# Chapter one

#### 1.1 Introduction

Epilepsy is defined as the repeated occurrence of sudden, excessive and/or synchronous discharges in cerebral cortical neurons resulting in disruption of consciousness, disturbance of sensation, movements, impairment of mental function, or some combination of these signs. Because of their sudden nature, seizures are called ictal events, from the Latin ictus meaning 'to strike'. The terms epilepsy, seizure and convulsion are not synonymous. A seizure always is a symptom of abnormal function in the central nervous system (CNS) rather than disease in itself. A seizure discharge may be initiated in an entirely normal cerebral cortex by a variety of acute insults, such as withdrawal from alcohol, low blood sodium, or certain toxins. Seizures are to be distinguished from epilepsy, which is a chronic condition in which seizures occur repeatedly due to an underlying brain abnormality which persists between seizures. A convulsion is a forceful involuntary contraction of skeletal muscles. A convulsion is a physical manifestation of a seizure, but the term is inappropriate as a synonym for epilepsy when epilepsy may consist only of a temporary alteration of consciousness or sensation. Epilepsy occurs in approximately 0.7% of the population at any one time. More than two-thirds of seizure problems begin in childhood, with a second peak of onset in the elderly. Usually, epilepsy does not significantly alter life expectancy, but quality of life may be seriously compromised when seizures are not satisfactorily managed. Epilepsy has many causes, but in most patients a cause cannot be identified. Among the pathologies most commonly considered to give rise to epilepsy are cerebro vascular lesions, perinatal or postnatal trauma, infections of the CNS, and tumours or congenital malformations of the brain (James Bowman, et-al, 2001).

Electrical signals at an excessively high rate and/or in an abnormal pattern. An epileptic seizure can originate only in certain structures of the brain but theseizure may then spread to other structures of the CNS (e.g. the basal ganglia). Once a patient has developed epilepsy, individual seizures may be precipitated by number of conditions a circumstance (James Bowman,et-al ,2001).

Epilepsy is a momentary imbalance within electrical and chemical circuits in the brain, such that groups of brain cells act in excessive fashion. This may Create a temporary disturbance in the way the brain controls awareness andresponsiveness and cause unusual sensations or abnormal movements and Postures (James Bowman, et-al ,2001).

Seizures are generally described in two major groups, primary generalized seizures and partial seizures. The difference between these types is in how and where they begin. A new way of naming seizures has been developed by epilepsy specialists, but most often these common names are still used.

Special test are performed in many children with epilepsy such as to confirm a clinical suspicion. In patients with epileptic seizures a recording of the brain wave activity (EEG) and a picture of brain MRI are obtained. (James Bowman,et-al, 2001)

#### 1.2 Problems of the Study:-

Some result of magnetic resonance imaging was normal inspite of clinical finding of epilepsy.

#### 1.3 Objectives of the Study:-

# 1.3.1 General objective:-

To evaluate MRI and clinical findings in diagnosing epilepsy.

# 1.3.2 Specific objective:-

- -To determine the most age group affected by seizures diagnosed by MRI.
- -To determine the underlying cause of epilepsy.
- -To correlate the different pathology cased epilepsy with the clinical findings.

# 1.4 Over view of the study

Chapter one:-Introduction.

Chapter Two:-literature review (Theoretical Background and Previous Studies)

Chapter Three:-Material and Method.

Chapter Four: - Results

Chapter Five:-Discussion, Conclusion and Recommendations

References

Appendices

# Chapter two

#### 2.1 Theoretical Background

### 2.1.1Anatomy of the Brain:-

The brain is delicate organ that is surrounded and protected by three membranes called meninges .the outermost membrane, the duramater (tough mother), is the toughest. This double-layered membrane is continuous with the perosteum of the cranium between the layers of the dura mater are the meningeal arteries and the dural sinuses (Richards. Snell. 2005).

### **2.1.1.1** The Meninges

The brain in the skull is surrounded by three protective membranes, or meninges: the dura mater, the arachnoid mater, and the pia mater (Richard S .Snell. 2012).

#### 2.1.1.2 Dura Mater of the Brain

The dura mater is conventionally described as two layers: the endosteal layer and the meningeal layer (Fig.2-1) (Richard S. Snell. 2012).

Dural Nerve Supply Branches of the trigeminal, vagus, and first three cervical nerves and branches from the sympathetic system pass to the dura. Numerous sensory endings are in the dura. The dura is sensitive to stretching, which produces the sensation of headache. Stimulation of the sensory endings of the trigeminal nerve above the level of the tentorium (Richard S .Snell. 2012).

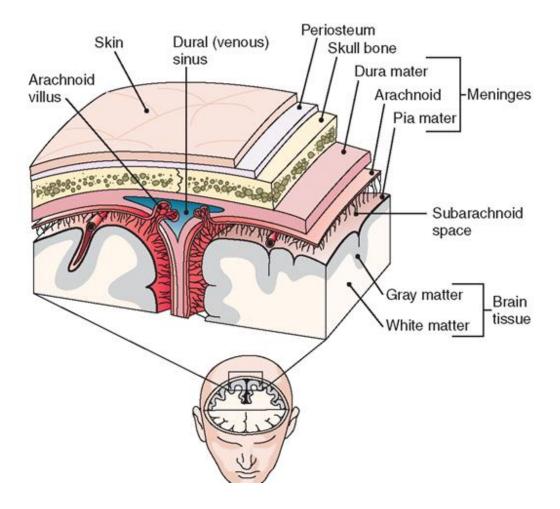


Figure (2.1) shows Coronal section of the upper part of the head the sinuses and the matter of the brain.

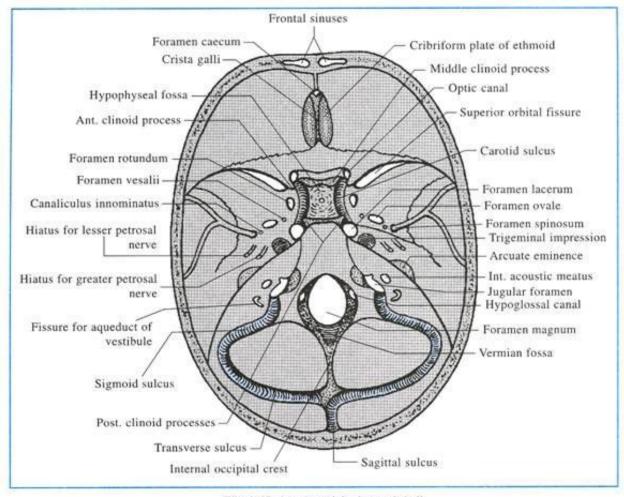


Fig. 1.45. Interior of the base of skull.

Figure (2.2) Interior of the skull

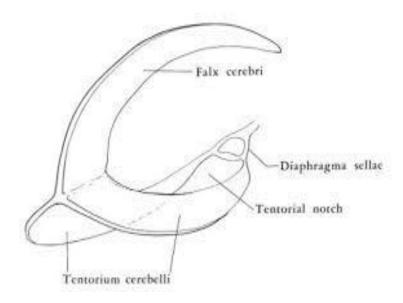


Figure (2.3) shows Diaphragma sellae and tentorium cerebelli.

Figure 17-8c Location and circulatory pattern of cerebrospinal fluid.

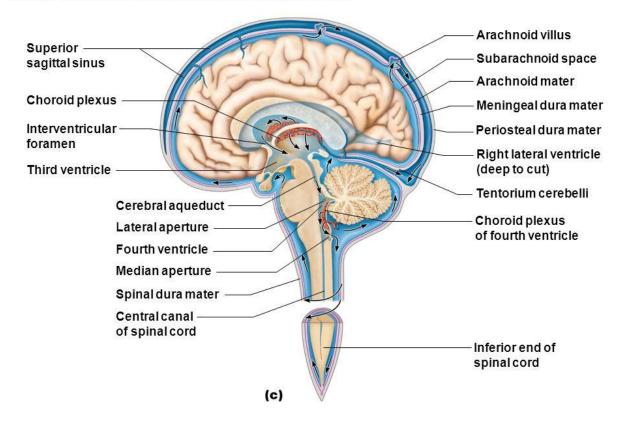


Figure (2.4) show Lateral view of the brain, the falxcerebri, tentorium cerebelli, brainstem.

## 2-1-1-3Dural blood supply:-

Numerous arteries supply the dura mater from the internal carotid, maxillary, ascending pharyngeal, occipital, and vertebral arteries. The venous drainage the meningeal veins lie in the endosteal layer of dura. The middle meningeal vein follows the branches of the middle meningeal artery and drains into the ptery/goid venous plexus or the sphenoparietal sinus. The veins lie lateral to the arteries (Richard S. Snell. 2012).

### 2-1-1-4Pia Mater of the Brain

The pia mater is a vascular membrane that closely invests the brain, covering the gyri and descending into the deepest sulci (Fig. 2.1)(Richard S .Snell. 2012).

#### 2-1-1-5 the Venous Blood Sinuses

Situated between the layers of the dura mater; they are lined by endothelium. Their walls are thick and composed of fibrous tissue; they have no muscular tissue. The sinuses have no valves. They receive tributaries from the brain, the diploë of the skull, the orbit, and the internal ear. The superior sagittal sinus lies in the upper fixed border of the falxcerebri. It runs backward and becomes continuous with the right transverse sinus. The sinus communicates on each side with the venous lacunae. Numerous arachnoid villi and granulations project into the lacunae (Fig. 2.1)(Richard S. Snell. 2012).

The sigmoid sinuses are a direct continuation of the transverse sinuses. Each sinus turns downward behind the mastoid antrum of the temporal bone and then leaves the skull through the jugular foramen to become the internal jugular vein (Richard S .Snell. 2008).

The occipital sinus lies in the attached margin of the falxcerebelli. It communicates with the vertebral veins through the foramen magnum and the transverse sinuses (Richard S .Snell. 2012).

