REVIEW OF LITERATURE

HISTORICAL ASPECTS:

Epilepsy has the longest medical history (Hoch and Knight, 1947). It was recognised in India before the tenth century B.C., and in the Greek literature as early as the fifth century B.C. (Joshi, 1973).

Various terms had been used - such as - the falling disease, fits, seizures and epilepsy. Famous person in the world like - Alexander the great, Julius Caesar, St. Paul Napoleon, Lord Byron and Maupassant had fits (Leleman, 1970), hence these attacks has also been known as 'sickness of the greatest'. Galen (131-201 AD) described epilepsy as "the seizure of mind and sense together with a sudden fall in some with convulsions and in others without". The word 'aura' meaning 'brease' was introduced by the teacher of Galen (Deai, 1966).

Four hundred years B.C., Hippocrates named it as "The sacred Disease". He clearly recognised that epilepsy had its seat in the brain. He gave a very precise and clear description of what today would be called temporal lobe epilepsy or psychomotor seizures.

Epilepsy is defined as "Apasmara" in Indian Medicine, the prefix 'apa' meaning negation or loss and 'smara' meaning recollection or consciousness. Charaka who lived in the second century B.C., described epilepsy
as "paroxysmal loss of consciousness due to disturbances of memory and understanding of mind attended with convulsive seizures". In the olden days many of the clinical features of the epilepsy were known. Aura was recognised and was called 'Apasamara Poorvaroopa'. Charaka, in his 'Nidana', gives list of symptoms which indicate the aura of the disease. Worthy of mention are subjective sensation of sounds, constriction sense in the chest, a sense of darkness, vertigo and dream-like state. Sushruta added to the description of aura, a feeling of persistent flow of thought towards a particular topic which the patient is unable to control. In addition to the description of the actual attack both of these authors mentioned mental symptoms occurring in epileptics (Kannamwarthy and Gurunathan, 1969).

Aretasius, in the second century described the various auras as hallucinations of vision, hearing and taste at the onset of a seizure. Erastus in 1560 also described the aura at the onset of the attack (Desai, 1968).

Frichard (1822) stated that delirium could occur without a fit. Bright (1831) described an epileptic who had no convulsions. The term 'Aura intellectual' was introduced by Falret (1860) to describe various complex psychological phenomena. Association between the mental disorder and epilepsy had been recognised since the beginning of the medical history.
DEFINITION OF EPILEPSY

Epilepsy is regarded as a state of 'Paroxysmal cerebral dysrhythmia' (Gibbs et al., 1937). Penfield (1941) later wrote that one could regard epilepsy 'Physiologically as a tendency to periodic involuntary neuronal explosions;'

Epilepsy is a paroxysmal and transitory disturbance of the functions of the brain which develops suddenly ceases spontaneously and exhibits a conspicuous tendency to recurrence. The most acceptable definition of epilepsy given by Richter (1967) is "Epilepsies are a group of disorders characterized by chronic, recurrent paroxysmal changes in neurologic function caused by abnormalities in the electrical activity of the brain".

Migraine may present as a stereo typed, repeated transient neurological symptoms which may not be followed by headache, mimic a seizure. Patient of migraine may have abdominal pain and vomiting without headache. Vomiting may be the only feature of temporal lobe epilepsy (Shukla and Mishra, 1981).

EPIDEMIOLOGY

Epilepsy is a common melody, prevalence rate i.e. the total cases of epilepsy in a fixed population per thousand at a given time has been found to vary from as low as 2-4/thousand to as high as 10.5/thousand(Table 1).
According to the college of General Practitioner, 1980 epilepsy affects 5/thousand population.

Table 1

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Place of Study</th>
<th>Prevalence rate per thousand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cromeis et al (1960)</td>
<td>England and Wales</td>
<td>4.2</td>
</tr>
<tr>
<td>Browir et al (1966)</td>
<td>Carlisle in Great Britain</td>
<td>5.5</td>
</tr>
<tr>
<td>Mathai (1971)</td>
<td>India</td>
<td>9.0</td>
</tr>
<tr>
<td>Grudinska (1974)</td>
<td>Poland</td>
<td>2.4</td>
</tr>
<tr>
<td>Hauser and Xusland (1975)</td>
<td>U.S.A.</td>
<td>5.3</td>
</tr>
<tr>
<td>Juul-Jensen (1976)</td>
<td>Denmark</td>
<td>6.9</td>
</tr>
<tr>
<td>Fry (1982)</td>
<td>Kent</td>
<td>10.5</td>
</tr>
</tbody>
</table>

These variations in prevalence were because of different criteria for selection of cases, for example, inclusion of only active cases in some studies or inclusion of patients with febrile seizure or simple seizure in other studies.

