establishing good criteria for selecting patients with epilepsy for CT scan examination of the brain. It is virtually impossible to scan every patient with epilepsy, due to the large number of patients referred for different types of CT scan examinations of the body (head, chest and abdomen) etc and also since the advent of cost sharing, this is also a significant factor.

LITERATURE REVIEW

History of Epilepsy

The word epilepsy is derived from the Greek word "Bfff/\HWA" "epilepsia" meaning to be seized or to be taken hold of or to be attacked. The idea of a disease seizing a man goes back to the old magic concept that all diseases were "attacks" or seizures by gods or demons. Epilepsy or epileptic attack was for many years regarded as the demonic disease par excellence. It acquired its name because it seized both the senses and the

Recorded reference to epilepsy date back to about 7000 ypars. Epilepsy was not however distinguished from psychiatric disease or other conditions producing a sudden loss of consciousness or odd behaviour. The ancient Greek culture provided the concept that epilepsy was due to a hereditary or acquired dysfunction of the brain (84).

Early theories of the origin of epilepsy were a mixture of magic and religious fantasy,. During the Biblical era epilepsy was ascribed to evil spirits and demon possession. Jesus at one stage is said to have cast out demons from a boy who had a falling sickness all his life (76). Often the early approaches to treatment did more harm than good. Hippocrates (»InPOtCPflTHZ") as early as 400 B.C. recognized that epilepsy was a disorder of the brain and that its origin was hereditary. He attacked the supernatural explanation as the cause of epilepsy. He recommended treatment with diet and drugs. Other methods used to treat epilepsy in the ancient world included phlebotomy, trephining and cauterization of the (to release pathologic humors), circumcision, castration. Galenic scholars subscribed to the philosophy of various humors in the body being responsible for health and Epilepsy was therefore thought to be a build up in the body of poisonous humors like plegm (76). These methods were used to treat the biological basis of epilepsy. supernatural belief that epilepsy was caused by demonic possession coexisted with the biological basis. supernatural belief prevailed in the European culture of the middle ages (84) .

From antiquity to the relatively recent past physicians preferred to refer to epilepsy as the "sacred disease" (morbus divinus). This was mainly due to the common belief that

diseases were phenomena more or less dependent on the supernatural and were considered to be divine, retribution for wickedness or as a consequence of possession by spirits. The scientific concept eventually and fortunately emerged victorious over the magic concept (38).

The life of the epileptic patient was sheer misery. The epileptic patients were regarded as unclean people and who ever touched them might become prey to the demon. They wprp viewed with fear, anxiety and even disgust. The unfortunate person who felt an epileptic attack coming (aura), rushed home or to a deserted place where he covered his head. No other disease has set individuals apart so far, so often and so long. The children with epilepsy were the most devastated by the disease, and were often confined with insane persons (38).

The concept of epilepsy as a supernatural disease was gradually replaced by an awareness of the biologic origin during the 1800s in Europe. The enlightment of the 18th century and the advancement in the science of neurology in the 19th century improved the knowledge and understanding of epilepsy. Demonic possession as the cause of epilepsy was therefore abandoned. Around this time in 1873 John Hughlings Jackson (Jacksonian unilateral epilepsy is named after him) produced abroad defination of epilepsy in London, which is still valid today, i.e. "Epilepsy is the name for occasional, sudden excessive and local discharges of cerebral grey matter". According to his defination there were many forms of epilepsy (38,84).

The turning point for the epileptic patient was due to the epoch making invention of the human electroencephalogram machine (EEG) by a German psychiatrist Hans Berger in 1924. This provided for the first time visual proof of Hughlings Jackson's theories i.e. "seizures of focal onset were due to

an abnormal electric discharge arising from localised area of the cortex and that specific clinical manifestations of individual seizures depend on the site of origin and spread(38,84).

An explosion of knowledge about epilepsy and EEG followed for the next two decades leading to the publication of the classic atlas of electroencephalography (EEG) in 1952 (38,84). A colleague of Hughlings Jackson performed the first surgical cortical resection for epilepsy in 1886, on a patient who suffered focal motor seizures due to post-traumatic scar. This was Victor Morsely who together with Hughlings Jackson provided ample proof that epilepsy indeed had nothing to do with demon possession or supernatural causes (84).

The role of temporal lobe in epileptogenesis was first appreciated during the 1930s and 1940s. This led to the development of the technique for anterior temporal lobectomy (84). The invention of computed tomography (CT) machine in 1972 (The EMI scanner) by Sir Godfrey Hounsfield revolutionized the study of neurological diseases including epilepsy. This was 78 years after the discovery of x-rays by Wilhelm Conrad Roentgen on 8th November 1895 (26,70,92).

In the 1980s, the development of cross-section neuroimaging techniques particularly the magnetic resonance imaging (MR!) ushered in the next phase in the surgical approach to epilepsy (84). Modern MR imaging will demonstrate preoperatively nearly every epileptogenic lesion seen on a standard microscopic pathologic specimen (84).

In Africa the history of epilepsy has not been well documented. Medical literature on epilepsy in Africa has been scarce until three and a half decades ago (76). That is not to say that epilepsy did not exist in Africa. The fact that

almost every tribe appears to have a name for the disease is proof enough that the disease has been in existence in Africa since time immemorial.

Previous papers dealing with epilepsy from the local scene were part of reviews of neurological diseases in East African hospitals. The earliest such review by Muwazi and Trowel 1 1940-1942 of neurological diseases among African natives of Uganda (at Mulago Hospital Kampala) recorded 48 cases of epilepsy out of a total of 727 patients with neurological diseases(2).

Shaper and Shaper (1957) in analysis of medical admissions at Mulago Hospital Kampala, Uganda found only 14 cases of epilepsy (3). Billington (1968) highlighted the problems of the epileptic patients in Uganda in a study of 28 epileptic patients carried out at Mulago Hospital in Uganda (11). Billinghurst (1970) did a study involving 85 epileptic patients at Mulago Hospital Kampala, Uganda (13). Another study also done in Uganda in 1970 was by Orley, who studied 83 cases of epileptics from rural areas (13).

From Tanzania Smartt (1959 and I960) studied demented and anti-social epileptics in a mental hospital in the Central Province of the former Tanganyika (4). Haddock (1965) in his review of neurological diseases in the former Tanganyika found that epilepsy was the fourth commonest neurological condition among inpatients (11). Jilek and Jilek (1970) in their study of the problem of epilepsy in a rural Tanzania tribe identified 201 patients with epilepsy out of a population of 10,000 Wapogoro tribesmen in the Mahenge region of Tanzania. This study by Jilek and Jilek reveals more than any other the deplorable life of the epileptic patients in Africa (13).

From Kenya Carothers (1953) wrote a World Health organization

(WHO), monograph on African mind in health and disease (11).

Ojiambo (1966) in a six month retrospective study on neurological diseases at Kenyatta National Hospital, Nairobi, found only one case of epilepsy (8).

Aloo (1977) in his dissertation for his Master of Medicine degree described the pattern of epilepsy admissions at Kenyatta National Hospital, Nairobi (31). Perhaps the epileptic outcast described by Giel (1968) in Ethiopia clearly reveals the horrifying life of patients with epilepsy. Giel describes how the epileptic patients were concentrated in cemeteries and were feared and even ignored by the medical profession. Compared to epileptic patients the lepromatous patients had a better deal, although they too were concentrated around cemeteries and the church yards (10).

From other parts of Africa various scientific papers were written by Levy, Forbes and Parirenyatwa (1964) on epilepsy in Central Africa (11) and by Hurst, Reef and Sachs (1961) on their observations on epileptic patients in South Africa (11). Dada and Odeku (1966) did a full investigation of 234 patients with epilepsy in Nigeria (11). Dada alone (1970) did an epidemiological study on epilepsy involving 117 patients in Lagos, Nigeria (13). Osuntokun (1969) did a retrospective study on febrile convulsive disorders involving 155 children at Ibadan, Nigeria (12).

Osuntokun and Odeku (1970) did a study involving 522 epileptic patients regarding the problems encountered in management and treatment of epilepsy. This was a long study from 1957 to 1968 and was carried out at the University College Hospital, Ibadan, in Nigeria (13). Mundy - Castle (1970) did an impressive study on epilepsy and electro encephalography (EEG) in Ghana. This study involved 583 epileptic patients (13).

From Rhodesia (now Zimbabwe), Zambia and Malawi, Levy (1970) did a study on the incidence and aetiology of epilepsy amongst Africans. This study involved 161 patients with symptomatic epilepsy and 665 patients with idiopathic epilepsy (11).

There was proliferation of scientific papers regarding epilepsy in Africa especially in the 1960s and 1970s. From the above studies traditional beliefs are a predominant factor.

TRADITIONAL DELIEF3 AND MANAGEMENT OF EPILEPSY

Various authors writing about epilepsy in Africa reveal similarities with the European beliefs of the middle ages, the 18th and 19th centuries about the supernatural and even demonic possession due to epilepsy. (84). In Africa the belief as to the causation and perpetuation of epilepsy vary from region to region. In Nigeria it is thought in some communities to be a sign of the visitation by the gods or demons. In others it is thought to be an infectious disease, that is acquired by coming into contact with the saliva of the sufferer during an attack (76). This later belief is fairly widespread throughout Africa (76). In Senegal people with epilepsy are thought to be spiritually holy men (76).

In Kenya certain communities regard it as a necessary prerequisite and accompaniment to successful and genuine practice of traditional medicine especially that which is inherited from parents or uncles (76). In other parts of Kenya it is considered to be a sign of demon possession. In other areas, it is thought to be a sign of punishment for sin committed by the sufferer from his long departed fathers and forefathers (76).

Smartt in 1959 writing about epilepsy in Tanzania mentioned that most people in the villages thought that epilepsy was caused by bewitchment or possession by the devils. The people

in the villages feared revealing the presence of epilepsy in their communities (4). Most epileptics were therefore treated with traditional medicines and only when they became demented or a danger to others were they sent for treatment in a modern hospital. He divided the patients into two groups. The demented group and the antisocial group.

The demented group of patients were said to show a progressive dementia often with catatonic symptoms, stupor and frequent fits. The other group of epileptic patients showed antisocial behaviour, and had fairly infrequent mental symptoms between fits (4). Periods of confusion lasting hours or days occurred after fits in the antisocial groups, with excitement, aggressiveness, noisy and violent behaviour (4). The antisocial group was also described by Carothers in 1947 in Kenya as having frenzied anxiety possibly due to post-ictal confusion i.e. Psychomotor epilepsy (4). The patient always denied all memory of the period of frenzy (4).

Jilek and Jilek in 1970 who treated epilepsy patients for 7 years beginning in 1961 from the Wapogoro tribesmen of the Mahenge region in Tanzania wrote that epilepsy was a dreaded disease by these people. The two authors further mentioned that the Wapogoro people were alarmed by the perceived the epileptic condition among their of The infants of these tribesmen manifested with febrile convulsions. There was a strong genetic basis of epilepsy with many members of the family affected. unusual frequency of epileptic manifestations in the Wapogoro tribesmen, who are said bo be highly endogamous encouraging marital union within the kin group, even between first The tribesmen had a high infant mortality and cousins. morbidity rate in families with two epileptic patients (13).

Medicine men of this tribe often specialized in the treatment of the disease "Kifafa" in "Kiswahili" using herbal remedies, some which were found to have anti-convulsive properties in experimental animals (13).

The authors mention that burns were a common feature amongst the epileptic tribesmen, many of whom fell into domestic fires, around which the family members gathered in the evenings. The burns could be lethal as the convulsing patient was looked at as highly contagious particularly foaming saliva from his mouth. The authors mention that it is a known fact though very sad, that no one would dare pull the patient from the fire for fear of contracting the disease and this is enough proof of how dreaded epilepsy is amongst the Wapogoro tribe (13). The epileptic patients among the Wapogoro tribe lead an ostracized existence similar to that reported by Giel in 1968 in Ethiopia (10). Discrimination against epileptic patients amongst the Wapogoro tribesmen is probably more marked than against the lepers, reported by Giel in Ethiopia, because the Wapogoro community views epileptics as contagious and demon possessed (10,13).

In Ethiopia traditional medicine men are called "debtera" and try to treat the evil spirit said to seize an epileptic patient. The belief is that the spirit so treated looks for someone else (10).

In Uganda epilepsy is known as "Ensimbu" in Buganda. the epileptic child is thought to be born with a lizard in its brain and the lizard grows as the child grows. When the lizard moves the child falls in an epileptic fit. The traditional Baganda doctor would then shave the child's head and apply a horn to suck out the lizard (11). Two types of epilepsies are recognised traditionally. "Ensimbu" with no aura where patients fall into water or fire and "Ensimbu

enzimire" with an aura and occurs at the time of full moon, and new moon (11).

In Nigeria Osuntokun in 1969 mentioned that Nigerians hardly ever admit that any member of their family suffers from any illness which may carry a social stigma, such as epilepsy, or mental illness (12). There is a high mortality rate due to febrile convulsions in Nigerian children, approximately 29% (12). The principle factor responsible for the high mortality rate is the administration of a concoction called "the grandmothers' native remedy", for all convulsive disorders. principle ingredient of the remedy is cow's urine concoction which contains a high concentration of hippuric acid (12). It also contains juices of several local herbs, fresh tobacco juice, a variable concentration of sometimes kerosene and occasionally sloan's liniment and shea butter (12). It is given orally and also rubbed on the skin after a seizure. This remedy causes prolonged hypoglycaemia and vaso-constriction. The hypoglycaemia is responsible for the high mortality rate in children with febrile convulsions (12,13). Other side effects of this concoction are choreoathetosis and cortical blindness, deafness, brain damage severe spasticity and an odour lasting for several hours aftpr its administration (12,13). Dada in 1970 mentioned that patients with epilepsy often present themselves late at modern medical centres in Nigeria. This is due to the strong bplief that epilepsy is caused by evil spirits or "Juju", black magic (13). The traditional doctors or native doctors are believed to be the only ones who can unravel the cause of epilepsy, and therefore treat it (4,13). Dada in 1970 noted that any person with epilepsy was ejected even from the household and was completely ostracised because epilepsy is thought to infectious. Known epileptics do not find it easy to get accommodation (13).