Important Structures Associated with the Cavernous Sinuses

The internal carotid artery and the 6th cranial nerve, which travel through it ,the lateral wall, the 3rd and 4th cranial nerves, and the ophthalmic and maxillary divisions of the 5th cranial nerve, The pituitary gland, which lies medially in the sellaturcica (Richard S .Snell. 2012).

The veins of the face, which are connected with the cavernous sinus via the facial vein and inferior ophthalmic vein, are an important route for the spread of infection from the face, the superior and inferior petrosal sinuses, which run along the upper and lower borders of the petrous part of the temporal bone (Fig. 2.2) (Richard S .Snell. 2012).

# 2.1.1.6 Pituitary Gland (Hypophysis Cerebri)

The pituitary gland is a small, oval structure attached to the undersurface of the brain by the infundibulum. The gland is well protected by virtue of its location in the sellaturcica of the sphenoid bone (Richard S .Snell. 2012).

#### 2.1.1.7Parts of the Brain

The brain is that part of the central nervous system that lies inside the cranial cavity. It is continuous with the spinal cord through the foramen magnum (Richard S. Snell. 2012).

#### 2.1.1.8Cerebrum:-

The cerebrum is the largest part of the brain and consists of two cerebral hemispheres connected by a mass of white matter called the corpus callosum. Each hemisphere extends from the frontal to the occipital bones the surface layer of each hemisphere is called the cortex and is composed of gray matter. The cerebral cortex is thrown into folds, or gyri, separated by fissures, or sulci (Richard S. Snell. 2012).

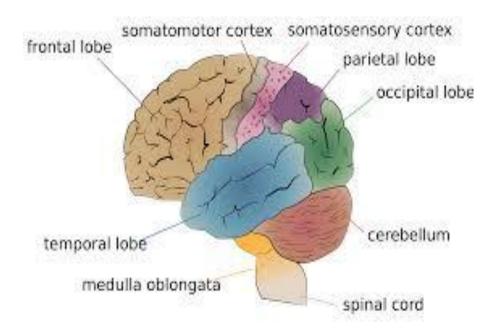


Figure (2.5) shows Right side of the brain showing some important localized areas of the cerebral function

#### 2-1-1-9 the frontal lobe:-

is situated in front of the central sulcus (Fig. 2.5) and above the lateral sulcus (Richard S .Snell. 2012).

# 2.1.1.10 The precentral gyrus

Lies immediately anterior to the central sulcus (Fig. 2.5). The large motor nerve cells in this area control voluntary movements on the opposite side of the body. Those controlling the movements of the face and hands in the lower part (Fig. 2.5) (Richard S .Snell. 2012).

## 2.1.1.11Thepostcentralgyrus

Lies immediately posterior to the central sulcus and is known as the sensory area (Fig. 2.5) (Richard S .Snell. 2012).

#### 2.1.1.12 The superior temporal gyrus

Lies immediately below the lateral sulcus (Fig. 2.5). The middle of this gyrus is concerned with the reception and interpretation of sound and is known as the auditory area.(Richard S .Snell. 2012).

#### **2.1.1.13Broca's area**,

Or the motor speech area, lies just above the lateral sulcus (Fig. 2.5). It controls the movements employed in speech (Richard S .Snell. 2012).

#### 2.1.1.14the visual area

Issituated on the posterior pole and medial aspect of the cerebral hemisphere in the region of the calcarine sulcus (Fig. 2.5) (Richard S .Snell. 2012).

# 2.1.1.15 Diencephalon:-

The diencephalon comprises the hypothalamus and thalamus. It is that part of the brain surrounding the 3rd ventricle (Harold ELIIS 2006).

#### **2.1.1.16Midbrain**

The midbrain is the shortest part of the brain stem; it is just under 1in (25mm) long and connects the pons and cerebellum to the diencephalon. It lies in the gap in the tentorium cerebelli and is largely hidden by the surrounding structures (Harold ELLIS-2006).

## **2.1.1.17** The pineal body

Is a small glandular structure that lies between the superior colliculi (Fig. 2-3). It is attached by a stalk to the region of the posterior wall of the third ventricle (Harold ELLIS-2006).

#### **2.1.1.18 The Pons**

The pons lies between the medulla and the midbrain and is connected to the cerebellum by the middle cerebellar peduncles. It is 1in (25mm) in length and 1.5in (38mm) in width (Harold ELLIS-2006).

# 2.1.1.19 The medulla oblongata

Is conical in shape and connects the pons above to the spinal cord below. A median fissure is present on the anterior surface of the medulla, and on each side of this is a swelling called the pyramid (Richard S. Snell. 2008).

#### 2.1.1.20The cerebellum

The largest part of the hind-brain and occupies most of the posterior cranial fossa. It is made up of two lateral cerebellar hemispheresand a median vermis. Inferiorly, the vermis, superiorly, it is only marked off from the hemispheres as a low median elevation (Harold Ellis 2006.).

#### 2.1.1.21 Ventricles of the Brain

The ventricles of the brain consist of the two lateral ventricles, the third ventricle, and the fourth ventricle. The two lateral ventricles communicate with the third ventricle through the interventricular foramina the third ventricle communicates with the fourth ventricle by the cerebral aqueduct. The fourth ventricle, in turn, is continuous with the narrow central canal of the spinal cord. (Richard S. Snell. 2012).

## 2.1.1.22 blood supply of the Brain

The brain is supplied by the two internal carotid and the two vertebral arteries. The four arteries anastomose on the inferior surface of the brain and form the circle of Willis (circulusarteriosus), the venous blood supply of the Brain emerge from the brain and drain into the cranial venous sinuses (Fig.2.3). Cerebral and cerebellar veins and veins of the brainstem are present. The great cerebral vein is formed by the union of the two internal cerebral veins and drains into the straight sinus (Richard S .Snell. 2012).

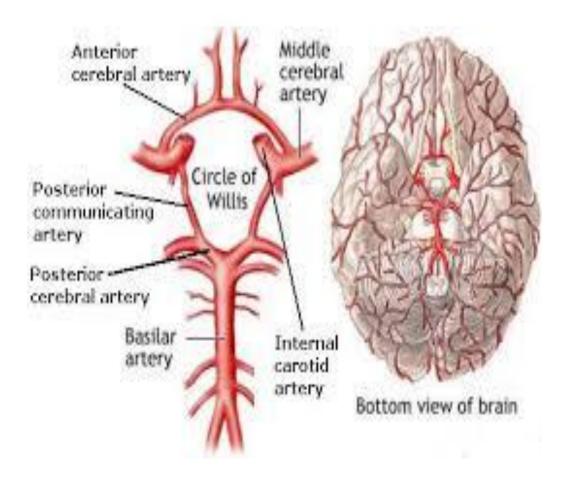


Fig (2.6) shows circle of willies, internal carotid artery ,basilar artery ,posterior communicating artery ,anterior cerebral artery and the middle cerebral artery .

## Y-Y-YPhysiology of the Central Nervous Systems CNS

The central nervous system is the control center for the body. It regulates organ function, higher thought, and movement of the body. The central nervous system consists of the brain and spinal cord. ("Article Sources and Contributors.2008)

#### 2.1.2.1General functions of the CNS

CNS: The "Central Nervous System", comprised of brain, brainstem, and spinal cord. The central nervous system (CNS) represents the largest part of the nervous system, including the brain and the spinal cord. Together, with the peripheral nervous system (PNS), it has a fundamental role in the control of behavior. The CNS is conceived as a system devoted to information processing, where an appropriate motor output is computed as a response to a sensory input (Article Sources and Contributors.2008).

## 2.1.2.2Structure of the central nervous system

Neurons are highly specialized for the processing and transmission of cellular signals. Given the diversity of functions performed by neurons in different parts of the nervous system, there is, as expected, a wide variety in the shape, size, and electrochemical properties of neurons. (Article Sources and Contributors.2008).

#### 2.1.2.3 Function of neuron:

Sensory afferent neurons convey information from tissues and organs into the central nervous system. Efferent neurons transmit signals from the central nervous system to the effector cells and are sometimes called motor neurons. Interneurons connect neurons within specific regions of the central nervous system. Afferent and efferent can also refer generally to neurons which, respectively, bring information to or send information from brain region. Classification by action on other neurons Excitatory neurons excite their target postsynaptic neurons or target cells causing it to function. Motor neurons and somatic neurons are all excitatory neurons. Excitatory neurons in the brain are often glutamatergic. Spinal motor neurons, which synapse on muscle cells, use acetylcholine as their neurotransmitter. Inhibitory neurons inhibit their target neurons. Inhibitory neurons are also known as short axon neurons, interneurons or microneurons (Article Sources and Contributors.2008).

## 2.1.2.4 The Nerve Impulse

When a nerve is stimulated the resting potential changes. Examples of such stimuli are pressure, electricity, chemicals, etc. Different neurons are sensitive to different stimuli (although most can register pain). The stimulus causes sodium ion channels to open. The rapid change in polarity that moves along the nerve fiber is called the "ACTION POTENTIAL." This moving change in polarity has several stages: Depolarization The upswing is caused when positively charged sodium ions(Na+) suddenly rush through open sodium gates into a nerve cell. The membrane potential of the stimulated cell undergoes a localized change from-65 millivolts to 0 in a limited area. As additional sodium rushes in, the membrane potential actually reverses its polarity so that the outside of the membrane is negative relative to the inside. During this change of polarity the membrane actually develops a positive value for a moment (+40 millivolts). The change in voltage stimulates the opening of additional sodium channels (called a voltage-gated ion channel). This is example of a positive feedback loop ("Article Sources and Contributors.2008).

## 2.1.2.5 Repolarization

caused by the closing of sodium ion channels and the opening of Thedownswing potassium ion channels. Release of positively charged potassium ions (K+) from the nerve cell when potassium gates open (Article Sources and Contributors.2008).

# 2.1.2 .6Refractory phase

Is a short period of time after the depolarization stage. Shortly after the sodium gates open they close and go into an inactive conformation. The sodium gates cannot be opened again until the membrane is repolarized to its normal resting potential. The sodium-potassium pump returns sodium ions to the outside and potassium ions to the inside. During the refractory phase this particular area of the nerve cell membrane cannot be depolarized ("Article Sources and Contributors.2008).