SEX AND AGE SPECIFIC PREVALENCE RATES FOR EPILEPSY

In the vast majority of studies quoted in table 1 males tend to predominate but in the study of Juul Jensen's and Hauser's studies there were more females and also in the Gosvar's study of 1000 cases the female : male ratio was 13:12. Several authors have suggested that the
high male rate is due to more frequent head injuries. The prevalence rates of post-traumatic epilepsy were estimated in the study female/male at 1.0 – 1.5 per thousand (Zielinski, 1977).

Age specific prevalence rates in several studies (Brew's et al, 1986; Grudzinska, 1974; Juul-Jensen, 1976; and Haerer et al, 1986) found to be showing almost similar pattern. The lowest rates, which usually occur in the first decade, increase in the older age groups and then show a marked drop after 50. Only in Haerer's (1986) study high prevalence rate was found in ninth decade. According to the office of health economics, 1971, the commonest age of onset is 0-4 years. The incidence of initial attacks then declines steadily throughout adult life with a further slight peak especially in males. Over the age of 65 years, similar figures have been reported from many other countries including Switzerland, Holland and United States.

PATHOPHYSIOLOGY

The work of Li and Jasper (1961) has demonstrated clearly that the epileptic process consists fundamentally of hyperactive and hypersynchronous neuronal discharges, the abnormality of neurones appears to be in the instability of cell membranes. An inhibitory feed back mechanism similar to Renshaw cell system of the spinal cord probably exists in the brain (Scales, 1965 and 1967) and is likely to play an important part in preventing the excessive
neuronal discharge that forms the basis of an epileptic attack (Phillip, 1959).

The bilaterally synchronous wave and spike cortical discharges which characterize petit mal attacks seem to originate subcortically, perhaps in the interthalamic nuclei (Jasper et al., 1947) and the resulting impairment of consciousness was interpreted by Williams (1950) as indicating a blockade of different impulses to the cortex. In the light of experimental work and behaviour of Petit Mal in man, Gastaut et al. (1960) suggested that a grand mal seizure seemed to depend on a thalamic discharge, which involved the non-specific reticular structures and was projected to the cortex in what might be considered a generalized recruiting response transmitted along the diffuse cortical projection pathway. For postictal paralysis Efren (1961) gave cogent reasons for suggesting that an active process of inhibition, resulting from persistent subclinical epileptic discharge, was a probable explanation.

Disturbances of consciousness, mood and behaviour which occurred as a result of discharges originating in the temporal lobe were thought to indicate a dysfunction played of that part of the brain (Penfield and Jasper, 1954).

Ward (1961) put forward evidences suggesting that epileptogenic focus were characterized by a standing negative potential of the order of 7-12 mv which could be attributed to a continuous state of dendritic depolarization. Aimone (1961) emphasized that the discharge of the
primary epileptic neurones was only the beginning of the process; before a clinical attack developed there had to be local recruitment of more and more neurones both locally and at a distance.

It has been shown in cat (Prince and Wilder, 1967) that the interictal discharges of an epileptogenic focus with their negative potentials are accompanied by intense inhibitory activity of the neurones in an extensive area of surrounding cortex. This is likely to be a factor in limiting the spread of the discharge.

Fundamentally, epilepsy is a physico-chemical disturbance, and the physico-chemical state of the neurones can be influenced by numerous agencies. Symonds (1939) suggested that the gamma-aminobutyric acid (GABA) might be a natural anticonvulsant formed in the brain. Local lesions might cause seizures either by allowing the local accumulation of an excitatory substance or by depressing the GABA concentration or the tonic inhibitory control of afferent impulses.

GABA and acetylcholine (Ach) have opposite effects upon neuronal excitability so that an imbalance between these two substances within the brain could be a factor predisposing to seizure production.

The balance between Ach and GABA may be upset for instance by pyridoxine deficiency as the latter substance is essential for the synthesis of GABA (Sutherland and Kadic, 1960) - Two receptor sites have been characterised
(Spero, 1987) - GABA/chloride - ionophor/benzodiazepine receptor - complex, and a specific phenytoin receptor.

Weldrum (1982), in reviewing pathophysiology of epilepsy, stressed the importance of metabolic factors like concentration of arterial P\textsubscript{A}O\textsubscript{2}, P\textsubscript{A}CO\textsubscript{2}, glucose, sodium, calcium, magnesium, urea and ammonia and the extent of change in serum osmolality which have been associated with seizure activity in man.