West African pepper (Alligator pepper or Xylopia aromatica) is rubbed into the eyes as part of the resuscitation act following an epileptic fit in Nigeria. The pepper causes a complication of conjunctivitis (13).

Osuntokun and Odeku in 1970 mentioned that there are suicidal tendencies amongst epileptic patients due to the social disgrace imposed by epilepsy. The two authors further mentioned the consequences of epilepsy as contributing to the loss of education, loss of professional careers, divorce due to nocturnal enuresis following fits, mental retardation, psychopaths with destructive tendencies not related to drug dementia (13).

Nigerians therefore consider epilepsy as one of the worst disasters that can befall anyone. They also believe that epilepsy can only afflict the possessed and it is infectious and that it can be transmitted though saliva. Therefore, there is no attempt to assist an epileptic patient having a fit (13,61).

In a research paper on doctors perspective on epilepsy in a developing country Danesi in 1988 at Lagos University Teaching Hospital, Nigeria showed that doctors in Nigeria admit having poor knowledge about epilepsy. There is inadequate instruction about epilepsy. Doctors' attitude epilepsy was more positive compared to that of the general population. However doctors were found to harbour some against epileptic patients probably out of (prejudice) ignorance (61). Danesi therefore proposed the adoption of a better approach to the teaching of epilepsy at undergraduate and post-graduate levels, and continuing education on epilepsy for practising doctors (13,61).

Out of 155 doctors interviewed by Danesi, the majority felt

that epileptics were less reliable at work, and 31.2* confirmed that they would not employ epileptics if they were employers. About 89% supported marriage for epileptics, and 47.5% agreed that they would object to their relatives marrying epileptics (61). These attitudes clearly show that most doctors lacked interest in epileptic patients and their problems (61).

Levy in his study on epileptic patients in Rhodesia now Zimbabwe, Zambia and Malawi in 1969 found that epilepsy was a common disorder in the three countries (13). Me also found out that burns sustained during epileptic fits were common in the cold months. The burns were sustained by both adult - and children (13). Levy mentioned that epileptic - patients are over burdened by misfortunes from mechanical accidents to ostracization by the local population (13).

Levy continued to mention that the traditional healer "Mganga" or local herbalist is revered by the local people just as much as a general practitioner is respected by his patients in the western countries. The "mganga" has not only medical and healing function but he also has a spiritual one The traditional healer combines organic medicine as well. with some religious psychotherapy (13). Respect for the ancestors is a very important issue in the lives of many local people. Diseases are interpreted by the local people as displeasure of an ancestor though frequently the living may be equally guilty of casting a "spell". Some of the "Wagangas" led themselves into antisocial activity. Other "Wagangas" will try to break the spell so cast. The Wagangas are thought to be a combination of a village doctor, parish priest, and mystic power, and stand very close to the people (13).

The consequence of these traditional healers is that most children were treated by them first. They were only taken to

modern doctors with formal medical education after the traditional treatment failed. All branches of medicine wore affected by this situation. Virtually all patients had scratch marks from the traditional healers, which is the hall marks of the waganga. Ordinary people believed that European Medicine cannot cure epilepsy and so there was no point of going for modern European treatment, because once the medicine were stopped the epileptic attacks came back (13). The traditional healers openly laid claim to being able to cure epilepsy. Even Levy himself agreed to have referred patients to the Waganga from time to time to satisfy the traditional belief that the anger of the spirit of the ancestors was the cause of the epilepsy (13).

There was therefore need to satisfy the psychological belief in the spirit of the ancestors by which epilepsy is interpreted in mystic terms (13). From 130 epileptic patients in the Semokwe region of Zimbabwe, 77 patients believed that they were bewitched by either spirits of their ancestors or by fairies, and 17 of these 77 believed that they were bewitched by some living persons who dislikedthem (13).

Only 10 patients of the 130 believed that epilepsy was "Gods" disease. The remaining 29 patients had no idea what caused their epileptic attacks. One patient who had fallen from a bicycle believed that it happened because he was bewitched (13). another patient had no money to go to a "Mganga" to find the cause of his fits. From the 130 patients only one had gone to seek help from hospital. Of the remaining 129 all 118 patients had received treatment from "Waganga". The treatment from the "Waganga" consisted of portions and inhalations made from such ingredients as fowls faeces, doves nests and the bones of birds e.g. "Go away birds". Some patients had received treatment from as many as 12 "wagangas" (13). Most patients on modern treatment e.g. with

phenobarbitone for about a month expected to be completely cured after that short duration. Relapses were therefore high because patients stopped treatment after a short time on their own volition. They therefore lost faith in their modern medication. Other patients had problems of getting their medication and therefore had relapses of epileptic fits. They too lost faith in their medication (13).

Billinghurst in 1970 mentioned that patients seen at the Mulago Neurological Clinic in Uganda included patients who were moderately sophisticated and educated and who were ambivalent towards the old ideas about epilepsy and had a willingness to stick to long term therapy, benefited from modern therapy with phenobarbitone (14).

Cardozo and Patel in 1976 in Zambia noted that there was a higher incidence of epilepsy in people aged about 20 years because epilepsy in Zambian children is often tolerated by their parents and is not brought to medical attention. In most cases the first attempts at treatment are by traditional healers, hence there is a considerable lapse of time before such cases are finally brought to hospital (29). Epileptic patients are discriminated against in the villages. Cardozo and Patel further mentioned in their study that Zambians most often attribute epilepsy to possession by spirits and in villages people strongly believe that this type of disease is best treated by traditional healers. In addition to his healing capacity the traditional healer is reputed to have spiritual powers concerned with appeasing the spirits and ancestors and nullifying the spells (29).

Medical clinics tend to see patients late, after the first attack (average 3-4 years) (29). At presentation considerable brain damage and even personality disorders may have occurred, in some cases by this time.

Hicks in 198R in a research paper on injury and epilepsy wrote that epileptic patients have to face the following problems:-

- (a) Physical injury e.g. falling to the ground against objects or into a fire in rural settings.
- (b) They are excluded from driving dangerous machines or moving machinery. In some countries if fits are well controlled for 2-3 years and the patient has had no fit he may be allowed to drive a private vehicle.
- (c) The epileptic patient faces social and emotional problem?: such as depression and marriage difficulties. The disease is still regarded as a personal stigma even though these disabilities are lessened with modern drugs for controlling fits (59).

Aluoch in 1992 in an article on the problems of the management of epilepsy in a developing country wrote that traditional belief had dominated the management of epilepsy in Africa with many communities believing that the condition is due to bewitchment or devil possession. Almost every tribe has a name for the disease unlike many other medical conditions. Many traditional healers in Africa have made their reputation from skills in "curing" the disease, although it is well known that their treatments have not particularly been successful. The range of treatment modes were as varied as the medicinemen themselves (76).

Many patients go to great lengths to keep epilepsy a secret. This is of course a futile attempt to hide what is impossible to hide. Sooner or later the condition is discovered. The discovery that one is an apileptic is of course a most shattering experience. The patient feels that all or everything is lost. The epileptic patient is confronted by many problems. The great problem confronting epileptic patients is of course the effect of the basic pathological lesion on intelligence or personality. The epileptic patient may be overwhelmed by superstition and fear (76). Prejudice

continues to be very powerful in the life of the epileptic patient both in terms of self image and in the eyes of others. In the developing world all these factors compound the problem of managing the patient. It is not surprising therefore the medication often fails (76). Several factors contribute to the treatment failure:-

- (1) The patient might not accept the diagnosis,
- (2) The patient fails to take the prescribed medications, on a regular basis. He might feel better when not taking the medication or he might not like the medication.
- (3) Regular medication may not be available and if available may be very expensive.
- (4) He does not like the idea.of taking the medication for as long as he lives (76).

Every community has its own stigma regarding epilepsy. It is well known that epilepsy is a word that has struck terror into the hearts of countless millions throughout the ages all over the world. Many people are struck with fear and apprehension when confronted by a patient suffering from epilepsy ev^n in this enlightened age (76).

In a research paper on the social cultural environment in the management of epilepsy in Kenya in 1995 Waruinge et al mentioned that lack of knowledge about epilepsy is rife. About 39.06% of the people covered by the study who included secondary school students, medical training colleges, university graduates, college graduates from teacher training colleges, teachers and technical college students, a total of 2500 respondents showed a glaring lack of knowledge about epilepsy. (95)

It is surprising that a large number of the respondents had a high degree of misconceptions about epilepsy. The largest number believed that epilepsy is an inheritable disorder (67.3%). Those who thought that epilepsy can be transmitted

through contamination with the patients saliva during an epileptic fit were 25.5%, contamination with the patients urine were 24.9%, farting 15.5%, through contamination with a patient's stool 8.8%, through public toilets 8.6%, through insect bites 6.7%, through curses 3.6%, through witchcraft 3.4% and through handshake 2.1% (95). Egually astounding is that out of 1,946 respondents 39.2% believed that there is no known type of treatment, while 32.5% had no knowledge of treatment. Of those who knew of at least one form of treatment (28.3%) about 66.2% knew of medical treatment, and 29.3% knew of combined herbal and traditional treatment. Another 3.2% knew of spiritual treatment and 1.2% knew of combined herbal and spiritual treatment. (95)

The study clearly illustrates a very negative attitude towards epilepsy. This explains why epileptic patients are deserted by many people during fits (31,95). These misconceptions are shared even by the well educated in our society unfortunately (31, 76, 95) and worse still even by the doctors (61, 76).

In most developing countries the medical practitioners have mixed attitudes towards epilepsy. Some doctors have a negative attitude towards epileptic patients out of sheer ignorance (61,76). This emphasises the need for continuing education on epilepsy for most doctors as well revolutionizing the teaching of epilepsy in medical schools. Lack of understanding by doctors may lead to poor results in the management of epilepsy. Any treatment failure, initial or long term may lead to poor results and also contribute to negative attitudes about epilepsy. The proper diagnosis of epilepsy especially the identification of the type is guite often missed by doctors. This can lead to erroneous treatment with disastrous results. The choice of the correct therapy much depends on the seizure type, efficacy and availability of the drugs, and patients compliance (61, 76).

CHAPTER 2

EPIDEMIOLOGY OF EPILEPSY

Incidence and Prevalence

The incidence and prevalence studies of epilepsy have been reported from many countries. Comparisons are often difficult because investigators have adopted different definations of epilepsy, case ascertainment methods, and classification schemes. Selection bias is also important.

Most studies have found incidence rates of 20-70/100,000 per year (range 11-134/100,000 per year) (49,67). These studies have prevalence rates of 4-10/1000 in the general population (range 1.5-30/1000) (67).'

The incidence varies considerably with age. The incidence rate is highest in early childhood, and falls (i.e. reach a nadir) in early adult life, a prevalency rate of 0.5-1.0% of the population, with 70-80% of cases beginning in childhood. About 0.5% of neonates have seizures and another 3% of children have febrile seizures (49). Prevalent rates show a similar but less pronounced age - related pattern. studies show a preponderance of epilepsy among males (11,12,13,29,49,67). The lifetime (or total) prevalence is a measure of the number of people in a population who have ever had epilepsy. Estimates vary from 2 to 5% (the latter according to the World Health Organization) of the population. Perhaps as many as 1 in 20 of the population will have had an epileptic seizure at some point in their lives and (at a conservative estimate) 1 in 200 will have epilepsy (49) (67). Put in another way about 2-5% of people in a general population will suffer at least one epileptic seizure in a life time. These will probably recur in over '/? (49). some people the remission of epilepsy is permanent. general remissions tend to occur early after the onset of

seizures. The longer the seizures continue the poorer the prognosis. Once a substantial remission has been achieved subsequent relapse is relatively unusual (49).

It is therefore noteworthy that epilepsy is the most prevalent serious neurological condition in the world. The prevalence rate is 10 times that of multiple sclerosis and 100 times that of motor neuron disease. These epilepsy figures exclude febrile convulsions, which occur in about 5% of children (67).

The underlying cause of seizure is an important factor for prognosis. Only 3% of persons with chronic epilepsy will require institutional care usually due to associated mental or neurological handicap rather than epilepsy itself Aetiological difference in general have only a moderate effect on prevalence rates, but higher rates in Latin America may reflect high local prevalence of neurocysticercosis (67,R2). Anybody can have seizures given an opportunity stimulus and Epilepsy has no sex or race preference. situation (76). is found in all continents and in all the people. distinguishing feature is the level of medical care delivery and development in various countries. In the third world, with less well developed primary and other health care facilities, the prevalence rates tend to be higher. In Africa the prevalency rates are higher in the rural areas as compared to the urban areas (76).

All social classes of people suffer from epilepsy including prominent personalities. It cuts across the social divide afflicting the rich and mighty as well as the poor and lowly. Some of the well known personalities who were either known or thought to suffer from epilepsy included Julius Caesar, Alexander the Great, Gautama Buddha, Alfred Nobel, St Paul and Napoleon Bonaparte. These were powerful men during their lifetime. One can therefore appreciate that having epilepsy

does not make it impossible for epileptics to achieve greatness in any area of human endeavour especially in our enlightened age (76).

Worldwide the average prevalency rate of epilepsy is about 4/1000 (67,76). The lowest prevalence rate is found in Japan 1.5/1000. The highest prevalence rate is found in United States of America 6.5/1000, population. (67,76) see table 1. Table Is Epilepsy prevalence rates for the developed world

Authors	Year of study	Country	Prevalence per 1000
SATO	1964	JAPAN	1.5
BORIS et al	1966	ENGLAND	5.5
GLASER 1966		U.S.A.	5.0
ANNEGERA AND MAUSER	1977	U.S.A.	6.5

Table 2: Epilepsy prevalence rates for the developing (excluding Africa) (76).

Authors	Year of study	Country	Prevalence per 1000
GOME et al	1978	COLOMBIA- URBAN	19.5
		COLOMBIA- RURAL	26.0
BARUCHA	1988	INDIA- URBAN	4.7

Table 3: Epilepsy prevalence rates for Africa - adopted from (76) with additions from (11,13,95).