#### 2.1.2.7 the Sympathetic and Parasympathetic Systems

The sympathetic nervous system activates what is often termed the fight or flight response, as it is most active under sudden stressful circumstances (such as being attacked). This response is also known as sympathetico-adrenal response of the body, as the pre-ganglionic sympathetic fibers that end in the adrenal medulla secrete acetylcholine, which activates the secretion of adrenaline (epinephrine) and to a lesser extent noradrenaline (norepinephrine) from it. Therefore, this response that acts primarily on the cardiovascular system is mediated directly via impulses transmitted through the sympathetic nervous system and indirectly via catecholamines secreted from the adrenal medulla (Article Sources and Contributors.2008).

The parasympathetic nervous system is part of the autonomic nervous system. Sometimes called the rest and digest system or feed and breed. The parasympathetic system conserves energy as it slows the heart rate, increases intestinal and gland activity, and relaxes sphincter muscles in the gastrointestinal tract. After high stress situations (ie: fighting for your life) the parasympathetic nervous system has a backlash reaction that balances out the reaction of the sympathetic nervous system (Article Sources and Contributors.2008).

## 2.1.3 Pathology

#### 2.1.3.1 Brain tumors:-

A tumor is a mass of tissue that's formed by an accumulation of abnormal cells. Normally the cells in body age die and was replaced by new cells. With cancer and other tumors, something disrupts this cycle. Therewere two types of tumors benign and malignant, the Difference between Benign and Malignant; thebenign brain tumors are noncancerous. Malignant primary brain tumors are cancers that originate in the brain, typically grow faster than benign tumors, and aggressively invade surrounding tissue. Although brain cancer rarely spreads to other organs, it will spread to other parts of the brain and central nervous system

(Article .D.craig hacking et-al)

#### 2.1.3.2 Hemorrhage

There was two type, Extradural haematoma and Subdural haematoma

Extradural haematoma the typical presentation is of a young patient involved in a head strike (either during sport or a result of a motor vehicle accident) that may or may not lose consciousness transiently. Following the injury they regain a normal level of consciousness (lucid interval), but usually have an ongoing and often severe headache. Over the next few hours they gradually lose consciousness.(Article .D.craig hacking et-al)

Subdural haematoma In adults SDHs are due to falls and there may not be a clear history of trauma. In young children, non accidental injury is a significant cause. The patient's level of consciousness gradually decreases with increasing mass effect and confusion is often encountered in the elderly.(Article .D.craig hacking et-al).

#### 2.1.3.3 Cerebral atrophy:-

Cerebral atrophy is a common feature of many of the diseases that affect the brain. Atrophy of any tissue means loss of cells. In brain tissue, atrophy describes a loss of neurons and the connections between them. Atrophy can be generalized, which means that all of the brain has shrunk; or it can be focal, affecting only a limited area of the brain and resulting in a decrease of the functions that area of the brain controls. If the cerebral hemispheres (the two lobes of the brain that form the cerebrum) are affected, conscious thought and voluntary processes may be impaired. Associated Diseases/Disorders: The pattern and rate of progression of cerebral atrophy depends on the disease involved. Diseases that cause cerebral atrophy include: stroke and traumatic brain injury Alzheimer's disease, Pick's disease, and fronto -temporal dementia cerebral palsy, in which lesions (damaged areas) may impair motor coordination Huntington's disease, and other hereditary diseases that are associated with genetic mutations (Article D.craig hacking et-al)

# 2.1.3.4 Epilepsy:-

Epilepsy is a condition in which persisting cerebral dysfunction causes recurring epileptic seizures without the need for immediate insults to provoke each seizure; exacerbants such as sleep deprivation can increase seizure frequency in epilepsy, however. Acute symptomatic seizures are generalized tonic-clonic seizures that occur in the absence of epilepsy in response to a wide range of provoking insults, such as hyponatremia and other electrolyte disorders and fever in infants and young children (Thomas R. Henry, MD.2012).

## 2.1.3.5 Classification of Seizures and Epilepsy

Classification is critical to the optimal care of a patient. It provides information on etiology, treatment and prognosis. Modern classification of the epilepsies is based on how the seizures begin.(James Bowman, et-al ,2001).

#### 2.1.3.6 Partial seizures

Partial seizures begin in a discrete cortical area. They are categorized as simple when consciousness is preserved and complex when consciousness is altered.(James Bowman,et-al, 2001).

# 2.1.3.7 Simple partial seizures

Simple partial seizures are the primary complaint in 10% of patients with epilepsy. They can occur frequently and may result in little disability. Simple motor seizures result from a discharging lesion in the precentralgyrus of the frontal lobe of the cerebral hemisphere opposite the muscle contractions. Some are sustained (tonic) and others intermittent (clonic), and they may involve any body part depending on the location of the abnormal brain discharge.(James Bowman,et-al ,2001).

## 2.1.3.8 Complex partial seizures

Complex partial seizures are the predominant seizure type in about 20% of patients with epilepsy. During these seizures there is a period of altered behavior for which the patient is later amnesic. The amnesia for ictal events is akey factor for the diagnosis of a complex partial seizure. A typical complex partial seizure consists of several phases. About 70% begin with an 'aura', a sometimes complex psychic experience that may be manifest in one or more of a wide variety of vivid forms: as an illusion, hallucination, dyscognitive state or emotional (affective)

experience, it is defined as a complex partial seizure. Dystonic (twisted, stiff) posturing of the arm or leg on the side opposite to where the seizure occurs is often observed. Primitive movements occur frequently. These are referred to as spontaneous automatisms, and may include aimless fumbling with clothing or walking in a daze, and chewing or swallowing.

(James Bowman ,et-al ,2001)

#### 2.1.3.9 Primary generalized seizures

Involve widespread areas of the cerebral cortex from the onset. These terms must not be confused with the term secondary generalized seizure, which refers to a partial onset seizure that spreads to wide areas of cortex. The abnormal electrical activity is the same in both the left and right hemispheres (bilaterally symmetrical). Generalized seizures are further subdivided into convulsive and non convulsive types Convulsive seizures are characterized by sometimes violent and sustained contractions of muscles. No convulsive seizures lack prominent motor activity. (James Bowman,et-al ,2001).

#### 2.1.3.10 Absence seizures

Absence (petit mal) seizures occur without warning and consist of a sudden interruption of consciousness. The hall marks of absence seizures are their brevity, general lack of motor activity, frequency, and lack of post ictal period. (JamesBowman,et-al ,2001).

The seizures usually last from2to10s, occasionally longer. Patients are often unaware of them. An observer may interpret an absence as a moment of daydreaming. The person stops talking briefly in mid sentence, stares, or stops responding. As many as sever ahundred such seizures may occur in 1day. Absence seizures almost always begin in

childhood or adolescence. They often disappear before adulthood. (James Bowman, et-al ,2001)

#### 2.1.3.11 Atonic seizures

Atonic seizures (drop attacks) manifest themselves as a sudden loss of tone in postural muscles. In a mild variant, only the head drops. However, |in theseever form,allof the postural muscles lose tone, and the patient suddenly collapses to the ground. Frequent falls may result in injury, especially to the head, so protective headgear may be needed. The duration of the attack is usually only a few seconds, but the seizure maybe more prolonged. When the attack is brief, no notable postictal symptoms occur. (James Bowman, et al., 2001)

### 2.1.3.12Myoclonic seizures

Myoclonic seizures consist of bilaterally synchronous involuntary muscle jerks that occur singly or in a brief salvo of repeated jerks. Some myoclonic jerks can be restricted to only one muscle, while others involve large muscle masses including both arms and legs or even the entire body.(JamesBowman,et-al, 2001)

# 2.1.3.13 Initiation and spread of seizures

A number of terms are widely used in describing the results of epilepsy research and so should be defined. The epileptogenic focus is a cortical area containing abnormally functioning neurons which is determined electroencephalographically during the inter ictal period thus,itis an electrophysiological concept .(James Bowman,et-al ,2001)

## 2.1.4MRIphysics

#### **2.1.4.1 Magnets**

Nearly all clinical scanners are superconducting magnets with a horizontal bore design. These are electromagnets that are made from superconducting alloys such as niobium—titanium. To achieve superconductivity and therefore zero electrical resistance they need to be cooled below a certain critical temperature. This is achieved using liquid helium, which surrounds the magnet windings in the cryostat, and needs to be replenished regularly. Lower field systems may be resistive electromagnets and some earlier machines even used permanent magnetic cores. Open design magnets permit interventional work during scanning and reduce claustrophobia. However, this is becoming less of an issue with modern wide, short bore scanners (Gary Liney-2006).

#### **2.1.4.2 Gradients**

Additional coils are needed within the magnet to provide the gradients in each orthogonal direction. A Maxwell pair is used to provide the z-direction gradient, whereas two sets of Golay coils are needed for the x-and y-directions. The rapidly changing fields of the gradients induce oscillating eddy currents and as a result, magnetic fields in the surrounding conductors. These produce distortions in the image and require the gradients themselves to be shielded in a similar manner to the main field (Gary Liney-2006).

#### **2.1.4.3 RF coils**

To provide the B1 field necessary for transmission and reception, RF coils are required, either working separately or as combined transceivers. Coils are designed to be appropriately sized for the anatomy under

examination, which ensures optimum signal-to-noise. Image quality is further influenced by the transmission/reception profile of the coil (Gary Liney-2006).

Quadrature or circularly polarized types detect signal at 90° apart, which improves the signal-to-noise and efficiency. The latest coil arrays are described as multichannel. Each coil element possesses a separate receiver, and is compatible with parallel imaging techniques to reduce scan time by a factor equal to the number of available channels. A selection of RF coils (Gary Liney-2006).