	(76) With	additions fr	om (11, 13,	93).
Authors	Year of study	Country	Preva- lence per 1000	Remarks
Levy et al	1964	Central Africa Semokwe Reserve (Zimbabwe)	7.3	Not representative majority were women and children.
Haddock	1968	Accra- Ghana	3.3	Very selective sample
Dada	1968	Lagos- Nigeria	3.1	Small urban sample
Dada	1970	Lagos- Nigeria	13-15	Lower level of true incidence was an extrapolated figure of 8-13.
Jilek and Jilek	1970	Tanzania (wapogoro tribesmen)	20	Using old census figures 1959)
Orly	1970	Rural Uganda	2.1	Excluded febrile convulsions
Gerrits	1983	Liberia (Basasa and Kipelle tribesmen)	50 -	
Feksi, et al	1988	Nakuru- Kenya	18.5	
Ndungu and Waruingi	1995	Country- wide - Kenya (7 districts)	16.7	Element of possible degree of non acceptance of epilepsy in oneself.

The peak age for epileptic sufferers is between 10-19 years (11,13). Of all epileptic sufferers in Kenya 75% are below the age of 25 years old (76).

The higher prevalence rate of epilepsy in the third world compared to the developed world can be explained on the basis of a higher prevalence rate of preventable causes of epilepsy in the developing world. Such preventable causes of epilepsy are perinatal injury and trauma, perinatal infections, childhood immunizable diseases, road traffic accidents etc. (76).

CHARACTERISTICS OF EPILEPSY IN THE GENERAL POPULATION Widely different distributions of seizure types have been reported in population studies of epilepsy, but this variability probably reflects differences in methods of evaluation. Many studies have found generalized seizures to be the most common type (11,13,14,29,53,67,76,77).

Researchers with rigorous neurological inquiry have however, shown that complex partial and secondary generalized seizures account for about 60% of prevalence cases. Primary generalized tonic-clonic seizures may account for about 30%. Generalized absence (petit mal) and myoclonus may account for less than 5% (67).

The different seizure types have different relative frequencies. In the United Kingdom (UK) the National General Practice Study of Epilepsy (NGPSE) started in 1985 and it is a prospective cohort population based study in which epidemiological and natural history of epilepsy data are being collected in the unselected population and is an ongoing long term research found the index attack (the attack initiating diagnosis) to be 36% for a secondary generalized seizure, 33% for a primary generalized tonic-clonic seizure, 16% for a complex partial seizure, 4% for a simple partial seizure. 1% for an absence seizure and 1% for myoclonus seizure.

In Nigeria Danesi and Oni in 1983 found out that the commonest type of epilepsy was partial epilepsy with complex

symptomatology. The partial epileptic seizures had onset before the age of 20 years in 76.1% of patients. These kind of seizures were very frequent and occurred daily in 25.2% of patients. They usually evolved to secondary generalized tonic-clonic seizures in 73.8%. The commonest precipitating factor was sleep. The commonest actiological factor was childhood febrile convulsions followed by head injury. In 61% of cases there were no detectable aetiological factors. Of these patients 78% had positive EEG changes (76).

Partial seizures are more likely to have underlying structural problems such as brain tumours or scars. They are also more difficult to control (76). In Kenya epilepsy is much stigmatized and a poorly understood condition. Most of the studies done have been hospital based and are not a true reflection of epilepsy in the community (76).

In 1988 Feksi et al carried out a community based study in Nakuru (Kenya) and found a prevalent rate of (18.5) 18/1000. In their study Feksi et all found out that upto 75% of their patients mostly young adults who have had seizures for a long time had never visited any modern health facility on account of their disorder for diagnosis and treatment (76).

In 1995 Ndungu and Waruingi did a nationwide epilepsy education programme (EEP) to schools and colleges. The institutions were randomly selected, from seven districts in Kenya, which are geographically far flung and inhabited by people of different ethnic backgrounds. They reached about 20,000 people including teachers, lecturers and high school students. Their study had a prevalency rate of 16.7/1000 which compares well with the figure reached by Feksi et al 18/1000 in Nakuru. Ndungu and Waruingi acknowledge that complete denial of epilepsy in self is common, because of the social stigma (95).

Kwasa in a 10 year study from January 1978 to December 1987 found out that epilepsy was the 2nd commonest presenting neurological condition with an annual prevalency rate of 201 patients i.e. Epilepsy prevalency rate (16.6%) was steady in the 10 year period with neither rise nor fall. The mortality rate from epilepsy was fairly low at an average rate of 2% in the 10 year period (77).

The different seizure types have different relative frequencies. In Kenya Telang and Hettiaratch in an analysis of 115 epileptic cases in 1981 found out that grand mal type of epilepsy is the commonest at Kenyatta National Hospital (37).

In other parts of Africa Grand mal epilepsy has been found to be the commonest type of epilepsy by various researchers (Table 4).

Table 4: Grand-mal epilepsy prevalency rate for African countries (11,13,14,29,53,76)

Authors	Year of study	Country	Percentage		
Billington	1968	Kampala - Uganda	85.5%		
Billinghurst	1970	Kamplala - Uganda	85.0%		
Orley	1970	Rural - Uganda	86.0%		
Dada	1970	Lagos - Nigeria	51.0%		
Osuntokun and Odeku	1970	Ibadan - Nigeria	71.0%		
Edoo and Haddock	1970	Accra - Ghana	79%		
Levy	1970	i) Harare - Zimbabwe (hospital group)	86%		
		ii) Semokwe District	91%		
		iii) Meadowlands	80%		
		iv) European group (Harare)	57%		
Cardozo and Patel	1976	Lusaka - Zambia	79%		
Matuja	1987	Dar-es-salaam - Tanzania	61.3%		

Danesi and Oni from 1979-1982 did a clinical and EEG study of 282 cases of partial epilepsy in Lagos, Nigeria. They found a very high prevalency rate of partial epilepsy with complex symptomalogy. The onset of this seizure type was before the age of 20 years in 76.1% of patients. The seizures were frequent occurring daily in 25.2% of patients, and usually evolved to generalized tonic clonic seizures in 73.8%. The commonest aetiological factor for this seizure type was childhood febrile convulsions, followed by head injury. The commonest precipitating factor was sleep (45,65,76).

Matuja (1989) in Dar-es-salaam, Tanzania also found a high percentage of partial epilepsies 69% (65). Patients with partial epilepsy usually have identifiable aetiological factors than those with generalized epilepsies (65).

Danesi and Oni found out that partial epilepsy with elementary symptoms had focal discharge from the frontal and parietal regions (Jacksonian epilepsy). This seizure type had a high percentage (67.5%) of positive EEG changes. Partial epilepsy with complex symptoms and secondary generalized epilepsy (Temporal lobe epilepsy) had positive EEG changes in 78%. These patients have focal left temporal and bitemporal discharges (45).

Right temporal focal discharges were very common in all patients, especially those with partial epilepsy with elementary symptomatology (Jacksonian epilepsy. This is consistent with the suggestion that some discharges are often found at some distance from the presumed area of origin "travelled spikes" (45). Travelled spikes do not identify the site of primary excitation and their presence provide a good argument against the use of EEG criteria alone (without clinical criteria) to classify epilepsy (45).

A relatively higher percentage of patients with Jacksonian epilepsy (partial epilepsy with elementary symptoms) had normal EEG changes compared to those with temporal lobe epilepsy (partial seizures with complex symptoms). This observation is in agreement with the observation that typical Jacksonian epilepsy frequently show no specific EEG changes, presumably because the focus is small and buried in the sulcus (Danesi (13).

Age is an important determinant of incidence rates in epilepsy. The 1st year and the 1st decade of life have an

especially high incidence rate. This gives the first peak in a graph of incidence rates against age. The 2nd peak occurs in later life (49, 67).

Both prevalency and incidence rates vary for different types of seizures. Partial epilepsy account for about 1/3 of all new cases. The prognosis is poorer, they have a high prevalence rate and present more difficult management problems (49).

Complex partial seizures are more commoner than simple partial seizures in most populations. Of the generalized seizures tonic-chronic (grand mal) seizures are the most common (49).

Of the generalized absence seizures (petit mal) are relatively rare accounting perhaps for about 1% of all epilepsies (49). Clonic seizures are very rare. The incidence and prevalence rates also vary considerably with aetiology. Overall mortality rates for persons with epilepsy are probably 2-4 times of age and sex-matched controls. This excess mortality is greatest amongst young adult subjects. The reasons proposed for this are varied e.g.: (49)

- 1. The conditions underlying the epilepsy may cause death.
- 2. Accidental death may occur because of a seizure.
- 3. The epileptic seizure itself may directly result in death.
- 4. Psychosocial implications of epilepsy may carry their own mortality.

The commonest single cause of death in patients with epilepsy are death during seizures and suicide (49).

Table 5: Age specific incidence rates of epilepsy (adopted from 49).

Age in years	Annual incidence/1000 persons
4	3.55
5-9	0.53
10-14	0.70
15-24	0.85
25-34	0.13
35-44	0.30
45-64	0.26
65 +	0.60

Excluding single seizures derived from Crombie et al 1960. Adopted from Diseases of the nervous system Vol.11. Asbury Arthur 1986.

CLASSIFICATION OF EPILEPTIC SEIZURES AND EPILEPTIC SYNDROMES

Epilepsy is a "symptom" not a disease and may occur in many different clinical settings, and may take a number of different forms (49).

A seizure is a discrete clinical event (distinct, separate) Epilepsy is a condition characterized by recurrent unprovoked seizures (84). All patients with epilepsy have seizures (84). However, all individuals who experience a seizure do not necessarily have epilepsy. An important distinction is made between epileptic syndromes and epileptic seizures (84). Seizures that occur in short term direct response to an acute event resulting in brain insult are termed "acute provoked seizures". Patients with such seizures are not considered to have epilepsy. Acute febrile convulsions in childhood are the most common example of acute provoked seizures (84). Other examples are seizures that occur in response to acute head trauma, acute stroke or alcohol withdrawal. Conversely patients who experience recurrent seizures over months or years are considered to have epilepsy (84). Patients who experience a single (provoked or unprovoked) seizure are not classified as epileptics (84).

Since epilepsy may occur in many different clinical settings and may take a number of different forms and because the pathophysiology of epileptic seizures is little understood, classification has proved problematic and has been the subject of considerable debate. (49,67,84). Modern clinical classifications of epilepsy has been of two main types. The first have categorized epilepsy according to seizure type. This has the disadvantage that similar types of attack may

occur in widely differing conditions. The second have classified epilepsy according to aetiology. This has the disadvantage that in a great many cases no aetiology can be established. Other systems based on age, putative pathophysiology and anatomical site have also b*men proposod. All these schemes are more or less empirical (49).

The current classification of seizure type was introduced by the International League Against Epilepsy in 1969 (49,67) ILAE. It was modified in 1981 (see Table 6).

The 1981 modification (see Table summary in Table 6) is based on the clinical seizure type and ictal and interictal EEG, expression only. Anatomical substrate, aetiology or age was also used in the earlier classification (49,67,84). The 1981 proposal is widely accepted. The International League Aqainst Epilepsy (ILAE) in 1984 adopted the 1981 classification and devised a new scheme - the classification of Epilepsies and Epileptic Syndromes and related seizure disorders in 1989. This is also now widely used and is an attempt to categorize the epilepsies more comprehensively (49,67,84) see (Table 7). Table 6: International Classification of Seizure Type (1981 Revision)

I PARTIAL SEIZURES (Focal)

- A Simple partial seizures (preservation of consciousness)
 - 1. With motor signs
 - 2. With somatosensory or special sensory hallucinations
 - 3. With autonomic symptoms and signs
 - 4. With psychic symptoms
- B Complex partial seizures (alteration of consciousness)
 - Simple partial onset followed by impairment of consciousness

- 2. With impaired consciousness at onset.
- C Partial seizures evolving to secondarily generalized seizures
 - Simple partial seizures evolving to generalized.
 - Complex partial seizures evolving to generalized
 - Simple partial seizures evolving to complex partial seizures evolving to generalized
- - A 1. Absence seizure
 - 2. A typical absences
 - B Myoclonic seizures
 - C Clonic seizures
 - D Tonic seizures
 - E Tonic-clonic seizures (grand mal)
 - F Atonic seizures (a static seizures)
 - * Combinations may occur such as (B) and (F) or (B) and (D)

III UNCLASSIFIABLE EPILEPTIC SEIZURES

ILAE (INTERNATIONAL LEAGUE AGAINST EPILEPSY) CLASSIFICATION OF SEIZURE TYPE (1981)

In this scheme the seizures are divided into 3 groups:-

- i) Partial seizures (focal seizures)
- ii) generalized seizures and
- iii) Unclassified seizures.

Partial seizures are further subdivided into:-

1) simple partial and

2) complex partial seizures (according to the preservation or alteration of consciousness).

Seizures with preservation of consciousness are regarded as simple partial seizures while those with alteration of consciousness are regarded as complex partial seizures.

Generalized seizures are those in which epileptic discharges involve both cerebral hemispheres widely and simultaneously from the onset, of the seizure, whereas partial seizures are those in which epileptic activity of the brain of partial seizures, simple or complex may spread to become generalized in which case the seizure is said to be secondarily generalized (see Table 6) (49,67).

The International League Against Epilepsy (ILAE) in 1989 in recognisation of the fact that a seizure - type classification does not account for other aspects of the heterogeneity of epilepsy devised a new scheme. This is now referred to as the classification of the epilepsies and epileptic syndromes and related seizure disorders. This is now also widely used as an attempt to categorize the epilepsies more comprehensively (49,67) see table 7.

ILAE CLASSIFICATION OF THE EPILEPSIES AND EPILEPSY SYNDROMES AND RELATED SEIZURE D180RDER8 (1989) see Table 7

The classification (see table 7) takes into account seizure type, EEG, and prognostic, pathophysiological and aetiological data. It retains the division of epilepsy into generalized and partial (now called localisation related) categories. Each category is subdivided into symptomatic and idiopathic epilepsy. Two new categories were added i.e. epilepsies and syndromes undetermined, whether focal or generalized and special syndromes. This is a complex scheme and may well confuse non-taxonomists, but it is a serious attempt to incorporate more than simple seizure type data into a comprehensive classification (49,67).

- Table 7: <u>INTERNATIONAL CLASSIFICATION OF EPILEPSIES</u> ANFI EPILEPTIC SYNDROMES AND RELATED SEIZURE DISORDERS (1989)
 - 1 Localisation related (local, focal, partial)
 epilepsies and syndromes
 - 1.1 <u>Idiopathic</u> (with age-related onset).