Figure (2.7) shows Head coil

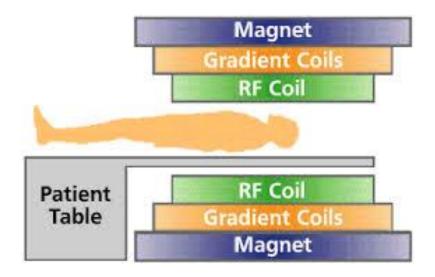
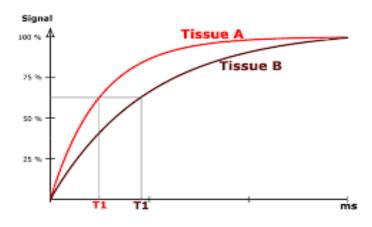


Figure (2-8) shows MRI magnet

#### Longitudinal magnetization recovery (T1)



**Fig** (2-9) shows  $T_1$  CURVE

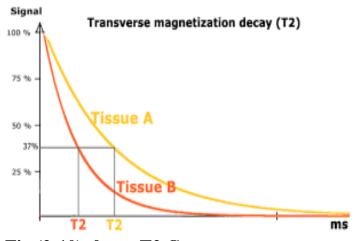


Fig (2-10) shows T2 Curve

## 2.1.4.4 PULSE SEQUENCES

#### **2.1.4.4.1Spin echo (SE)**

A spin echo (SE) pulse sequence (also known as conventional spin echo (CSE)) usually uses a 90° excitation pulse followed by a 180° rephasing pulse to produce a spin echo. Some SE sequences use a variable flip angle, but traditionally the excitation pulse has a magnitude of 90°. This amplitude of the flip angle is consistently assumed in the protocols. SE sequences can be used to generate one or several spin echoes. One echo is usually used for T1 weighting while two echoes are used for proton density (PD) and T2 weighting. SE pulse sequences are the most commonly implemented sequences as they produce optimum SNR and CNR (Catherine Westbrook.2008).

For T1 weightingin SE use: For PD/T2 weightingin SE use: short TE min–20 ms short TE 20 ms (first echo PD) short TR 300–600 ms long TE 70 ms (second echo T2) long TR 2000 ms(Catherine Westbrook.2008).

#### 2.1.4.4.2 Fast spin echo (FSE) or turbo spin echo (TSE)

Fast spin echo (FSE) uses a 90°flip angle followed by several 180°rephasing pulses to produce several spin echoes in a given TR. Each echo is phase encoded with different amplitude of gradient slope, so that data from each echo are collected and stored in a different line of K space (Catherine Westbrook.2008).

## 2.1.4.4.3(Inversion recovery (IR/IR-FSE)

Inversion recovery (IR) pulse sequences begin with a 180° pulse that inverts the net magnetization vector into full saturation. When the inverting pulse is removed, the magnetization begins to recover and

return towards B0. After a specific time TI (inversion time), a 90°excitation pulse is applied which transfers the proportion of magnetization that has recovered to B0 into the transverse plane. This transverse magnetization is then rephased by a 180°rephasing pulse to produce an echo. In IR-FSE several 180° rephasing pulses are applied as in FSE, so that more than one line of K space can be filled per TR, so reducing the scan times (Catherine Westbrook.2008).

There are two main uses of this technique. Short TI inversion recovery (**STIR**) uses a short TI that corresponds to the null point of fat so that the excitation pulse specifically nulls the signal from fat. Fluid attenuated inversion recovery (**FLAIR**) utilizes a long TI corresponding to the null point of cerebrospinal fluid (**CSF**) (Catherine Westbrook.2008).

For T1 weightingin IR use: short TE min–20 ms long TR ≥2200 ms medium TI 200–600 ms (depending on the field strength.

# 2.1.4.4.4 (Coherent gradient echo (GRE) (T2\*)

Coherent gradient echo (GRE) pulse sequences use a variable flip angle followed by gradient rephasing to produce a gradient echo. This sequenceutilizes the steady state so that the transverse component of magnetization is allowed to build up over successive repetition time (Catherine Westbrook.2008).

# 2.1.4.4.5 Echo planar imaging EPI

EPI Echo planar imaging (EPI), is a gradient-intensive sequence capable of acquiring all the necessary phase-encoding steps in a single TR (single shot), producing images at a rate of a few milliseconds per slice. Usually a reduced resolution is acquired (typically 64 or 128 matrixes). It is useful

whenever short image times are needed, for example pediatric imaging and fMRI).(Gary Liney-2006)

#### 2.1.4.4.6 MRS Localization

Many different MRS sequences are used to localize the desired volume of interest in either a single or multivoxel technique. The two most commonly employed sequences are STEAM and PRESS, which utilize slice selection in each orthogonal direction to measure either a stimulated echo or a spin-echo from the intersected volume of interest (VOI)(Gary Liney-2006).

#### 2.1.4.4.7 Other Notable Sequences

For completeness here are a few brief comments on other types of sequences you may come across (Gary Liney-2006).

# 2.1.4.4.8 Steady state free precession (T2)

This is a steady state sequence that uses medium flip angles and a short TR to maintain the steady state so that residual magnetization builds up in the transverse plane. These sequences generate contrast by sampling this transverse magnetization, which is mainly T2 weighted. The T2 weighted echo is repositioned by a gradient so that the TE is longer than the TR. Hence true T2 weighting can be achieved in conjunction with a short TR. The actual TE selected at the console is  $2 \times 10^{12}$  the TR minus the time between the echo and the next RF pulse (usually called, very confusingly, the TE). Therefore the shorter the TE selected at the console, the longer the actual TE and hence the greater the T2 weighting of the image.

For T2 SSFPuse: short TR  $\leq$ 50 ms short TE shortest edium flip angle  $30^{\circ}$ – $45^{\circ}$  (Catherine Westbrook.2008).

## 2.1.4.4.9 Driven Equilibrium:-

In a driven equilibrium sequence, a -90° pulse is used to force the transverse magnetization to return quickly along the zdirection rather than waiting for T1 recovery. This ensures a high fluid signal with reduced scan times. Examples of this type of sequence include DRIVE and FRFSE (Gary Liney-2006).

#### 2.1.4.4.10 Magnetization Transfer (MT) Imaging

This refers to off-resonance RF pulses that are used to saturate signal from large macromolecules such as proteins. The effect on mobile water protons produces a new type of image contrast and has been used in demyelination, in MRA to improve vessel visualization, and also in contrast-enhancement studies (Gary Liney-2006).

#### 2.1.4.4.11 Pre-scan and Localizer

The pre-scan is usually an automatic routine performed by the scanner prior to image acquisition. During this period the coil is tuned to the appropriate resonant frequency, and the receiver and transmitter gains are adjusted accordingly. Failure to do this results in a general degradation of image quality (Over-ranging Artifact) (Gary Liney-2006).

#### 2.1.4.4.12 Saturation Bands

These involve the application of slice-selection and dephasing gradients to remove the signal contribution from specific userdefined volumes of tissue. It is useful to eliminate unwanted signal contributions, for example, flow artifacts or contamination from outside MRS voxels (Gary Liney-2006).

#### 2.1.5 Brain MRI Technique:-

**2.1.5.1**Equipment: - Head coil (quadrature or multi-coil array). Immobilization pads and straps. Earplugs High-performance gradients for EPI, diffusion and perfusion imaging. (Catherine Westbrook.2008)

#### 2.1.5.2 Patient positioning

The patient lies supine on the examination couch with their head within the head coil. The head is adjusted so that the interpupillary line is parallel to the couch and the head is straight. The patient is positioned so that the longitudinal alignment light lies in the midline, and the horizontal alignment light passes through the nasion. Straps and foam pads are used for immobilization (Catherine Westbrook.2008).

#### 2.1.5.3 Suggested protocol

#### 2.1.5.3 .1 Sagittal SE/FSE/incoherent (spoiled) GRE T1

Medium slices/gap is prescribed on each side of the longitudinal alignment light from one temporal lobe to the other. The area from the foramen magnum to the top of the head is included in the image

(Catherine Westbrook.2008).

#### 2.1.5.3 .2 Axial/oblique SE/FSE PD/T2

Medium slices/gap is prescribed from the foramen magnum to the superior surface of the brain. Slices may be angled so that they are parallel to the anterior–posterior commissure axis(Catherine Westbrook.2008)

#### **2.1.5.3 .3 Coronal SE/FSE PD/T2**

As for Axial PD/T2, except prescribe slices from the cerebellum to the frontal lobe (Catherine Westbrook.2008).

### **2.1.5.3 .4 Axial/oblique IR T1**

Slice prescription as for Axial/oblique T2. This sequence is especially useful in imaging the pediatric brain (Catherine Westbrook.2008).

# 2.1.5.3 .5 Axial/oblique FLAIR/EPI

Slice prescription as for Axial/oblique T2. This sequence provides a rapid acquisition with suppression of CSF signal (Catherine Westbrook.2008).

### 2.1.5.3 .6Axial/oblique SE/FSE/incoherent (spoiled) GRE T1

Slice prescription as for Axial/oblique T2. Pre- and post-contrast scans are common especially for tumorassessment (Catherine Westbrook.2008).

#### 2.1.5.3.7 SS-FSE T2

Useful for rapid imaging in uncooperative patients (Catherine Westbrook.2008).

#### 2-2 previous study

Balogh 2016 stuidied The Propagation and Semiology of Focal Epileptic Seizures Cases Connected to the Insula. The oretical Considerations werethe preoperative, intra and postoperative data are analyzed of three insular and one parietal epileptic patient in point of view of their seizure neuro-imaging, noninvasive symptoms. Complex and invasive electrophysiology, intensive long-term video-EEG monitoring, computerized EEG analysis, functional mapping, intra operative corticography were used. The etiology was confirmed with histology the result was observed that on seizure semiology our patients play the insula a double role. In some cases, it is the focus of insular seizures with their symptoms difficult to identify. However, in the majority of cases and as a consequence of its rich neural connections, the insula has a peculiar property in the evolution of the symptomatogenic features of seizures. These observations are developing new relationships between the mechanism of seizure propagation and its semiological consequences and the conculusion One pileptological point of view there are brain structures which has peculiar role in the "design" of propagation of the epileptic excitement. The numerous new methods in neuroimaging and neurophysiology allowed the connectomical examination of the epileptic networks. The role of the epileptic diathesis is approachable with the metholdology of the brain connectivity. Theoretically the node of the epileptic network consist of the potential pathes where the localised excessive excitement can propagete. The route where the actual seizure can go adhead is determined by the actual edpileptic propensity of the above mentioned potential pathes.

COAN AC 2014 studied 3T MRI quantification of hippocampal volume and signal in mesial temporal lobe epilepsy improves detection of

hippocampal sclerosis. Two hundred three patients with mesial temporal lobe epilepsy defined by clinical and electroencephalogram criteria had 3T MRI visually analyzed by imaging epilepsy experts. As a second step, we performed automatic quantification of hippocampal volumes with FreeSurfer and T2 relaxometry with an in-house software. MRI of 79 healthy controls was used for comparison, result Visual analysis classified 125 patients (62%) as having signs of hippocampal sclerosis and 78 (38%) as having normal MRI findings. Automatic volumetry detected atrophy in 119 (95%) patients with visually detected hippocampal sclerosis and in 10 (13%) with visually normal MRimaging findings. Relaxometry analysis detected hyperintense T2 signal in 103 (82%) patients with visually detected hippocampal sclerosis and in 15 (19%) with visually normal MR imaging findings. Considered together, volumetry plus relaxometry detected signs of hippocampal sclerosis in all except 1 (99%) patient with visually detected hippocampus sclerosis and in 22 (28%) with visually normal MR imaging findings. the conclusion

In 3T MRI visually inspected by experts, quantification of hippocampal volume and signal can increase the detection of hippocampal sclerosis in 28% of patients with mesial temporal lobe epilepsy.

MajidGhaffarpour 2011 studied Evaluation of partial epilepsy in Iran: role of video-EEG, EEG, and MRI with epilepsy protocol in Forty-two consecutive patients underwent complete neurological examination, EEG, and MRI with a modified epilepsy protocol. A subset of these patients (n = 29) also underwent VEM. Data were presented using descriptive statistics and were analyzed using Chi square and McNemartests.the results were analyzeTwenty-four women and eighteen men entered the study. The mean (±SD) age for patients, was 25.2(±10.1) and mean (±SD) age at onset was 10.9(±8.1). All patients had abnormal ictal or

interictal EEG. Fifteen patients had normal MRI. Temporal lobe involvement was the most common involvement in both EEG (27 patients) and MRI (14 patients). Interictal EEG was abnormal in 81% of patients which showed epileptiform discharges in about half of the cases. In half of patients who had lateralized finding on MRI, site of the lesion was congruent between MRI and interictal EEG. Thirty-six patients had symptoms suggesting a specific lobe, of which interictal EEG was able to show the concordant lobe in 22 (61%) patients. McNemar test showed superiority of EEG over MRI in correct diagnosis of the involved lobe based on the clinical manifestations (P < 0.01).

Dr:Sanjida 2010 studied Clinical Ahmed Profile of Early Childhood Epilepsy: A Cross Sectional Study in a Tertiary Care Hospital. This cross sectional study was conducted at outpatient department of Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka from January 2010 to December 2010 to explore clinical profile of early childhood epilepsy. Total 50 Children with two or more unprovoked seizure after 28 days up to 36 months of age were included in this study. Majority cases were in the age group of 1 to 12 months (56%) with male predominance (78%). Onset of first seizures was found to be the highest at the age group 0-1 month (50%) with generalized seizures (66%) as the most common type of initialseizures. Highest 16(32%) patients presented with tonic clonic seizure followed by clonic seizure in 15(30%) and tonic in 11(22%) patients. Myoclonic seizure was found to be 4(8%). Only 4(8%) cases were presented with infantile spasm. Majority cases associated with cerebral palsy (72%). Family of epilepsy was present in 16%. EEG was done in all patients. Among them 62% were abnormal. Total 21 cases had done MRI scan of brain. 6(28.6%) were normal and 15(71.4%) Among them were abnormal. The abnormal findings were cerebral atrophy 73.3%,

ventricular dilatation 13.3%, encephalomalacia 6.7% and cerebral infractions were found in 26.7%. In this study majority of cases in 22(44%) were treated with Phenobarbitone (PHB) as a first line drug followed by Valproate (VPA) in 19(38%). Limited study has been conducted on early childhood epilepsy in Bangladesh. The result of this study might be helpful for further large scale study in the field of early childhood epilepsy.

Sean O. Caseya, 2000 studied Posterior Reversible Encephalopathy Syndrome: Utility of Fluid-attenuated Inversion Recovery MR Imaging in the Detection of Cortical and Subcortical Lesions, Posterior reversible encephalopathy syndrome (PRES) is typically characterized by headache, altered mental functioning, seizures, and visual loss associated with imaging findings of bilateral subcortical and cortical edema with a predominantly posterior distribution. Our goal was to determine whether fluid-attenuated inversion recovery (FLAIR) imaging improves the ability to detect subtle peripheral lesions of PRES, as compared with conventional MR techniques; sixteen patients with clinical and imaging findings consistent with PRES were studied. Thirteen patients had undergone transplantation and had cyclosporinA neurotoxicity. Fast-FLAIR images were compared with spin-echo proton density-and T2weighted images,FLAIR imaging improved diagnostic confidence and conspicuity of the T2 hyperintense lesions of PRES, typically in the subcortical white matter of the parietooccipital regions bilaterally. On all 23 abnormal MR studies, FLAIR was judged superior to proton densityand T2-weighted images for the detection of PRES in the supratentorial brain. In a mean of 6.7 of 23 studies, FLAIR findings prompted a raise in the grade of disease severity. FLAIR also showed cortical involvement in 94% of patients with PRES and in a mean of 46% of the total lesion burden. In four cases, subtle lesions were virtually undetectable without FLAIR. Brain stem or cerebellar disease was encountered in 56% of patients.

FLAIR improves the ability to diagnose and detect subcortical and cortical lesions in PRES as compared with proton density—and T2-weighted spin-echo images. We therefore believe that FLAIR should be performed in patients with suspected PRES to allow more confident recognition of the often subtle imaging abnormalities.

Serviço de Cirurgia 1997. Studied unilateral mesial temporal atrophy after a systemic insult as a possible etiology of refractory temporal lobe epilepsy, Mesial temporal sclerosis is the main pathological substrate present in refractory temporal lobe epilepsy and its presence is often related to the occurrence of febrile seizures in infancy. There is an ongoing discussion on the nature of mesial temporal sclerosis as it related to epilepsy: cause or consequence. A previously normal child developed hyperosmolar coma after abdominal surgery at the Serviço de Cirurgia 1997age of 6. Three months afterwards he developed simple and complex partial seizures with an increasing frequency and refractory to multiple mono- and polytherapic drug regimens. He was evaluated for surgery at the age of 13. Ictal and interictal recordings showed left temporal lobe abnormalities. Early CT scanning suggested left temporal atrophy. MRI showed mesial temporal sclerosis. Neuropsychological testing showed verbal memory deficits and he passed a left carotid artery amytal injection. He was submitted to a cortico-amygdalo-hippocampectomy and has been seizure-free since then. The clinical data obtained from this patient suggest that at least in this case mesial temporal sclerosis would be related to the cause of epilepsy and not resultant from repeated seizure activity.

# **Chapter three**

# Materials and methods

# 3.1 materials

# 3.1.1 Patient data:

100 patients participate in the study all having seizures with different clinical data, severity and duration.

# 3.1.2 machine

MRI machine 1.5 tesla, Toshipa, place of manufacturing: china, manufacturing date: January 2012.

Head coil (quadrature or multi-coil array).

- Immobilization pads and straps.
- Ear plugs.
- High-performance gradients for EPI, diffusion and perfusion imaging.

# 3.2 methods:

# 3.2.1 Patient prepration

Proper screening policy which includes checking for:

Pacemakers

Aneurysm clips

- Intra-ocular foreign bodies
- Metal devices or prostheses

- Cochlear implants
- Spinal implants
- Possibility of early pregnancy
- Removal of all jewellery, credit cards, money, watches, etc.

Patients asked to remove all metallic jewelers.

Ask the patient to change his\her clothes into a gown.

Care must be taken when transferring patients either on to trolleys or into the examination room This is especially important if the patient is physically disabled, traumatized or in pain.

# 3.2.2 Patient positioning

The patient lies supine on the examination couch with their head within the head coil. The head is adjusted so that the interpupillary line is parallel to the couch and the head is straight. The patient is positioned so that the longitudinal alignment light lies in the midline, and the horizontal alignment light passes through the nasion. Straps and foam pads are used for immobilization.

The researcher attended MRI-brain exams of all patients had epilepsy randomly from the referring doctor which were done by experience technician, patients who had involuntary movement take diazepam to relax and sleep during examination and this was for children too.

# 3.2.3 Technique used: brain MRI pre contrast:

Axial T1, Coronal T1, Sagittal T1, Axial T2, Axial flair, Diffusion weighted image, Post contrast Axial T1.

#### 3.2.4 Data collection:

Patient questionnaire include

Patients' age, severity of seizures, onset duration of seizures, reason of seizures, clinical finding of seizures, past history of surgical operation past history of medical treatment past history of injury, time which seizures occur history of fever, cause of seizure, neurological disorders such as weakness paralysis learning disability and memory problems, result of the examination such as (MRI finding).

These variables were designed in form of questions about research, all questions answered by the patients verbally and filled by the researcher.

# 3.2.5 Data analysis

Data was analyzed by Microsoft excel, we used seizure as main criteria for selecting our participants so 100 patients having seizure.