 Benign childhood epilepsy with centro-temporal spike.

Childhood epilepsy with occipital paroxysms. Primary reading epilepsy.

1•2 Symptomatic

- a) Chronic progressive epilepsia partialis continua.
- b) Syndromes characterised by seizures with specific modes of precipitation
 - i) Temporal lobe epilepsies
 - ii) Frontal lobe epilepsies
 - iii) Parietal lobe epilepsies
 - iv) occipital lobe epilepsies

1.3 Cryptogenic

2. Generalized epilepsies and Syndromes

2.1 Idiopathic (with age - related onset)
Benign neonatal familial convulsions
Benign neonatal convulsions
Benign myoclonic epilepsy in infancy
Childhood absence epilepsy
Juvenile absence epilepsy
Juvenile myoclonic epilepsy
Epilepsy with grand mal seizures (GTCS) on awakening other generalized idiopathic epilepsies
Epilepsies with seizures precipitated by specific modes of activation

35 **VSc**

2.2 cryptogenic pr sytnptom9tjg epilepsy

West Syndrome

Lennox-Gastaut syndrome

Epilepsy with myoclonic -astatic seizures

Epilepsy with myoclonic absences

2.3 Symptomatic

2.3.1 Non specific aetiology

Early myoclonic encephalopathy
Early infantile epileptic encephalopathy
with suppression burst

Other symptomatic generalized epilepsies

2.3.2 <u>Specific syndromes</u>

Epileptic seizures complicating other disease states

- 3. <u>Epilepsies and syn</u>drom<u>es unde</u>termined whether foca<u>l or</u> generalized
 - 3.1 With both generalized and focal seizures

Neonatal seizures

Severe myoclonic epilepsy of infancy

Epilepsy with continuous spike wave during slow wave sleep

Acquired epileptic aphasia

other undetermined epilepsies

- 3.2 Without unequivocal generalized or focal features
- 4. Special syndromes
 - 4.1 Situation-related seizures

Fibrile convulsions

Isolated seizures or isolated status epilepticus Seizures occurring only with acute metabolic or toxic events.

PHYSIOLOGIC NEUROIMAGING IN EPILEPSY

There are three types of neuroimaging modalities for evaluation of seizure disorders, namely (i) interictal positron emission tomography (PET), (ii) Xenon computerized tomography (Xenon CT) (iii) single photon emission computerized tomography (SPECT) (80, 81,84).

The three methods are used to identify the "functional deficit zone". This is the area of brain that functions abnormally in the interictal period (80, 81, 84). The xenon computerized tomography (xenon CT) is used to a lesser extent (84).

PET studies of cerebral oxygen utilisation, glucose metabolism and blood flow, have been using various radionuclides. The radionuclides that have been used commonly are oxygen 15 (15 0), carbon 11 (" C) and fluorodeoxyglucose (FOG) (52, 71, 74, 80, 81, 84). SPECT studies employ iodine 123 (123 I), some Iodamines, and technetium 99m ("•" TC) Hexamethyl - propylene amine oxime (11MPAO) (84).

Both PET and SPECT tend tq demonstrate a zone of hypometablism or hypoperfusion during the interictal period. The zone of hypometabolism or hypoperfusion is considerably larger than the epileptogenic zone (80,84).

The sensitivity of SPECT is roughly half that of PET in temporal lobe seizure zone localization (84).

PET is accurate in identifying patients with brain tumours and mesial temporal sclerosis (MTS), also called Ammon's horn or hippocampal sclerosis. Magnetic Resonance Imaging (MRI) is also highly accurate in identifying similar pathology (84).

Mesial temporal sclerosis (MTS), causes medically intractable temporal lobe epilepsy. Computerized axial tomography is not good for localisation of temporal lobe sclerosis due to bone artefacts.

Positron Emission Tomography

This method requires expensive equipment. A cyclotron is necessary for the production of the isotopes. It also requires a large team of specialises, making it therefore a research tool (70,71,84).

Most commonly used radionuclides are isotopes of oxygen (15 o) (with a half-life of 2 minutes), Nitrogen (13 N), with a half-life of 20 minutes (92). Commonly occurring organic molecules e.g. glucose combined with fluorine (1a F), (Fluorodeoxyglucose (FDG), is also used. This has a half life of 110 minutes (74,84,92).

A positron is a particle with a positive charge (+ve) and a mass egual to that of an electron. The two particles undergo annihilation process in which masses of the two particles are converted into electromagnetic radiation in the form of two photons (annihilation gamma rays) each with an energy of 0.511 Mev, and travel in opposite directions of nearly 180° apart. The detection of the annihilation technique is by PET system which is used to produce an image of the distribution of positron activity in the tissue (80,81,84,92).

An array of radiation detectors are used in which two detectors sense the annihilation photons simultaneously or nearly simultaneously (92).

The radionuclides are given intravenously or by inhalation. The constructed images enable information to be obtained about regional blood flow, oxygen extraction, or utilization by glucose uptake (74,80,81,84,92).

PET using fluoro-deoxyglucose (FDG) gives a general view of brain activity which can be compared with MRI results (80, 81, 84, 92).

In high-grade brain malignancy there is high glucose uptake. This is not clear from either MRI or CT scans (80,81,84,92).

High resolution PET (2.6 mm) is also used for imaging patients with complex partial seizures of the temporal lobe, e.g. in mesial temporal sclerosis (MTS) (81).

Single Photon Emission Computerized Tomography (SPECT)
This utilises the gamma camera which is made to rotate around the patient. The radioactivity is recorded at numerous angles. Sectional images are obtained enabling reconstruction to be carried out. An intravenous injection of a gamma emitting radio-isotope labelled compound usually small lipophilic molecules are used (These are able to cross the blood brain barrier (92).

These molecules remain in place long enough to be monitored by a computer controlled gamma camera (92).

SPECT is a good method of measuring regional blood flow which is largely proportional to the rate of local metabolism. In epilepsy hypoperfusion is shown between epileptic attacks and hyperperfusion during focal seizures.

SPECT does not reguire a cyclotron and is therefore used widely. Radioisotopes used are Iodine 123 ($^{1?}$ 'l) and technetium 99m (99m Tc) - Hexamethyl-propylene amine oxime (HMPAO) (84, 92).

MAGNETIC RESONANCE IMAGING IN EPILEPSY (MRI)

The recent installation of two Magnetic Resonance Imaging (MRI) machines in Nairobi in October 1996 is a very good development in the field of neuroimaging, especially for patients with certain types of epilepsy, that are not detectable by Computerized Tomography (CT). Computerized Tomography (CT) of the head is known to have limitations in imaging certain types of epilepsy e.g. temporal lobe epilepsy (TLE). Beam hardening artefacts in the temporal lobes interfere with the CT images and therefore make it difficult to detect lesions in the temporal lobe that cause epilepsy (51, 55, 72, 73, 80, 83, 84).

Even selection of particular positioning of patients or special angulation of the CT gantry (e.g. 15-20°) from Reid's baseline does not eliminate the limitation of CT in the evaluation of temporal lobe lesions (55).

Although CT can demonstrate many epileptogenic lesions the overall sensitivity of MRI in the detection of structural epileptogenic abnormalities is higher (80,84). MRI is a better imaging modality for the detection of certain pathological conditions e.g. mesial temporal sclerosis (MTS), and small neuronal migrational anomalies (51, 55, 64, 66, 69, 80, 83, 84).

Mesial temporal sclerosis is the most common single lesion found in post mortem brains of chronic epileptics who die a natural death (21). Evidence shows that it usually arises in infancy often as a result of prolonged febrile convulsions. Thereafter it becomes a potent epileptogenic lesion. It is usually unilateral (21).

This is cured by temporal lobectomy with excellent results (21, 71, 80, 84). These patients experience medically intractable seizures (21,71, 80, 84). Before the advent of MRI, hippocampal sclerosis (Mesial temporal sclerosis, Ammon's horn sclerosis) was rarely diagnosed preoperatively. The critical age in children for developing mesial temporal sclerosis is between 6 months and 4 years (21).

There is no universally accepted criteria that define when an MRI, examination is indicated in a patient with epilepsy (84). It is wise however to examine early patients who have partial onset seizures. This is because these patients have medically refractory seizures (84). Also patients who have generalized epilepsy who are poorly controlled medically should be examined with MRI (84). Usually patients with generalized epilepsy respond well to medical therapy and do not require examination with MRI (84).

Epileptic patients with brain lesions usually do not respond well to medical therapy (84).

In patients with intractable epilepsy most reports indicate that CT is able to detect a focal abnormality in approximately one fourth (1/4) of the patients. With MRI a focal abnormality is detected in approximately one half ($^{\prime\prime}_2$) of ^{the} patients (80).

MRI is also superior in the detection of heterotopic gray matter. These are cortical neurons in abnormal locations. They are caused by abnormal migration of neuroblasts from the ventricles to the brain surface. Heterotopia is typically found adjacent to the lateral ventricles, or within the white matter close to the sylvian fissure. Heterotopia also causes seizures (84). Prior to the era of MRI heterotopia was frequently not diagnosed (84).

Congenital lesions such as glial harmatomas, vascular malformations, and migrational disorders are also well diagnosed with MRT (84). These congenital lesions cause seizures (84). Developmental anomalies associated with seizures e.g. schizencephaly, lissencephaly, hemimegalencephaly, and anterior temporal lobe encephaloceles, and cortical dysplasias, are well diagnosed with MRT (84).

MRI gives better views of indolent gliomas located in the medial temporal lobe structures (51,71). MRI should be used for patients with medically uncontrollable seizures and in whom CT scan results are negative. Also patients with focal epilepsy who have negative CT scans findings. Other patients include those with late onset epilepsy and those who are candidates for surgical treatment e.g. patients with mesial temporal sclerosis (MTS) (51, 64, 66, 71, 74, 80, 84, 85, 87).

Limitations of MRI include its high cost, therefore rendering it out of reach for many patients who cannot afford it. Other limitations are related to its powerful magnetic field which is hazardous to patients with pace-makers, orthopaedic plates, aneurysm clips and even credit cards (74).

The most obvious improvement of MRI over CT is the better differentiation between grey and white matter because grey matter contains more hydrogen in the form of water, than white matter. Also the hydrogen atoms are less bound in fat and large molecules e.g. caudate and lentiform nuclei are visible in the basal ganglia. The medulla, pons and the posterior fossa, structures are also better visualised without the CT bone artefacts (74).

MOTIVATION

In Kenya Neuroradiology is a well established mode of imaging, in Nairobi and Mombasa. The availability of computerized tomography machines in Nairobi and one in Mombasa has greatly improved neuroradiology imaging in the country. Inspite of this proliferation in the two major metropolitan areas in the field of neuroimaging, patients have to travel long distances to have a computerized tomography examination done. This makes the examination more expensive for patients living far from the two cities. It is also inconvenient for sick people who have to travel long distances for this type of examination. Computerized tomography examination of the brain is still an expensive examination at the moment.

This mode of investigation is routinely requested by clinicians for patients suffering from epilepsy. The number of epileptic patients in Kenya is estimated at about 500,000 people. This number is expected to rise due to an increase in road traffic accidents (RTA). The demand for computerized tomography of the head for evaluation of epilepsy and other diseases is high and will continue to rise. It is therefore impracticable to do computerized tomography of the brain for all epileptic patients. A reasonable criteria needs to be established for selecting patients who would definitely benefit from computerized tomography for the evaluation of epilepsy.

OBJECTIVES OF THE STUDY

- 1. To assess the radiological manifestations of epilepsy by computerized tomography of the brain.
- To establish whether intravenous contrast media used for computerized tomography of the brain examination assists in procuring more pathological information during computerized tomography examinations.
- 3. To outline a reasonable criteria for selecting patients with epilepsy who would benefit most from computerized tomography examination of the brain and establish whether such a criteria would be of benefit to the clinicians in deciding which epileptic patients really need to undergo computerized tomography of the brain for evaluation, according to the study results.

STUD1LDESIGN

Inclusion Criteria

- 1. Patients who satisfied the criteria of the International League Against Epilepsy (ILAE) 1981 revision (Table 6) for classification of epilepsy.
 - (or) patients who satisfied the International League Against Epilepsy and Epilepsy Syndromes and related seizure disorders classification of 1989 (table 7).
- 2. Patients who had clinical or electroencephalographic (EEG) pattern of epilepsy according to ILAE classifications (tables 6 and 7).
- 3. All patients must have undergone a computerized tomography examination of the brain for evaluation of epilepsy or convulsive disorders as outlined above.

Exclusion Criteria

- 1. Patients were excluded from the study if they had metabolic causes of convulsions e.g. patients who had hypomagnesemia, hypoglycaemia, or hypocalcaemia.
- 2. Patients who had provoked seizures e.g. febrile convulsions were excluded unless their convulsions continued long after their febrile episodes were over.
- 3. Patients with known nutritional diseases which could present with convulsions e.g. pyridoxine deficiency and aminoacidopathies.
- 4. Patients who had altered (metabolic) causes of seizures e.g. renal failure, and hepatic failure.
- 5. Patients who had drug induced seizures.
- 6. Patients who had incomplete clinical or demographic data, especially if they were referrals from other health institutions where it was impracticable to obtain such data.

ETHICAL CONSIDERATIONS

This was a retrospective study. The author reviewed only films and relevant files belonging to the patients. Confidentiality was kept to the extreme. No attempt was made

to expose patients data or relevant records. There were no further investigations needed. So patients were not exposed to any radiation hazards, or to any further expenses.

DATA ANALYSIS

Data is presented in a tabulated format, so as to analyse them as regards:-

- Correlation of computerized tomography of the brain (CT) results to causes of epilepsy.
- 2. Correlation of computerized tomography (CT) of the brain results to duration of seizures.
- 3. Correlation of computerized tomography of the brain (CT) findings with age of patient.
- 4. Compare computerized tomography of the brain (CT) with seizure patterns.
- 5. Age and sex distribution of epilepsy according to computerized tomography (CT) of the brain findings.
- 6. Correlate computerized tomography (CT) of the brain findings to surgically treatable causes of epilepsy as revealed in the study.
- 7. Compare pre and post contrast media enhancement of computerized tomography of the brain examination in patients with epilepsy.