# 3.2.6 Study area

This study was done in alzaytouna specialize hospital in Khartoum state and patients was done MRI from referring doctors all had seizures

# **Chapter four**

# **Results**

Evaluation of magnetic resonance imaging and clinical findings in diagnosing of epilepsy where as 100 patients had epilepsy and the data was collected by graghs and figures:

**Table 4.1:** shows the frequency and percentage of MRI findings for seizure patients.

MRI findings	Frequency	Percentage
Normal	19	19%
Abnormal	81	81%
Total	100	100%

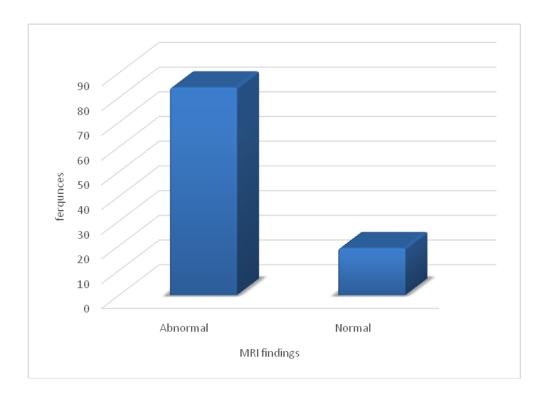


Fig (4.1) shows the correlations between the frequency and MRI findings.

**Table 4.2**: shows frequencies and percentage of abnormal and normal cases caused seizures:-

Pathology	Frequency	Percentage
Atrophy	٦,	%1.
Meningio-	١.	%).
enchephalities		
Tumor	٥	%00
Stroke	٥	%°
Congenital	١	%)
abnormalities		
(hydrocephalus)		
Normal	19	%19
Total	1	%1

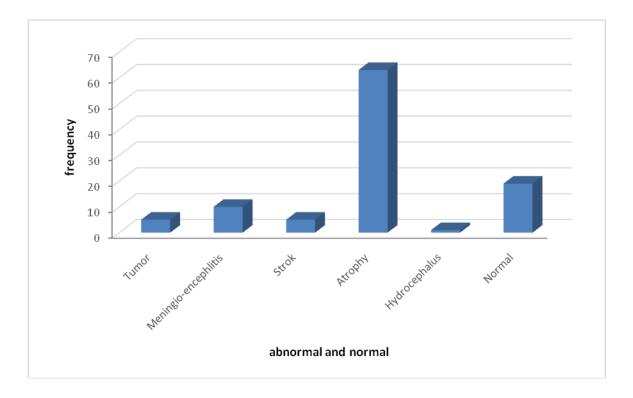
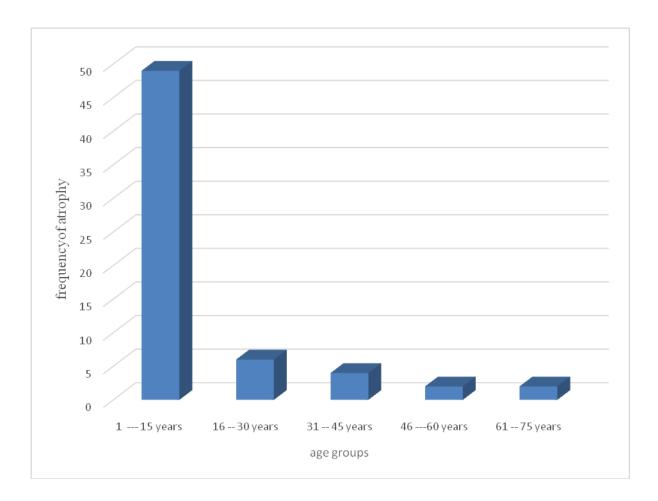


Fig (4.2) shows the correlations between abnormal, normal cases and frequency

Table 4.3: shows the distribution of atrophy according to age group:-

Age group vs atrophy	Frequency	Percentage
1≥-15y	٤٦	% ∀∀
16-30y	٦	%1.
٣١-45y	٤	%∨
٤٦-60y	۲	%٣
71-75y	۲	%٣
Total	٦٠	%)



**Fig 4.3**: shows the correlations between the frequency of atrophy cases and age groups.

**Table 4.4**: shows the distribution of meningio-enchephalitiesaccording to age group:-

Age group vs. meningio-enchephalities	Frequency	Percentage
1≥-15y	٩	%٩.
16-30y	١	%1.
٣١-45y	•	%.
٤٦-60y	•	%.
71-75y	•	%•

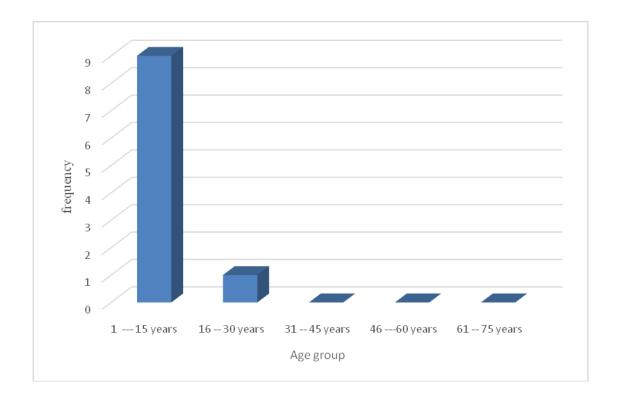
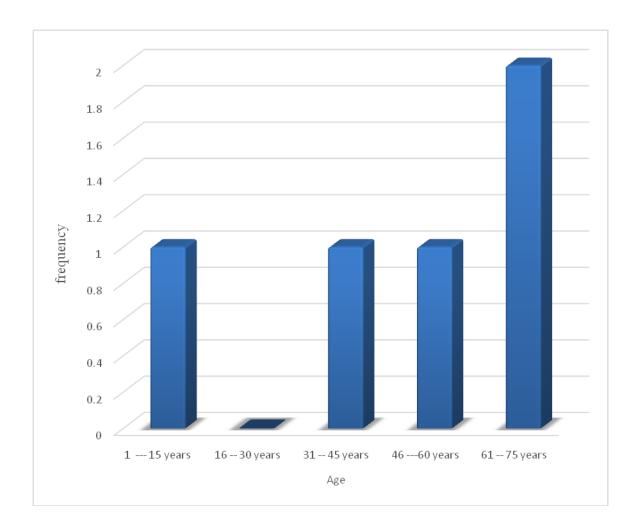


Fig 4.4: shows the correlations between the frequency of meningioenchephalities and age group.

Table 4.5: shows the distribution of stroke according to age group:-

Age group vsstroke	Frequency	Percentage
1≥-15y	1	%7.
16-30y	•	%.
٣١-45y	1	%7.
٤٦-60y	1	%7.
11-75y	۲	% € .
Total	٥	%1



**Fig 4.5**: shows the correlations between the frequency of stroke cases and age group.

Table 4.6: shows the distribution of tumor cases according to age group:-

Age group vstumour	Frequency	Percentage
1≥-15y	•	%.
16-30y	١	%7.
٣١-45y	٣	%1.
٤٦-60y	*	%.
71-75y	١	%7.
Total	٥	%1

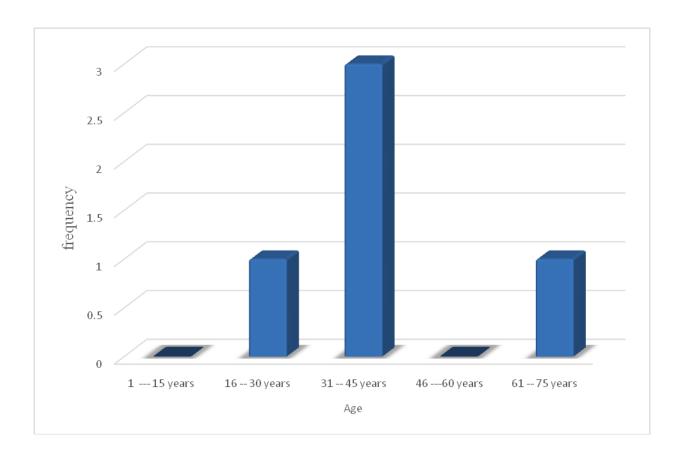


Fig 4.6: shows the correlations between the frequency of tumor and age group

**Table 4.7**: shows the distribution of congenital abnormalities cases according to age group:-

Age group vs.congenital abnormalities	Frequency	Percentage
1≥-15y	١	%1
16-30y	•	%•
٣١-45y	•	%•
٤٦-60y	•	%•
11-75y	•	%,
Total	)	%1

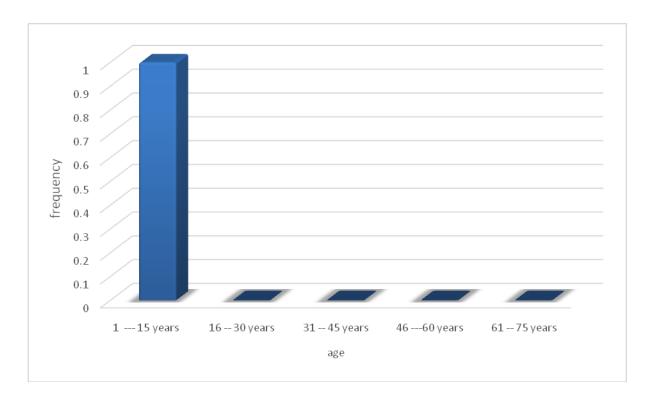


Fig 4.7: shows the correlations between the frequency of congenital abnormalities and age group

Table 4.8: shows the distribution of normal cases according to age group:-

Age group vs.normal cases	Frequency	Percentage
1≥-15y	0	%٢٦
16-30y	٤	%٢١
٣١-45y	٧	%٣٧
٤٦-60y	٣	%17
11-75y	•	%.
Total	19	%1

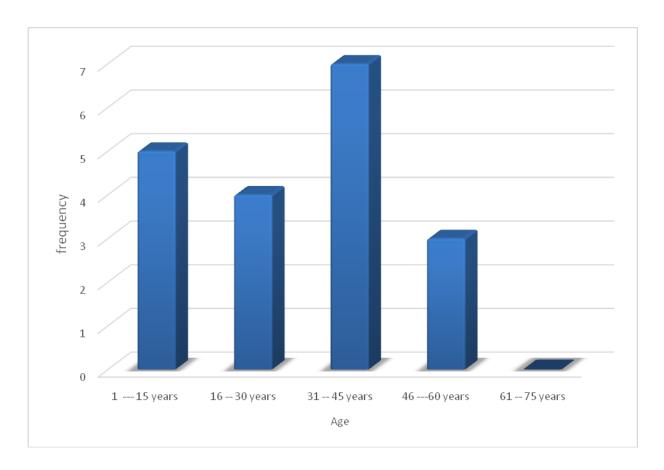


Fig 4.8: shows the correlations between the frequency of normal cases and age group

**Table 4.9**: shows the distribution of abnormal and normal cases according to age group:-

Age group	Atrophy	Meningio- enchalities	Stroke	Tumor	Congenital Abnormalities	Normal
1m-15	٤٦	٩	١	•	1	٥
17-30	٦	١	•	١	•	٤
31-45	٤	•	١	٣	•	٧
46-60	۲	•	١	•	•	٣
71-75	۲	•	۲	١	•	•
Total 100	٦٠	١.	٥	0	1	19

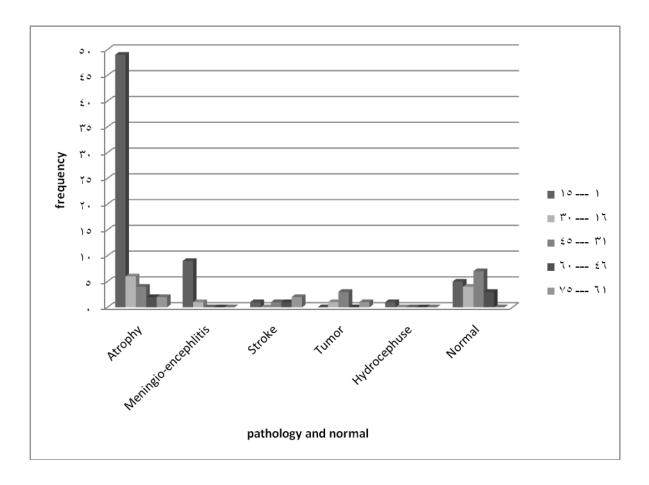


Fig 4.9: shows the correlations between the frequency of abnormal and normal cases according to age group

Table 4.10: shows the distribution of clinical finding among atrophy cases:-

Clinical finding	Frequency	percentage
Fever	٤٠	%YA
Convulsion	٥٧	%٣٩
Non-taking	٤	%٣
unconcious	٧	%0
Stuper	١٧	%17
Non walking	٧	%0
headache	۲	%1
vertigo	٥	%٢
Hypoxia	۸	%0

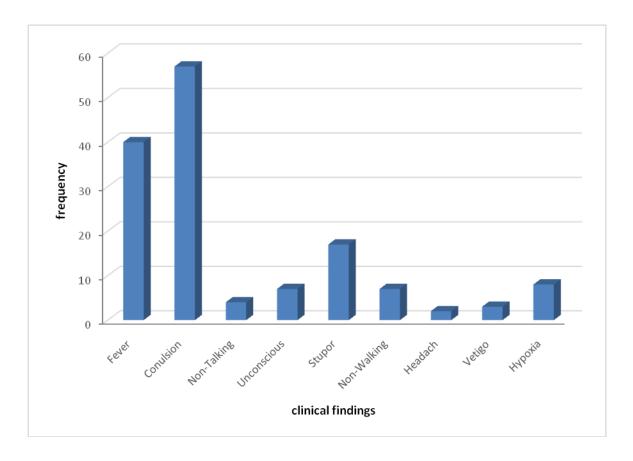


Fig 4.10: shows the correlation between trophy and their clinical findings

Table 4.11: shows the distribution of clinical finding among tumor:-

Clinical finding	Frequency	percentage
Fever	٣	%٣£
Convulsion	٣	%٣٤
Non-taking	•	%•
Unconscious	1	%11
Stuper	۲	%۲۲
Non walking	•	%•
Headache	•	%•
Vertigo	•	%•
Hypoxia	•	%•

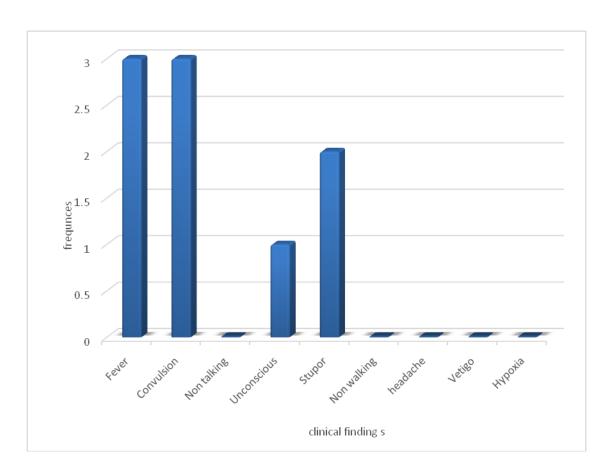


Fig 4.11: shows the correlation between tumor cases and their clinical findings

**Table 4.12**: shows the distribution of clinical finding among meningioenchephalities cases:-

Clinical finding	Frequency	percentage
Fever	٩	%٣٤
Convulsion	١.	% <b>٣</b> ٨
Non-taking	1	%ξ
Unconscious	•	%•
Stuper	1	%€
Non walking	۲	%A
Headache	•	%•
Vertigo	۲	%∧
Hypoxia	1	%€

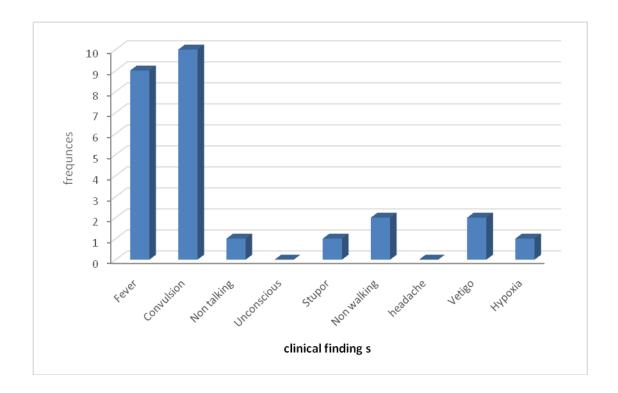


Fig 4.12: shows the correlation between meningio-enchephalities

Cases and their clinical findings

Table 4.13: shows the distribution of clinical finding among stroke cases:-

Clinical finding	Frequency	percentage
Fever	١	%1
Convulsion	٥	%٦
Non-taking	•	%•
Unconscious	۲	%٢
Stuper	•	%•
Non walking	1	%1
Headache	•	%•
Vertigo	•	%•
Hypoxia	•	%•

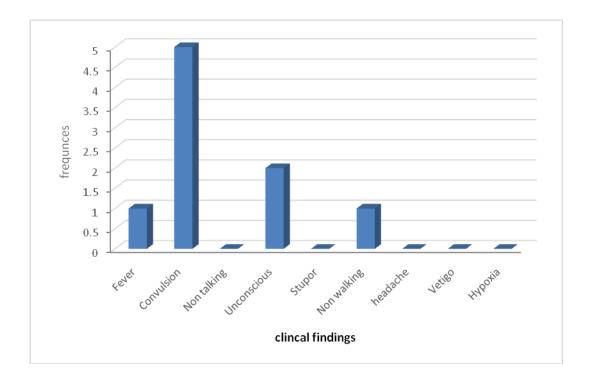


Fig 4.13: shows the correlation between stroke cases and their clinical findings

**Table 4.14**: shows the distribution of seizure with normal MRI images with clinical findings`

Clinical finding	Frequency	percentage		
Fever	٦	%1 A		
Convulsion	١٧	%°7		
Non-taking	۲	%٦		
Unconscious	۲	%٦		
Stuper	۲	%٦		
Non walking	•	%•		
Headache	1	%٣		
Vertigo	1	%٣		
Hypoxia	۲	%٦		

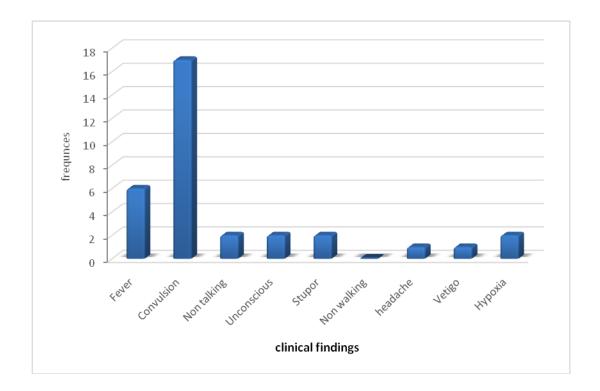


Fig 4.14: shows the correlation between frequency of normal cases and their clinical findings

Table (4.15) show the relation of clinical finding and the final diagnosis:-

	Pathology	Count	fever	Conv	Non	Нуро	Un	stuper	Non	Head	Vertigo
Final				tion	tak	xia	conscio		walk	ache	
Diag					ing		us		ing		
nose	Atrophy	Freq	٤٠	٥٧	٤	٨	٧	١٧	٧	۲	٥
		Perc	%۲A	%٣٩	%٣	%°	%0	%17	%0	%١	%٢
	tumor	Freq	٣	٣	*	•	١	۲	•	*	•
		Perc	%٣٤	٣٤%	• %	%.	11%	%٢٢	% •	%.	% •
	Meningio- enchephali tes	Freq	٩	١.	,	1	•	1	۲	٠	۲
		Perc	% <b>٣</b> ٤	% <b>٣</b> ٨	% €	٤%	% •	% €	%∧	%.	%∧
	stroke	Freq	١	٥	•	•	۲	•	١	•	•
		Perc	%١	%٦	%.	%•	%٢	%•	%١	%•	%.
	normal	Freq	٦	١٧	۲	۲	۲	۲	•	١	1
		Perc	۱۸%	%07	%٦	%٦	<b>%</b> ٦	<b>%</b> ٦	%•	% <b>r</b>	%٣

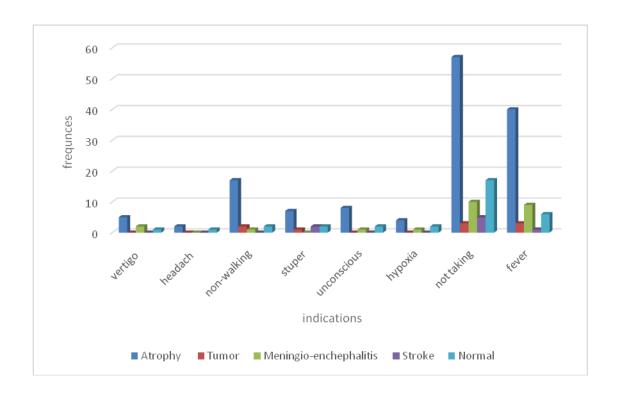


Fig 4.15 :show the correlation of clinical finding on the final diagnosis

# 5.1Discussion

**Table & fig** (4.1) show the frequency and percentage of MRI findings for seizure patients whereas 81(81%) had up normal MRI findings and 19(19%) had normal MRI findings for seizure patient agree with MajidGhaffarpour 2011 All patients had abnormal ictal or interictal EEG. 15 patients had normal MRI, Coan Ac 2014,10 (13%) with visually normal MR imaging findings.