The above data was collected and analysed by an International Business Machine (IBM) compatible computer and a statistical package for the social sciences (SPSS) was used.

SAMPLE.8IZ EDETERMINAT10N

Using a confidence level of 95% and taking into account that the population of Kenya is now approximately 27.214 million and since Kenyatta National Hospital is the main referral hospital a degree of accuracy of between 0.02 and 0.05 level (i.e. between 2 and 5%) would be realistic.

The sample size was calculated bearing in mind the above parameters using the formula

 $n = Z^2 \text{ pcj.}$ (44, 50, 57)

- Where n= the desired sample size (when the population is greater than 10,000.
 - p = the proportion in the target population estimated
 to have epilepsy in Kenya is 500,000 or (0.02 of
 the total population of Kenya),
 - z = the normal standard deviation usually set at 1.96 (or more simply at 2.0) which corresponds to the 95 percent confidence level,
 - q = 1.0 p
 - d = degree of accuracy desired which was at between
 0.05 (low degree of accuracy) to 0.02 (high degree
 of accuracy). It is usually set at 0.05 or
 occasionally 0.02 (44).
 - $n (2.0)^2 X 0.02 x 0.98) = 0.0784$

0.02 0.0004

- 196

MATERIALS AND METHODS

The study was retrospective and included patients who had a computerized axial tomography (CT) examination of the head done at Kenyatta National Hospital (KNH) due to convulsive disorders.

The study period was from 11/11/92 when a Philips computerized tomography machine (Tomoscan CXQ) was installed and became operational at Kenyatta National Hospital up to 31/7/96. During this period the CT machine was grounded for repairs for a total period of 15 months leaving a cumulative total study time of two and a half years.

Patients were selected from the records kept at the X-ray Department (Computerized tomography section) of KNH. Further information such as relevant history, presenting symptoms (EEG) reports, previous history of surgery if any were obtained from the Records Department of KNH.

The study included patients of all age groups, children and adults who had a CT scan examination of the head done due to convulsive disorders.

Patients were included in the study if they had a clinical or electroencephalographic (EEG) pattern of epilepsy according to the International League Against Epilepsy (II.AE) criteria, (1981) revision, or according to the International League Against Epilepsy and Epilepsy syndromes and Related Seizure Disorders classification of (1989).

All the available CT scan films of the patients were reported by consultant Radiologists. Patients data were recorded in the data collection sheet (see Ap. 1). This included the age, sex of the patient, x-ray registration number, hospital registration number, referring ward or clinic, intravenous contrast medium use or not, clinical history, seizure pattern, duration of seizures if known, and EEG report where available. Provisional diagnosis and radiological diagnosis were also recorded. Patients with incomplete clinical or demographic data were excluded from the study.

Patients were referred for CT san of the head from Paediatric Neurology Clinic, Neurology Clinic (adults) and from Neurosurgery Clinic of the hospital. Other patients were referred from the medical, surgical and paediatric wards of the hospital.' Patients were also referred for CT scan of the head due to convulsive disorders from various Provincial and District hospitals, private clinics as well as private hospitals. All patients had axial scans done from the base to the vertex. Patients with space occupying lesions detected during the CT scan examination had image reconstructions done in either sagittal or coronal planes or both, if this was considered necessary.

The results are presented in a tabulated form.

RESULTS

A total of 484 patients were studied. The age distribution is presented in Table 8. The highest number of patients (97) (20.0%) were below 5 years of age. The lowest number of patients were above 80 years with only one case (0.2%).

The sex distribution is presented in Table 9. The number of male patients was 283 (58.5%) and the number of female patients was 201(41.5%).

Age and sex distribution is presented in Table 10.

There was a decrease of the number of patients above 35 years old (males and females). There was an increase of the number of patients below 20 years. Of the patients below 5 years old 58 were males (20.5%) and 39 (19.4%) were females.

Table 11 summarizes the presenting symptoms. All 484 patients had convulsions generalized, focal or status epilepticus etc. (100%). The second presenting symptom was headache with 39 patients (8.1%) followed by decreased level of consciousness with 36 cases (7.4%). Visual disturbance including blindness was the fourth commonest presenting symptom. Other symptoms are presented in Table 11. Signs found in epileptic patients are presented in Table 12. The commonest sign was weakness of

limbs (upper and lower limbs). Left sided weakness was predominant with 44 cases (9.1*) and right sided weakness had 27 cases (5.6*). Cranial nerve palsies, hemiplegia, dysphasia followed with 8 cases each (1.7*).

Table 13 summarizes the types of seizure pattern seen in the study. Generalized convulsive seizures were predominant with 395 cases (81.6%) followed by simple partial seizures with 10.5%.

Table 14 summarizes the CT scan results . Patients with abnormal or positive CT scan findings were 273 (56.40%). Brain atrophy had the highest abnormal or positive CT scan findings with a total of 132 patients. Diffuse brain atrophy was predominant with 94 patients (94 of 132) or (71.2%), and focal brain atrophy was second with 38 patients (38 of 132) or (27.8%).

Brain tumours with 38 cases (7.9%) was second in the number of patients with abnormal or positive CT scan findings. Infections with 29 cases (6.0%) was third in the number of patients with abnormal CT scan findings. Amongst the infections brain abscesses had 17 patients (17 of 29) or (58.6%) abnormal CT scan findings. Granuloma followed with 7 cases (24.1%) and tuberculoma had 5 cases (17.2%).

Trauma cases were fourth in the number of abnormal CT scan findings 24 cases (5.0%) of all patients in the study. The predominant finding was chronic subdural haematoma with 7 cases (7 of 24) or 29.2%. This was followed by intracerebral haematoma with 6 cases (25.0%).

There were 19 cases (3.9%) of cerebral infarcts as well as 19 cases (3.9%) of hydrocephalus. In patients with hydrocephalus 15 cases (15 of 19) or (78.9%) were communicating hydrocephalus and 4 cases (21.0%) were obstructive hydrocephalus. There were only 5 cases of arteriovenous malformations (A-V malformations).

Table 15 presents the relationship of CT scan results and, the age of the patient at the time of the examination. There were 18 patients above 65 years old (83.3%) who had abnormal CT scan findings. The highest number of patients were in the age group below 10 years with 133 cases (27.5%) of the total number in the study. Abnormal CT scan findings in this group were (67.7%).

Table 16 outlines the relationship between the duration of seizures at the time of the examination and the CT scan findings. The highest number of patients i.e (87) were in the group with a short duration of seizures (less than 6 months). This group had the highest abnormal CT scan findings with

(65.5%). There were 30 patients with duration of seizures above 5 years. Abnormal CT scan findings in this group were 40%.

Table 17 relates the type of seizure pattern to the CT scan findings. Generalized convulsions had the highest number of patients. The percentage of abnormal cases in this group was 55.7%. Simple partial seizures had 51 patients and abnormal CT scan findings of (66.6%). This was the highest number of abnormal CT scan findings.

Table 18 summarises the use of contrast medium during CT scan examinations. Only 207 cases received intravenous contrast medium during the CT scan examinations.

Table 19 represents the relationship of CT scan results of brain tumours with age of the patient.



TABLE 8: AGE DISTRIBUTION: NO. OF PATIENTS

AGE IN YEARS	NO. OF PATIENTS (%)
0 - 5	97 (20.0)
6 - 10	36 (7.4)
11 - 15	62 (12.8)
16 - 20	49 (10.1)
21 - 25	36 (7.4)
26 - 30	33 (6.8)
31 - 35	27 (5.8)
36 - 40	21 (4.3)
41 - 45	21 (4.3)
46 - 50	10 (2.1)
51 - 55	10 (2.1)
56 - 60	5 (1.0)
61 - 65	10 (2.1)
66 - 70	5 (1.0)
71 - 75	5 (1.0)
76 - 80	7 (1.4)
ABOVE 80	1 (0.2)
UNSPECIFIED	49 (10.1)
1 TOTAL	484 (100%)

TABLE 9: SEX DISTRIBUTION

SEX	FREQUENCY	PERCENTAGE
MALE	283	58.5
FEMALE	201	41.5
1 TOTAL	484	100

TABLE 10: AGE, SEX DISTRIBUTION: NUMBER OF PATIENTS (%)

AGE IN YEARS		MALE	FEMALES		
	NO.	%	NO.	%	
0 - 5	58	(20.5)	39	(19.4)	
6 - 10	20	(7.1)	16	(8.0)	
11 · 15	29	(10.2)	33	(16.4)	
16 - 20	23	(8.1)	26	(12.9)	
21 - 25	23	(8.1)	13	(6.5)	
26 - 30	18	(6.4)	15	(7.5)	
31 - 35	20	(7.1)	7	(3.5)	
36 - 40	15	(5.3)	6	(3.0)	
41 - 45	16	(5.7)	5	(2.5)	
46 - 50	5	(1.8)	5	(2.5)	
51 - 55	7	(2.5)	3	(1.5)	
56 - 60	3	(1.1)	2	(1.0)	
61 - 65	6	(2.1)	4	(2.0)	
66 - 70	3	(1.1)	2	(1.0)	
71 - 75	2	(0.7)	3	(1.5)	
76 - 80	5	(1.8)	2	(1.0)	
ABOVE 80	1	(0.4)		-	
UNSPECIFIED	29	(10.2)	20	(10.0)	
TOTAL	283	(100%)	201	(100%)	
%TOTAL (MALE AND			484	(100%)	
FEMALE)		58.5%		41.5%	

TABLE 11: SYMPTOMS FOUND IN PATIENTS REFERRED FOR CT SCAN OF THE HEAD DUE TO EPILEPSY

GVMDEOMG	NO 07 7	
SYMPTOMS	NO. OF P	PATIENTS (%)
HEADACHE	39	(8.1)
DECREASED LEVEL OF CONSCIOUSNESS	36	(7.4)
VISUAL DISTURBANCE (INCLUDING BLINDNESS)	17	(3.5)
ATAXIA	12	(2.5)
FEVER	11	(2.3)
COMA	10	(2.0)
MENTAL CHANGES (INCLUDING PSYCHOSIS)	9	(1.9)
ENLARGED HEAD	9	(1.9)
SPEECH DISTURBANCE	8	(1.7)
CONFUSION	7	(1.4)
VOMITING	7	(1.4)
DELAYED MILESTONES	5	(1.0)
EAR DISCHARGE (CHRONIC)	4	(0.8)
LOSS OF MEMORY	3	(0.6)
UNILATERAL FACIAL WEAKNESS	3	(0.6)
LOSS OF HEARING	2	(0.4)
BLEEDING FROM THE EAR	1	(0.2)
OTORRHEA	1	(0.2)
HALLUCINATIONS	1	(0.2)
LOSS OF HEARING BLEEDING FROM THE EAR OTORRHEA	2 1 1	(0.4) (0.2) (0.2)

TABLE 12: SIGNS FOUND IN PATIENTS REFERRED FOR CT SCAN OF THE HEAD DUE TO EPILEPSY

SIGNS	NO.	OF PATIENTS (%)
WEAKNESS OF (LT SIDED WEAKNESS) LIMBS (RT SIDED WEAKNESS)	44 27	(9.1) (5.6)
CRANIAL NERVE PALSIES	8	(1.9)
HEMIPLEGIA	8	(1.7)
DYSPHASIA	8	(1.4)
PAPILLOEDEMA	6	(1.2)
NECK STIFFNESS	6	(1.2)
TODD'S PARALYSIS	6	(1.2)
PUPILLARY DILATATION	5	(1.0)
PARAPARESIS	4	(0.8)
HYPEREFLEXIA	3	(0.6)
PTOSIS	2	(0.4)
NYSTAGMUS	1	(0.2)
UNILATERAL PARESTHESIA	1	(0.2)
DECEREBRATE RIGIDITY	1	(0.2)
PIN POINT PUPILS	1	(0.2)
HYPOTONIA	1	(0.2)

TABLE 13: SETZURE PATTERN

TYPE OF SEIZURE ;	FREQUENCY	PERCENTAGE
SIMPLE PARTIAL SEIZURES (SPS)	51	10.5
PARTIAL COMPLEX SEIZURES (PCS)	11	2.3
PARTIAL SECONDARILY GENERALIZED SEIZURES (PSG)	17	3.5
GENERALIZED CONVULSIVE SEIZURES (GCS)	395	81.6
STATUS EPILEPTICUS	3	0.6
OTHERS*	7	1.4
I TOTAL	484	100

TABLE 14: RESULTS OF COMPUTERIZED TOMOGRAPHY OF THE HFAD FOR PATIENTS WITH EPILEPSY

RESULTS	NO	OF CASES	(%)
NORMAL	211	01 0110110	(43.6)
BRAIN (a) DIFFUSE	94		(19.4)
ATROPHY (B) FOCAL	38		(7.9)
OBSTRUCTIVE	4		(0.8)
HYDROCEPHALUS COMMUNICATING	15		(3.1)
ARTERIOVENOUS MALFORMATION (A-V MALFORMATION)	4		(0.8)
CEREBRAL INFARCTS	19		(3.9)
BRAIN TUMOURS (NEOPLASMS)	38		(7.9)
TUBERCULOMA	5		(1.0)
INFECTIONS BRAIN ABSCESS	17		(3.5)
GRANULOMA	7		(1.4)
ACUTE SUBDURAL	3		(0.6)
CHRONIC SUBDURAL	7		(1.4)
EXTRADURAL	3		(0.6)
TRAUMA SUBARACHNOID	1		(0.2)
INTRACEREBRAL	6		(1.2)
FRACTURES	4		(0.8)
OTHERS *(See below)	8		(1.7)
TOTAL	484		(100%)
% ABNORMAL	56.	4 %	(0.2)

•Others - small cerebellum, cerebellar Infarct, basal ganglia calcification, hyperostosis, frontalis interna; oedema.