**Table& fig**(4.2) show the frequency and percentage of different pathology caused seizure where as atrophy had maximum participants by frequency of 60(60%) consequently the normal seizure MRI by frequency of 19(19%), meningio-enchephalities 10(10%), tumor and stroke 5(5%) and the minimum participants congenital abnormalities 1(1%) such as hydrocephalus and the total number of differ pathology caused seizure was 100 agree with Serviço de Cirurgia 1997and agree with Coan Ac 2014 Automatic volumetry detected atrophy in 119 (95%) patients with visually detected hippocampal sclerosis.

**Table & fig** (4.3) show the distribution of atrophy according to the age group, in the group between  $(1 \ge -15y)$  was the maximum participants with maximum frequency by 46(77%) consequently age group between (16-30y)by 6(10%), the age group between (31-45y)by 4(7%), the minimum participants in the age group between (46-75) by 2(3%), the total participants in all age group in atrophy was 60 agree with Dr.Sanjida Ahmed 2010were cerebral atrophy 73.3%.

**Table &fig** (4.4) show the meningio-enchepalities according to age gro\up, the maximum participants in the age group between  $(1 \ge -15y)$ by 9(90%) consequently the age group between (16-30y) by 1(10%) and had

no participants in other age groups agree with Dr.Sanjida Ahmed 2010were encephalomalacia 6.7%.

**Table & fig**( (4.5) show the distribution of the stroke according to the age groups, the maximum participants in the age group between (61-75y)by 2(40%)consequently the age group between  $(1\ge-15y)$ and (31-60y)by 1(20%) and there was no participants in the age group between (16-30y), the total participants in stroke according to age groups was 5.

**Table & fig**( (4.6) show the distribution of tumor according to the age groups, the maximum participants in the age group between(31-45y)by 3(60%) consequently the age group between (16-30y) and the age group between (61-75y) by 1(20%) and there was no participants in other age groups with total participant of 5.

**Table & fig**( (4.7) show the distribution of congenital abnormalities according to the age groups, the maximum participants in the age group between  $(1 \ge -15y)$  by frequency of 1(100%) and there was no participants in other age groups with total participant of 1.

**Table & fig**( (4.8) shows the distribution of normal participants with seizures according to the age groups, the maximum participants in the age group between(31-45y)by 7(37%) consequently the age group between(1m-15y)by 5(26%), the age group between (16-30) by 4(21%) and the minimum participants was in the age group (46-60)by 3(16%) and there was no participants in other age groups with total participant of 19. agree with MajidGhaffarpour 2011 All patients had abnormal ictal or interictal EEG. 15 patients had normal MRI and agree with Coan Ac 2014,10 (13%) with visually normal MR imaging findings.

**Table & fig** (4.9) show the participant in age group between  $\geq 15_{years}$  was maximum in atrophy cases consequently meningioencephalitis, normalcases, stroke and congenital abnormalities (hydrocephalus) and there was no participant intumorcases agree with Serviço de Cirurgia 1997and Coan Ac 2014 Automatic volumetry detected atrophy in 119 (95%) patients with visually detected hippocampal sclerosis.

In age group between (16-30years)the most participants was in atrophy cases consequently the normal cases, tumorand meningio encephalitis and there was no participant in this age group in stroke and congenital abnormalities cases.

The most participant in age group between (31-45years) innormal cases consequently atrophy,tumor, stroke cases and there was no participant in meningio-encephalitis and congenital abnormality cases.

In age group between (46-60y) themaximum of the participants in normal cases consequently atrophy, stroke and no participant in meningioenchephalities, tumor and congenital abnormalities cases.

**Table & fig** (4.10) show The maximum of participant in brain atrophy by 57(39%) was convulsion consequently 40(28%) of participants was fever then17(12%) was stupor and 7(5%) of participants with hypoxia, unconscious and 4(3%) of participant non walking ,3(2%) of participant with vertigoand2(1%) of participant with headache .so that maximum clinical data in brain atrophy was convulsion and fever and the minimum clinical data of participants was headache and vertigo agree with Serviço de Cirurgia 1997 and Coan Ac 2014 Automatic volumetry detected atrophy in 119 (95%) patients with visually detected hippocampal sclerosis.

**Table & fig**(4.11) the maximum participants in tumor with 3(34%) was fever and consequently3(34%) of participants with convulsion 2(22%) of participant withstupor,1(11%) with unconscious, no participant in other clinical data so the fever and convulsion and the stupor was most clinical data to the patients with tumor which cause seizure.

**Table & fig** (4.12) the maximum participant with 10(38%) to the patients with convulsion consequently 9(34%) to the patients with fever ,2(8%)in patients with vertigo and nonwalking 1(4%) of participants with stupor and non taking and no participant unconscious. So the most participants in cases of seizures causedby meningioenchephalities were fever, convulsion and the minimum was unconscious.

**Table & fig** (4.13) the maximum participants in stroke cases with convulsion by 5(6%) consequently 2(2%) of participants unconscious 1(1%) non walking and fever and participants in other clinical data so that maximum participants in seizure caused by stroke was convulsion.

**Table & fig** (4.14) show 17(52%) of the participants in normal cases had convulsion consequently 6(18%) of the participants had fever ,2(6%) for non taking, unconscious, stuper and hypoxia ,1(3%) for vertigo and headache and no participant non-walking. so the most clinical data caused seizure for normal patients was convulsion , fever agree with MajidGhaffarpour 2011 All patients had abnormal ictal or interictal EEG. 15 patients had normal MRI and Coan Ac 2014,10 (13%) with visually normal MR imaging findings.

**Table & fig** (4.15) show the relation between clinical finding and the final diagnosis:-

brain atrophy by 57(39%)was The maximum of participant in convulsion consequently 40(28%) of participants was fever then17(12%) was stupor and 7(5%) of participants with hypoxia, unconscious and 4(3%) of participant non walking ,3(2%) of participant with vertigoand 2(1%) of participant with headache .so that maximum clinical data in brain atrophy was convulsion and fever and the minimumclinical data of participants was headache and vertigo agree with Serviço de Cirurgia 1997and Coan Ac 2014 Automatic volumetry detected atrophy in 119 (95%) patients with visually detected hippocampal sclerosis.

The maximum participants in tumor with 3(34%)was fever and consequently3(34%) of participants with convulsion 2(22%) of participant with stupor,1(11%) with unconscious, no participant in other clinical data so the fever and convulsion and the stupor was most clinical data to the patients with tumor which cause seizure.

The maximum participant with 10(38%) to the patients with convulsion consequently 9(34%) to the patients with fever ,2(8%) in patients with vertigo and non walking 1(4%) of participants with stupor and non taking and no participant unconscious. So the most participants in cases of seizures caused by meningioenchephalities were fever, convulsion and the minimum was unconscious.

The maximum participants in stroke cases with convulsion by 5(6%) consequently 2(2%) of participants unconscious 1(1%) non walking and fever and no participants in other clinical data so that maximum participants in seizure caused by stroke was convulsion .

The maximum participants 17(52%) of the participants in normal cases had convulsion consequently 6(18%) of the participants had fever ,2(6%) for non taking ,unconscious, stuper and hypoxia ,1(3%) for vertigo and headache and no participant non- walking. so the most clinical data caused seizure for normal patients was convulsion ,fever.

# 5.2 conclusions

Epilepsy in male, female in different age was come with different causes, severity and different clinical findingsdue to different type of disease. The researcher abstracted the following characteristic of different disease in different age group caused epilepsy:-

Atrophy was the main cause for epilepsy in different age group with different duration by clinical signs of convulsion, Fever, stuper, hypoxia, none walking, vertigo and headache

The age group ≥ 15 was the maximum group affected by atrophy and meningio-enchephalities was the main cause of seizures

The age group  $\leq 31$  was the maximum age group affected by stroke with clinical findings of convulsion.

Convulsion, fever, stupor, non-walking, non-taking and unconscious was the most clinical signs detected for patients with seizures caused by different pathology.

Normal MRI findings with clinical signs did not mean it was actually normal so that further study can detect it positive (+) such as EEG, VEEG.

# 5.3 recommendations

The researcher recommends the followings:-

- 1. Families should observe their childrens with fever at least the age  $\geq 15$ .
- 2. More seminar and refresh courses for technologist for seizures magnetic resonance protocol.
- 3. Different sequences in different plane for accurate results.
- 4. Future study to use large sample size to obtain more accurate results.
- 5. Further study such as electroencephalography-video electroencephalography and functional magnatic resonance imaging to detect the causes of seizures which was normal in MRI imaging

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