i RESULTS OF CT		0-10 yrs	11-20 yrs	21-30 yrs	31-45 yrs	46-65 yrs	>65 yrs	UNSPE- CIFIED	TOTAL	%
i NORMAL	(M) (F)	24 19	29 36	20 16	19 7	8 4	1 2	15 11	116 95	(23.6) (19.6)
BRAIN ATROPHY DIFFUSE FOCAL	(M) (F) (M)	23 15 S	8 4 2	1 1 3	9 4 5	5 6 1	7 5 -	2 4 2	55 39 21	(11.4) (8.0) (4.3)
HYDROCEPHALUS	(F) (M) (F)	10 7	6 - 2	- -	2 	2 	- -	_	17 10 9	(3.5) (2.0) (1-9)
: A-V ; MALFORMATIONS	(M) (F)	-	- 1	2 –	-		<u>-</u>	1 1	3 2	(0.6) (0.4)
i CEREBRAL ¹ INFARCTS	(M) (F)	2 -	1 2	4 1	2 1	3 2	-	1 1	13 7	.(2.7) (1.4.)
BRAIN TUMOURS	(M) (F)	3 2	4 5	3 2	7	1 2	2	4 3	24 14	(5.0) (2.9)
OLD HEAD TRAUMA	(M) (F)	3 2	1 1	5 -	6 1	2	1	2	20	(4.1) (0.8)
INFECTIONS	(M) (F)	4 6	5 3	3 1	2 2	1	<u>-</u>	2 -	17 12	(3.5) (2.5)
BRAIN STEM 1 BLEED	(M) (F)	_	_ 1	-	-	-	<u>-</u>	<u>-</u>	<u> </u>	(0.2)
OTHERS *	(F) (M)	2 -	1 -	- 1	- 1				3 2	(0.6) (0.4)
· TOTAL		133	112	67	6S	37	IS	49	484	(100%)
% ABNORMAL		67.7%	42.0%	46.3%	61.8%	1 67.6%	83.3%	46.9%	56.4%	L

TABLE 16: COMPARISON OF COMPUTERIZED TOMOGRAPHY (CT) RESULTS WITH DURATION OF SEIZURES

j RESULTS OF CT	< 6 MONTHS	6 Mos - 1 yr	1 - 5 yrs	> 5 yrs	TOTAL	8
NORMAL	30	10	21	18	75	(16.3)
DIFFUSE i BRAIN ATROPHY	16	4	6	5	31	(6.4)
FOCAL	7	2	1	4	14	(2.9)
HYDROCEPHALUS						
OBSTRUCTIVE	-	-	_	_	_	_ (0.6)
COMMUNICATING _	2		1	_	3	(0.0)
ARTERIOVENOUS (AV) I MALFORMATIONS	-	_	1		1	(0.2)
BRAIN TUMOURS GLIOMA(S) MENINGIOMA PITUITARY MASS CRANIOPHARYNGIOMA ANGIOMA DERMOID CYST	6 - 1 1 - -	- - - - -	3 1 1	1	9 1 2 2 1 1	(1.9) (0.2) (0.4) • (0.4) (0.2) (0.2)
CEREBRAL INFARCTS	4	2	1	-	7	(1.4)

TABLE 16: COMPARISON OF COMPUTERIZED TOMOGRAPHY (CT) RESULTS WITH DURATION OF SEIZURES (CONT•)

RESULTS OF CT	< 6 MONTHS	6 Mos - 1 yr	1 - 5 yrs	> 5 yrs	TOTAL	૪
; TRAUMA 1) ACUTE SUBDURAL						
HAEMATOMA	1			-	1	(0.2)
2) CHRONIC SUBDURAL 3) EXTRADURAL	2	-	1		3	(0.6)
BLEED 4) INTRACEREBRAL	1 2				1	(0.2)
1 5) FRACTURES	1		1		2 2	(0.4) (0.4)
"INFECTIONS ! TUBERCULIMA BRAIN ABSCESS	2 8			1	2	(0.4)
i GRANULOMA	O	1	1	1 1	9 3	(1.9) (0.6)
i HYGROMA (BIFRONTAL)	1	-	-	-	1	(0.2)
OTHERS ' BRAIN STEM BLEED	1				1	(0.2)
: CEREBELLAR , INFARCT	1				1	(0.2)
PATIENTS WITH UNKNOWN DURATIONS					307	(63.4)
¹ TOTAL	87	19	41	30	484	(100%)
' %ABNORMAL	65.5%	47.4%	48.8%	40.0%	83.7%	

TABLE 16: COMPARISON OF COMPUTERIZED TOMOGRAPHY (CT) RESULTS WITH DURATION OF SEIZUR ES

RESULTS OF CT	SIMPLE PARTIAL SEIZURES	PARTIAL COMPLEX SEIZURES	PARTIAL SECON- DARILY GENE- RALIZED	GENE- RALISED NON CONVUL- SIVE	GENE- RALISED CONVUL- SIVE	SPECIAL TYPES OF SEI- ZURES	STATUS EPILEP -TICUS	TOTAL	%
i NORMAL	17	9	7	-	175	3	_	211	(43.6)
DIFFUSE BRAIN 1 ATROPHY	10	1	6		75	1	1	94	(19.4)
FOCAL	4	1	1		29	3		38	(7.9)
OBSTRUCTIVE	1	-	-	-	3	-	-	4	(0.8)
j HYDROCEPHALUS									
COMMUNICATING	2	-	-	-	12	-	1	15	(3.1)
ARTERIOVENOUS (AV) ' MALFORMATIONS	1	-	2	_	1	-	-	4	(0.8)
i CEREBRAL 1 INFARCTS	2	-	-	-	16	-	1	19	(3.9)
BRAIN TUMOURS	6	-	-	-	32	-	-	38	(7.9)

TABLE 17: COMPARISON OF COMPUTERIZED TOMOGRAPHY (CT) RESULTS WITH SEIZURE PATTERNS (continued).

TABLE 17. COMPAN	CIBON OF COL	ME O I EIX I Z ED	TOMOGNATITE	(CI) NEC	OUIS WIIII	SEIZONE E	ATTEMNS	(COIICIII de	<i>a)</i> .
RESULTS OF CT	SIMPLE PARTIAL SEIZURES	PARTIAL COMPLEX SEIZURES	PARTIAL SECON- DARILY GENE- RALIZED	GENE- RALISED NON CONVUL- SIVE	GENE- RALISED CONVUL- SIVE	SPECIAL TYPES OF SEI- ZURES	STATUS EPILEP -TICUS	TOTAL	%
TRAUMA CASES ACUTE SUBDURAL									(0.6)
CHRONIC SUBDURAL					7			7	(1.4)
EXTRADURAL					3			3	(0.6)
INTRACEREBRAL					5			6	(1.2)
SUBARACHNOID					1			1	(0.2)
FRACTURES					3			4	(0.8)
INFECTIONS									
TUBERCULOMA					5			5	(1.0)
BRAIN ABSCESS	4				13			17	(3.5)
GRANULOMA	1				5			7	(1.4)
•OTHERS (see below)					8			8	(1.7)
TOTAL	51	11	17		395			484	(100%)
%ABNORMAL SPECIAL TYPES O?	66.6%	18.2%	58.8%		<u>55.7%</u>	57.1%	100%	56.4%	
mmm-mm^m* ry-A I r		n r> r\c- I .	SPASMS,		HYTHMIA, e		• •:	• i *• \ r-:•>- i	r^nc-

TABLE 18 (i)r RESULTS OF INTRAVENOUS (IV) CONTRAST MEDIUM USE DURING CT SCAN EXAMINATION FOR EPILEPSY

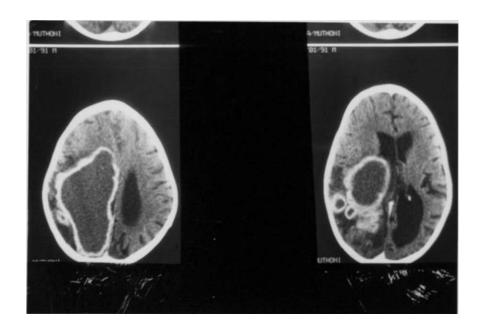
I.V CONTRAST MEDIUM	FREQUENCY	PERCENTAGE
USED	207	(42.8)
NOT USED	277	(57.2)
TOTAL	484	(100%)

TABLE 18(ii) RESULTS OF INTRAVENOUS (IV) CONTRAST MEDIUM USE DURING CT SCAN EXAMINATION FOR EPILEPSY

I.V CONTRAST MEDIUM	FREQUENCY	PERCENTAGE
NOT USED	277	(57.2)
USEFUL	96	(19.8)
NOT USEFUL	111	(22.9)
TOTAL	484	(100%)

TABLE 19: COMPARISON OF COMPUTED TOMOGRAPHY RESULTS WITH THE AGE OF THE PATIENT (BRAIN TUMOURS)

RESULTS OF CT SCAN	0-1 yrs M		11- yrs M	-	21- yrs M		31- yrs M		46-65 yrs M F	У	65 rs F	UNS CIF		TOTAL	%
GLIOMAS a) UNSPECIFIED CYSTIC b) CEREBRAL ASTROCYTOMA C) CEREBELLAR ASTROCYTOMA d) MEDULLOBLASTOMA	1 1	1	1 1	1 2	1	1	1 2		1	1	-	2	2	12 7 1 1	31.6% 18.4% 2.6% 2.6%
MENINGIOMAS	1			2		1	1	1	1	_	1	1	1	10	26.3%
PITUITARY TUMOURS	-		1		1		1		1	_	_	_	_	4	10.5%
CRANIOPHARYNGIOMA		-	-	-	1		_	_		- -	_	_	-	1	2.6%
BLOOD VESSEL TUMOURS ANGIOMA							1							1	2.6%
DERMOID CYST												1		1	2.6%
¹ TOTAL	3	2	4	5	3	2	6	1	3	1	. 1	4	3	38	
; % TOTAL M	7.9 5.3		10. 13.		7.9 5.3		15. 2	. 6%	7.9%		2.6%	10. 7.	. 5% . 9%		100%



CASE NO.29: Figure 1:

A $2^1/j$ year old male child with a 2 months history of post meningitic generalized convulsive seizures, inability to talk, abdominal pains^ opisthotonos but no fever.

Plain skull radiograph revealed osteomyelitic changes of the right parietal bone.

Brain CT scan done on 2/3/94 showed multiple cerebral abscesses in the right temporo-parietal and occipital lobes with surrounding oedema. Small abscesses were seen at the left frontal lobe and right parietal osseous destruction. Emergency surgery was done on 3/3/94 and 100 mis of pus was drained through Burr holes. CSF culture was negative.

Diagnosis: Multiple brain abscesses.

Figure 2:

An RO year old male patient with focal seizures and left hemiparesis.

Brain CT scan done on 28/4/93 showed a fairly well defined mass of mixed attenuation on the right fronto-parieta] lobes compressing the ipsilateral frontal horn of the lateral ventricle, there was some enhancement on intravenous contrast medium.

Diagnosis: Glioma with cerebral atrophy.

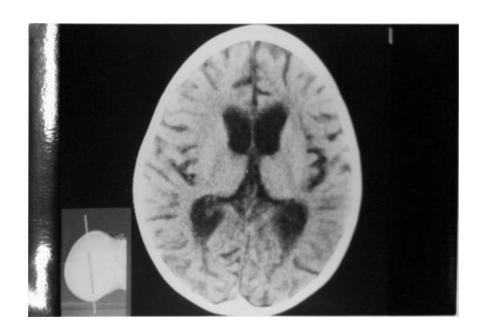


Figure 3:

A 3 year old male child with generalized seizures following meningitis on 20/9/93. The child also developed generalized hypereflexia and decorticate rigidity. He lost consciousness for two days, on 22/9/93. Brain CT scan done on 15/10/93 revealed prominent sulci in the cerebral cortex and in the cerebellum. There was compensatory enlargement of the ventricles and basal cisterns.

Diagnosis: ••^••cerebral and cerebellar atrophy.



Figure 4:

A $3^1/\ll$ year old male child with a history of a fall in June 1993. He developed sunset eyes, head enlargement and generalized convulsions for 2 months. He was treated for meningitis in July 1993.

EEG - indicated a left hemispheric pathologic process. Brain CT scan done on 17/5/94 showed a large left chronic subdural haematoma; compressing the ipsilateral ventricular system, plus a small right frontal chronic subdural haematoma. He had emergency surgery on 20/5/94 and 150 mis of old organised blood was evacuated, through Burr holes. He made a good recovery.

Diagnosis Large left chronic subdural haematoma

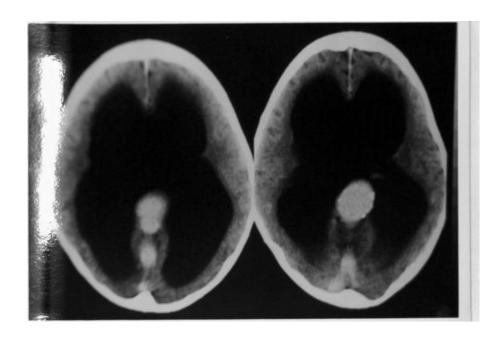


Figure 5

A 7 year old male child with a 10 months history of poor vision, sun set eyes and enlarged head. He also complained of headaches, vomiting, and generalized tonic chronic seizures for 3 months.

EEG indicated a right frontal pathology. Plain skull radiographs showed copper beaten appearance with minimal sutural diastasis.

Brain CT scan on 29/11/94 showed a well defined area of high attenuation with calcification in the region of the 3rd ventricle, with dilatation of the ventricular system. A second area of high attenuation on the interhemispheric fissure with contrast enhancement was seen (2° seeding). Median pressure VP shunt was inserted on 14/12/94.

Di^qnosisflPineal tumour with secondary seeding (or) Meningioma

2) Obstructive Hydrocephalus

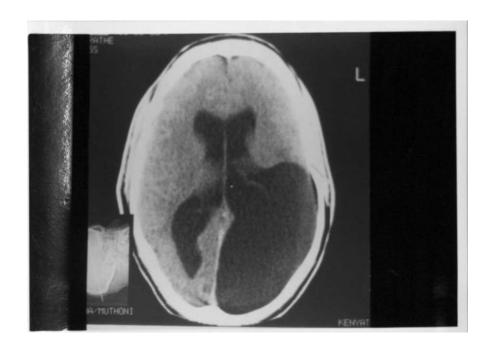


Figure 6:

A 28 year old male patient with poorly controlled seizures since June 1978, with a history of birth trauma. He presented with left sided headaches, 5th cranial nerve palsy, and right hemiparesis

EEG: indicated an epileptogenic focus over the left fronto-temporal area. Brain CT scan done on 6/12/94 showed a well defined area of reduced attenuation on the left parieto-occipital lobes communicating with the occipital horn of the left ventricle.

Diagnosis Porencephalic cyst.

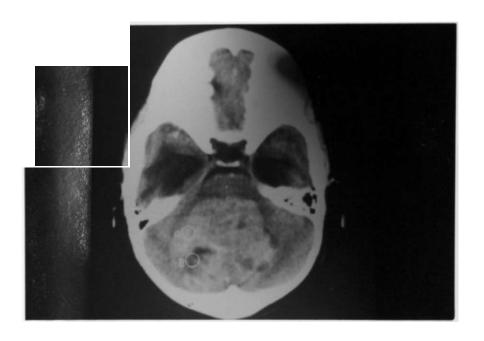


Figure 7:

A 5 year old female patient with a one weeks history of generalized convulsions and no fever.

Brain CT scan done on 11/4/95 showed a well defined area of mixed attenuation in the cerebellum compressing the 4th ventricle, with accompanying dilatation of the lateral and third ventricles. The mass enhanced with intravenous contrast, medium.

<u>Diag</u>nosis Medulloblastoma with obstructive hydrocephalus Differential Diagnosis cerebellar astrocytoma.

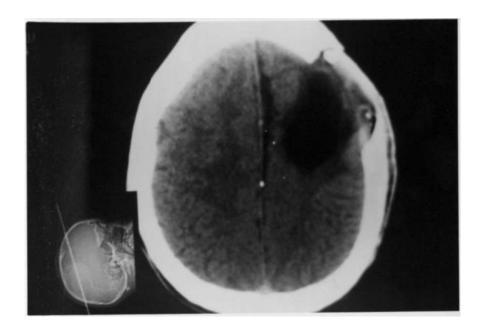


Figure 8

A 19 year old male patient with a history of assault (with an axe) sustained severe head injury. He developed generalized seizures and multiple brain abscesses. These were drained on 8/8/93. Subsequently he had surgical elevation of the bone fragments at the left fronto-parietal region due to a compound fracture. Three bone fragments were removed. Brain herniation with irreparable dural tear were noted.

CT scan on 2/1/96 revealed a post surgical bone defect at the left fronto-parietal region and extension of the anterior horn of the left lateral ventricle.

Diagnosis Post traumatic porencephalic cyst.

Figure 9

A 35 year old male patient with a 4 months history of focal seizures involving the right side of the body.

Brain CT scan done on 26/2/96 revealed a large well rounded high attenuation lesion at the left parieto-central region with surrounding oedema and areas of necrosis and midline shift to the right side. There was contrast enhancement of necrotic areas.

Diagnosis; Falx meningioma

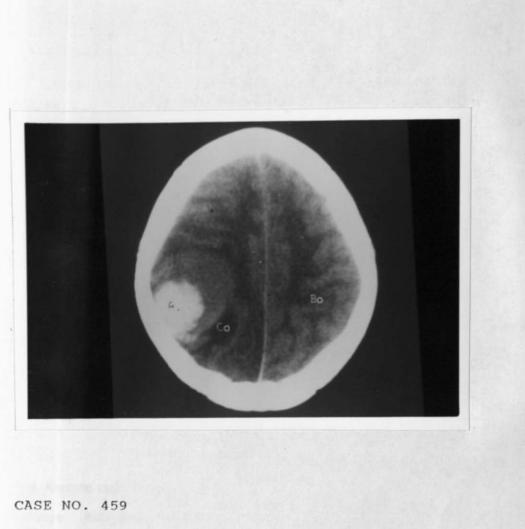


Figure 10:

A 14 year old female patient with generalized convulsions, right sided weakness, dizziness, vomiting and neck stiffness.

Brain CT scan done on 18/3/96 showed an area of varying attenuation, on the right parietal lobe compressing and displacing the ventricular system plus accompanying oedema.

Diagnosis: Meningioma and 2° haemorrhage.

CHAPTER 9

DISCUSSION

Epilepsy is one of the commonest neurological disorder affecting mankind (39). The world prevalence rate of epilepsy is about 4/1000 (67,76). The prevalency rate of epilepsy in developing countries especially in Africa is higher than in the developed countries (13,88). The highest prevalency rate of epilepsy in the world has been reported in some African communities e.g. 20/1000 in the Wapogoro tribesmen of Tanzania and 50/1000 in the Basasa and Kipelle tribesmen of Liberia (76,88) (see Table 3). The lowest prevalence rate has been in Japan 1.5/1000 (see Table 1) (76). Epilepsy affects all age groups and cuts across the social divide (76).

In Kenya epilepsy is a common neurological disorder affecting about 500,000 people. (76). It is the second commonest neurological disorder at Kenyatta National Hospital after infections (77). The prevalency rate of epilepsy in Kenya is high 16.7 - 18.5/1000 (76,77,95).

The peak age for epileptic patients is 10-19 years (11,13). In Kenya 75% of epileptic patients are below 25 years of age. (76). This study obtained a figure of 64.4% after adjusting for unspecified ages.

There is a preponderance of epilepsy in males compared to females (11, 12, 13, 29, 49, 67). This study had a male to female ratio of 1.4:1. Partial seizures are more likely to have underlying structural problems, such as brain tumours, scars, infections, cerebrovascular accidents or as a consequence of trauma (76).

The diagnosis of epilepsy is based on clinical presentation. The role of electroencephalography (EEG) is restricted to classifying the seizure disorder, localising the epileptogenic source and in guiding prognosis. (47,77). The value of computerized tomography of the brain in detecting structural abnormalities in epileptic patients is well documented, (33,36,90).

Computerized tomography of the brain is however limited by beam hardening artefacts in detecting certain abnormalities of the brain which cause epilepsy . eg. mesial temporal sclerosis (MTS), and small neuronal migrational anomalies (51, 55, 72, 73, 80, 83, 84). Mesial temporal sclerosis arises in infancy due to prolonged febrile convulsions. It becomes a potent epileptogenic lesion (21).

It is well detected by magnetic resonance imaging (MRT), and physiological neuroimaging ie. (single photon emission computerized tomography (SPECT), and positron emission

tomography) (PET). (80, 84).

AGE/SEX

Epilepsy affects all age groups. In this study the age range was from 1 month to 86 years. The male infant aged one month had a normal CT scan. The 86 year old patient had a hpad injury and diffuse brain atrophy. There was also a female infant aged two months with brain abscess on CT scan. In this study the highest number of patients were below 15 years of age i.e. 195 (40.3%) of the total number of patients. This compares well with the figure of 46% by Matuja et al in Tanzania (65).

There was a large number of patients below the age of 5 years with epilepsy i.e. 97(20.0%). The male to female ratio was 1.5: 1 for patients below 5 years of age.

The total number of males in the study was 283 cases (58.5%) and that of females was 201 cases (41.5%) giving an average male to female ratio in the study sample of 1.4:1 (see table 9). This compares well with the study by Kwasa et al with a male to female ratio of 1.5:1 (78). Dada in Nigeria in 1970 found a male to female ratio of 1.5:1 in the 11-20 year age group (13).

There was a gradual decrease in the number of patients aged 31 years and above (Table 8). There was a total of 27 patients (5.8%) between 31 and 35 years of age (males and females), and 10 patients (2.0%) between 61 and 65 years of age. There were only 5 patients (1.0%) between 71 and 75 years of age and only 5 patients (1.0%) aged 80 years and above.

Presenting symptoms and signs

The commonest presenting symptom was convulsion either focal or generalized. All 484 patients in the study had different seizure types (Table 13 and 17). The majority of patients 395 (81.6%) had generalized convulsive seizures (GCS). Patients who had simple partial seizures (SPS) were 51 (10.5%), and those with partial seizures secondarily generalized (PSG) were 17 (3.5%). Patients with partial complex seizures (PCS) were 11 (2.3%) (Table 13). The tendency to eguate partial complex seizures with temporal lobe epilepsy is not correct (49,65,67,71).

It is noteworthy that although patients with generalized convulsive seizures were the majority, 395 (81.6%), the percentage of abnormal CT scan findings were only 57.1%. Patients with simple partial seizures were only 51 but the percentage of positive or abnormal CT scan findings were 66.6%. Patients with partial seizures secondarily generalized had 58.8% positive or abnormal CT scan findings, and those

with partial complex seizures had 18.2% positive or abnormal CT scan findings. McGahan found positive or abnormal CT scans in (71%) of those patients who had partial seizures secondarily generalized (33, 36) (Table 17). Osuntokun et al in Nigeria in 1970 found 377 (72.2%) out of 522 patients with grand mal epilepsy with or withour aura (13).

The second presenting symptom was headache with 39 cases (8.1%). Decreased level of consciousness third with 36 cases (7.4%). Visual disturbance and ataxia had 17 (3.5%), and 12 (2.5%) cases respectively (Table 11). The commonest clinical findings were weakness of limbs (with 71 (14.7%) cases. Left sided weakness was seen in 44 (9.1%) cases and was more common than right sided weakness 27 (5.6%), cranial nerve palsies, and dysphasia each had 8 cases (1.7%) (Table 11 and 12).

The highest number of abnormal CT scans findings were in the group with less than 6 months duration of seizures i.e. 57 cases (65.5%). This was followed by the group with a duration of seizures of 1-5 years which had 20 cases (48.8%) with abnormal CT scan findings. Patients with seizures of greater than 5 years duration were .12 or had an abnormality of (40.0%). McGahan found 51% abnormality in patients with less than 6 months duration of seizures (i.e 23 out of 45 patients) and 60% abnormality (3 of 5) in patients with a duration of 6 months - 1 year (33).

Computeri2 ed tomography (CT) results

Normal CT results

A total of 211 (43.6%) patients had normal or negative CT scan findings. Of the 211 (43.6%) patients males were 116 (23.6%) and females were 95 (19.6%) (Tables 14 and 15).

Computerized tomography results (General considerstions)

Patients below the age of 10 years who had abnormal CT scan findings were 90 (90 of 133) or (67.7%). Patients between 11 and 20 years who had abnormal CT scan findings were 47 (47 of 112) or (42.0%). Likewise patients between 21 and 30 years of age who had abnormal CT scan findings were 31 (31 of 67) or 46.3%.

Patients with the highest abnormal CT scan findings were those above 65 years of age (15 of 18) or (83.3%). Patients between 31 to 45 years of age had 61.8% abnormal CT scan findings and those between 46 and 65 years of age had 67.6% abnormal CT scan findings (Table 15).

These abnormal or positive CT scan findings compare well with those of McGahan at the University of California (Davis Medical Centre) who found 86% positive or abnormal CT scan findings in patients above 65 years of age. The study by McGahan also found 57% abnormal CT scan findings in patients between 46 - 65 years of age (33,36).

McGahan had no significant difference between the age groups 10 to 25 years, who had 35% positive or abnormal CT scan findings. This agrees with findings of this study of 42.0* positive or abnormal CT findings between the age group 11 to 20 years and 46% positive or abnormal CT scan findings in the age group between 21-30 years (33, 36. Table 15). The results in this study contrasts sharply with those of McGahan who found positive or abnormal CT scan findings in only 12% (2 of 17 patients) who were in the age group below 10 years. In this study positive or abnormal CT scan findings were 67.7% (90 of 133) for patients in the age group below 10 years. (Table 15) (33,36).

This study shows that epilepsy in our set up is a disease affecting young people mainly those below 20 years of age and particularly those below 10 years of age. It is noteworthy that the majority of children affected are below 5 years of age (Table 10). Patients who had febrile convulsions were excluded from the study. A male preponderance was noted in this study. This has been reported in other studies (11,12,13,29,49,67).

There were 32 patients (children and adults) who had "acute provoked seizures" (i.e. seizures due to a short term direct response to an acute event resulting in brain insult (84). Patients with alcohol withdrawal seizures, convulsions due to

intravenous injections of drugs, acute head trauma with diffuse cerebral oedema and deranged metabolic causes e.g. uraemia were also part of those with acute provoked seizures and were also excluded from the study (84).

Cerebral atrophy:

This was the commonest abnormal CT scan finding. There were two types of brain atrophy seen, diffuse and focal.

There were 94 cases (94 out of 132) or (71.2%) of diffuse brain atrophy only. Another nine cases of diffuse brain atrophy were found coexisting with other pathological findings. There were 38 cases (38 out of 132) or (28.8%) with focal brain atrophy. Another 5 patients were found having focal brain atrophy coexisting with other pathological findings. These 14 cases were not purely focal or diffuse brain atrophy.

There was a preponderance of males compared to females in patients with either diffuse or focal brain atrophy. In diffuse brain atrophy males were 55 (55 out of 94), or (58.5%) compared to females who were 39 (39 out 94) or (41.5%). In focal brain atrophy male patients were 21 (55.3%) compared to female patients who were 17 (17 out of 38) or (44.7%) (Table 15). McGahan et al in California in their study found 54% cases of diffuse brain atrophy and 46% cases of focal brain atrophy (33, 36). Longe found 67 (47.2%) out of 142 patients

had cerebral atrophy (90). Young et al found 16 (10.8*) of 52 patients with CT abnormalities had brain atrophy (41).

The highest number of patients with combined diffuse and focal brain atrophy were in the age group below 10 years (males and females) with 49 cases (37.1%). The lowest number of cases were found in the age groups between 21-30 years, with 9 cases (6.8%) out of a total of 132 patients. Brain atrophy was found to decrease with age in the older age groups (Table 1*5). Brain Tumours

The commonest tumours encountered in the study were primary brain tumours. The predominant type of these tumours were gliomas. There were 21 cases of gliomas, (21 of 38) or 55.1%.

Amongst the gliomas, there were 12 cases of unspecified cystic gliomas (12 of 21) or (57.1%). There were 7 cases of cerebral astrocytoma (7 of 21) or (33.3%) of the gliomas. There was one case of cerebellar astrocytoma (1 of 21) or (4.8%) of gliomas. Another type of glioma seen was medulloblastoma which had 1 case (1 of 21) or (4.8%). Osuntokun in Nigeria in 1970 found 19 cases (3.6%) of brain tumours (5 gliomas, 11 meningiomas, two metastases, and one retinoblastoma) out of 522 patients (13).

The second commonest type of primary brain tumours were meningiomas with 10 cases (10 of 38) or (26.3%). McGahan

found only 6 cases (4.0%) of neoplasms out of 150 patients (33).

Other primary tumours that were encountered in the study were 4 cases of pituitary tumours (4 of 38) or (10.5%) and 1 case of craniopharyngioma (1 of 38) or 2.6%).

There was also 1 case of angioma (2.6%) and 1 case of dermoid cyst (2.6%). Contrast enhancement was found useful in the case of some of the tumours e.g. meningiomas, some gliomas, angiomas, etc. (Table 19).

Infections

Infections with 29 cases (6.0%) were the 3rd commonest cause of convulsions. Top of the list of infections were brain abscesses with 17 cases (17 of 29) or (58.6%), 13 of which presented with generalized convulsions. This was followed by 5 cases (5 of 29) (17.2%) of tuberculomas and 8 cases (8 of 29) or (27.6%) of granuloma (Table 17).

Head Trauma

The head trauma cases were 24 or (5.0%) of the total number of patients in the study. These included 7 patients (7 of 24) or (29.2%) with chronic subdural haematoma, and 6 patients (6 of 24) or (25.0%) with intracerebral bleed.

There were three cases each or (12.5%) of acute subdural haematoma and extradural haematoma. There was only one case

Arteriovenous malformations (A-V malformations)

There were only 5 cases of A-V malformations or (1.0%) of th*> total number of patients.

Two of these cases were found in male patients in the agp group 21-30 years old. Two of the cases were in the age unspecified group. The other case was a female patient in thp age group of 11-20 years. Young et al found 3 cases (5.8%) of A-V malformations out of 52 patients with CT scan abnormalities (43). Longe found 2 cases (2.3%) of 86 patients with CT abnormalities had A-V malformations (90).

Three of the cases of A-V malformations presented with partial seizures. One case presented with generalized convulsions. There was contrast enhancement in all the cases of A-V malformations (Table 15 and 17).

Intravenous contrast medium findings

During CT scans of the brain, intravenous contrast medium was given to a total of 207 patients (42.8%). Contrast medium was found to be useful in only 96 cases (19.8%) i.e. these patients had contrast enhancement of their abnormal CT scan findings. Cases of brain abscesses were better visualised compared to pre-contrast enhancement films. Tumours like A-V malformations, craniopharyngiomas, and meningiomas also had



contrast enhancement compared to pre-contrast scans. Contrast media use did not add any further information from what was found in pre-contrast scans in 111 (22.9%) of cases. Longe et al (90) found out that contrast enhancement did not improvp the interpretation of the CT scan examinations except in patients with mass effect on pre-contrast scans.

Surgical cases

In this study there were 96 patients who retired surgery either for evacuation of haematomas, draining of cerebral abscesses, insertion of ventriculo-peritoneal shunts (V-P shunts for hydrocephalus or surgical excision of tumours. This is a low yield of surgically treatable cases. Loncpfpt all reported a low yield in Saudi Arabia 11 cases (8%) out of 142 patients, with surgically treatable cases (90). This study found 96 out of 273 patients (35.2%) with CT abnormalities required surgical intervention.

Young et al in Britain reported 15 (28.8%) cases out of 52 patients with CT abnormalities in a group of 220 epileptics (43).

McGahan in California, United States of America (U.S.A.) found 5 cases of hydrocephalus, 6 cases of neoplasms and 6 cases of vascular abnormalities i.e. (28.3%) of 60 patients with CT abnormalities out of 150 patients (33,36).

CONCLUSIONS

Computerized tomography (CT) examination of the head 1b a simple, guick non invasive method of screening and definitive evaluation of seizure patients. There are limitations due to beam hardening artefacts especially in the temporal lobe type of epilepsy. This type of epilepsy is caused by mesial temporal sclerosis (MTS) and hippocampal atrophy. CT scan examination is limited in the detection of neuronal loss and gliosis which is found in the temporal lobe, in the case of mesial temporal sclerosis. CT scan has limitations also in detecting heterotopic grey matter (cortical neurons in abnormal locations) and cases of neuronal migrational abnormalities of the cortical mantle.

Despite these limitations computerized tomography detection rate of intracranial lesions is high. The overall detection rate of intracranial lesions is highest in patients with seizures of short duration (less than six months). The detection rate of intracranial lesions is also high in old patients who develop seizures especially above 65 years of age. Patients who have simple partial seizures, complex partial seizures or partial seizures secondarily generalized are more likely to have abnormal CT scans.

There are many patients with generalized convulsive seizures. These patients have a lower yield of abnormal CT scans of the brain. Patients with this type of seizure are also well controlled medically. Patients with intracranial space occupying lesions are poorly controlled medically. Tt is hoped that now with the recent installation of two magnetic resonance imaging (MRI) machines in Nairobi, those patients who have normal CT scans of the brain and who have medically intractable epilepsy (« 2 years duration) will benefit from this mode of investigation.

RECOMMENDATIONS

Computerized tomography (CT) is of extreme value in the screening and evaluation of epileptic patients.

It is an expensive method and should only be used for the maximum benefit of the patient with epilepsy who might get a positive or abnormal CT scan finding.

Attempts to use it as a screening procedure should be avoided because this will mean denying and delaying other needy patients the chance to benefit from its use. Careful clinical assessment supported by an electroencephalogram remain of paramount importance in the management of epilepsy. The following criteria should be used in deciding which patients should undergo computerized tomography examination due to epilepsy.

FOR ADULTS

- Patients with a short duration of seizures (less than 6 months) have a high probability for CT scan abnormalities.
- Older patients have a higher number of abnormal CT scans of the brain, particularly if they are over 65 years of age. (Greater than 80% of cases have abnormal CT scan findings).
- 3. Patients with positive neurological findings have high probability of CT scan abnormalities (> 65% of cases have

- abnormal CT scan examinations).
- 4. Patients with focal seizures (simple partial seizures) also have a high (> 70% of cases) abnormal CT scan findings)
- 5. Patients with generalized convulsive disorders have the lowest probability of abnormal CT scan examinations.

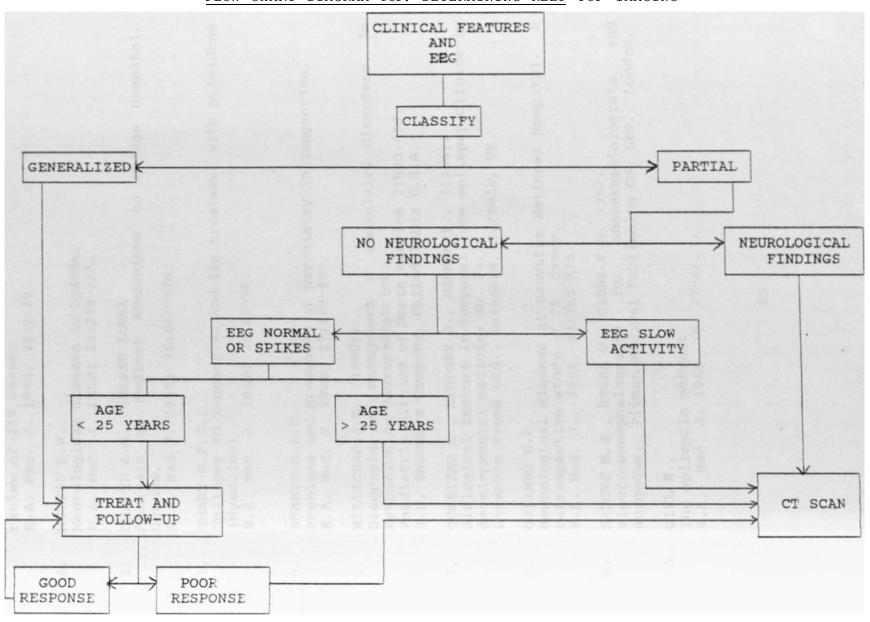
 These patients are usually the majority in many studies.
- 6. Contrast enhancement during CT scan examinations should be used judiciously in patients whose pre-contrast films suggest intracranial pathology. In patients with brain abscesses, A-V malformations, meningiomas, craniopharyngiomas or sturge Weber Syndrome contrast enhancement can be put to good use.
- 7. Patients with medically intractable epilepsy (or drug resistance epilepsy) with a normal CT scan of the brain should be assessed with a magnetic resonance imaging machine (MRI).

Cr<u>iteria for selecting children wi</u>th epilepsy for CT scan examination

of age (special sensory, autonomic, psychic, and memory disturbance). These children have complex partial seizures which may involve the temporal or frontal lobes. This includes children with Infantile spasms (West syndrome, Lennox-Gastaut syndrome).

- About 40% ot abnormality is detected with CT scan in these children. Children should be CT scanned after the age of six months to maximise the diagnostic yield.
- 2. Epilepsy in children who have a changed seizure pattern, from a previous pattern on ERG exmaination.
- Epilepsy in children with focal EEG, changes particularly with focal slowing.
- 4. Epilepsy with seizures intractable or resistant to medical therapy especially where surgery is contemplated.
- 5. Status epilepticus in children (although the diagnostic yield on CT scan is low or rare).
- 6. Post traumatic epilepsy.

FLOW CHART DIAGRAM FOP. DETERMINING KEEP FOF IMAGING



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Appendix I

THE ROLE OF <u>COMPUTERIZED</u> TOMOGRAPHY IN THE DIAGNOSIS AND MANAGEMENT OF <u>EPILEPSY</u> A_STUDY AT KENYATTA NATIONAL HOSPITAL

(A referral and teaching hospital)

	DATA_COLLECTION	SHEET
NAME SURNAME (Surname) SEX. DATE. INPATIENT/OUTPATIENT WARD/CLINIC. R X-RAY DEPARTMENT RE INTRAVENOUS CONTRAS COMPUTERIZED TOMOGR. CLINICAL HISTORY SEIZURE PATTERN DURATION OF SEIZURE ELECTROENCEPHALOGRAM	CASE NO T NO EFERRED FROM . GISTRATION NO. T MEDIUM APHY SCAN DATE	FIRST (First)
PROVISIONAL DIAGNOS a) Congenital less b) Mass lesions (c) Inflammatory 1 d) Post-traumatic e) Vascular lesio f) Others	ions (brain) . (Brain) esions (Brain)	
RADIOLOGICAL DIAGNO (COMPUTERIZED TOMOG		
FINAL DIAGNOSIS (COMPUTERIZED TOMOG	RAPHY REPORT)	
FINAL DIAGNOSIS (HI	STOPATHOLOGY/POS	T MORTEM)
DOCTOR'S NAME		DATE

CAUSES OF LATE CHILDHOOD AND ADULT ONSET SEIZURES (Adopts fmm '.9)

- a) <u>Specific epileptic syndromes</u>: (1) Primary generalized epilepsy.
 - (?) Secondary generalized epilepsy.
- b) Genetic/hereditary diseases: (1) Sinple p.ene disorders
 - (2) Chromosomal disorders
 - (3) Hereditary malformation
- <") Perinatal damage: (1) Birth trauma
 - (2) Hypoxia
 - (3) Haemorrhage
 - CO -Prematurity
- d) <u>Cerebral Infection</u>: (i) Bacterial (ii) Viral (iii) Parasitic (iv) Spirochaete (Syphilitic)
 - 1) Meningitis
 - 2) Encephalitis
 - 3) Cerebral abscess
 - U) Subdural or epidural abscess
 - 5) Intracerebral granuloma
- °) Cerebral trauma:
 - 1) Open head injury
 - 2) Closed head injury
 - 3) Neurosurgery
- f) Cerebral Vascular disease:
 - 1) Haemorrhage (a) Extradural (b) Subdural
 - (c) Intracranial (d) Subarachnoid
 - 2) Infarction (embolic or thrombotic)
 - 3) Arteriovenous malformation
 - 4) Hypertensive vascular disease
 - 5) Degenerative vascular disease
 - 6) Venous thrombosis
 - 7) Cortical thrombophlebitis

Appendix 2 contd.

g) Intracranial tumour:

- 1) Primary cerebral tumours
- 2) Secondary cerebral tumours (metastases)
- 3) Intracranial tumours
- 'l) Extracerebral tumours

li) Cerebral degenerative diseases:

- 1) Most grey matter degenerative diseases
- 2) Many white matter degenerative diseases.

i) Metablic and Systemic diseases:

- Disturbances of serum electrolyte and acid base balance
- 2) Renal diseases
- 3) Hepatic diseases
- 14) Haematologic diseases
- 5) Endocrine diseases
- fi) Cardiorespiratory diseases
- 7) Nutritional diseases

i) Toxic causes and Poisoning

- 1) Poisons
- 7) Alcohol
- 3) Drug abuse
- •0 Narcotics
- y) Iatrogenic: (see Table 17)
 - Drug withdrawal eg. anticonvulsants, alcohol, narcotics.
 - ?) Intravenous drugs eg. lidocaine to control vetricular tachycardia

1) Miscellaneous conditions

- 1) Eclampsia
- ?) "Chronic encephalitis"
- 3) Multiple sclerosis

Appendix 3

The causes of Seizures Adopted from (86) 199M

Age given in years

infant (n-2) - rerina^l hypoxia and rsohaemia

- Intracranial birth injury
- Acute Infections
- Metabolic disturbances (hypoglycaemia, hypocalcaemia, hypocaloaemia, hypomagnesemia, pyridoxine deficiency
- Congenital malformation
- Genetic disorders

Child 2-12 - Idiopathic

- Acute infection
- Trauma
- Febrile convulsion

Adolescent - Idiopathic

- Trauma
- Drug, alcohol withdrawal
- Arterio venous malformations

Young Adult Trauma

18-35 Alcoholism

Brain tumour

Older Adult - Brain tumour

(35) - Cerebrovascular disease

- Metabolic disorders (uraemic, hepatic failure) electrolyte abnormality, hypoglycaemia
- Alcoholism



APPENDIX 'I

Causes of Brain atrophy (Adopted fmm lrmj>e/9n)

- 1. Infections in children
- 2. Drugs
- 3. Alcohol
- M. Aging
- 5. Trauma
- £. Rr^in infarcts
- 7. Hypoxic Ischaemic insults
- ft. Degenerative diseases
- 9. Toxic enoephalo pathies
- 10. Drug related cerebellar atrophy caused by antiepileptic drugs (I-onge 90)