Within the first 30 minutes after a generalized seizure activity, arterial hypertension, a rise in cerebral venous pressure, an increase in cerebral blood flow, hyperglycaemia, hyperkalaemia, haemococoncentration and a low normal P\textsubscript{A}O\textsubscript{2} with a high arterial P\textsubscript{A}CO\textsubscript{2} are usual, while after 30 minutes there is often arterial hypotension, a raised or normal, cerebral venous pressure, a normal cerebral blood flow, hypoglycaemia with persistent hyperkalaemia and see hyperpyrexia (Weldrum, 1982). Prolactin L\textsubscript{H} and FSH may also rise post-ictally, the latter only in females (Dana et al, 1983). Low vitamin D and serum calcium levels are often found in patients with chronic epilepsy (Davia et al, 1983).

**ETIOLOGY OF SEIZURES**

Etiology of the seizures depends upon the age of onset of the seizures. There are different causes operating in the neonates, infants, early childhood, childhood and adolescence, early adult life and late adult life.
According to the age group causes of epilepsy are as follows (Laidlaw and Richens, 1982).

1. **Neonatal (1st month)**: Birth injury, birth anoxia, congenital abnormalities, metabolic disorders, meningitis and other infections.

2. **Infancy (1-6 months)**: As above and infantile spasms.

3. **Early Childhood (6 months-3 years)**: Febrile fits, birth injury, infection, trauma, poisons and metabolic defects, cerebral degenerations.

4. **Childhood and Adolescence**: Idiopathic or primary epilepsy, birth injury, trauma, infection, cerebral degeneration.

5. **Early adult life**: Trauma, tumour, idiopathic or primary epilepsy, birth injury, infection, cerebral degeneration.

6. **Late adult life**: Vascular disease, trauma, tumour cerebral degeneration.

Now the common causes of epilepsy are discussed in brief.

**Head Injury**

Only a small proportion of head injured patients suffer from fits once they have recovered from the acute stage of injury. However, head injuries are so common in occurrence that this comprises a sizable number of patients. Fits due to head injury may develop soon after the injury or months later. The risks of epilepsy are related to
whether the durameter is penetrated or not. In a study (Coveness et al., 1962) incidence of convulsions were found to be 40% in those who suffered from missile wounds of the head. While only about 5% of those with non-missile head injuries developed seizures (Janett, 1962 and 1963). Trauma is more likely to cause partial than generalised epilepsy and may be responsible for fits in about 5-15% of all cases of epilepsy (Gibbs and Gibbo, 1952). Epilepsy after head injury is divided into early or late epilepsy. Jennet (1962) proposed that definition of early epilepsy should be reserved for the fits in the first week after injury. Patients who develop fits in first two weeks after injury, some 27% will continue to have persistent recurrent seizures, but in those whose fits develop after two weeks or later, risk is of order of 70% (Jennet, 1969). Jennet et al (1973) have above to identify factors likely to be associated with persistent post traumatic epilepsy after head injury. In investigation of 800 patients with epilepsy after non-missile injuries there were over 400 with early epilepsy, over 400 with late and 90 with both (Laidlaw, 1982). It is late epilepsy which is usually meant when the term traumatic epilepsy is used, because it is this which constitutes persisting disability. In a study (Annegers et al, 1980) of 2747 patients of mild head injury, incidence of seizures was not significantly greater than in general population.
Brain Tumour

Brain tumour is considered to be an important cause of late onset of epilepsy from a very long period (Parker, 1930 and Penfield et al, 1940). In fact brain tumours are responsible for later onset epilepsy only in about 10% of all cases (Sheehan, 1958; Raynor et al, 1959; Hylister and Rakkenberg, 1963; Jum, 1964a). Incidence of tumours rises steeply in the cases of partial fits, where a figure of 30-40% is more appropriate (Raynor et al, 1959; Jum and Yeardall, 1963). However, the incidence in complex partial seizures is not that high being about 15% (Currie et al, 1971). Meningiomas and benign gliomas characteristically cause seizures, while malignant gliomas do so less frequently. The incidence of fits is 67% in meningiomas, 70% in astrocytomas, and 37% in malignant gliomas (Penfield et al, 1940). Secondary deposits from lung and breast is also a common cause of fits (Laidlaw, Richens, 1982). Fits are commonest in tumours of fronto-parietal region and very rare with lesions in thalamus, basal ganglia or parapituitary area (Williams, 1963).

Tumour as a cause of fit in children is uncommon, partly because most childhood tumours arise in non-epileptogenic areas such as the cerebellum, brain stem and diencencephalic and also because so many other epilepsy appears in childhood.

Local cerebral lesion - other than tumours are also responsible for epilepsy. Among the many lesions
described at autopsy have been chronic localized encephalitis (Rasmussen et al, 1938), focal cortical dysplasia (Taylor et al, 1971), neuronal heterotopias, hemangiomas, meningiomas, and other vascular malformations (Matheron, 1992; Lehman et al, 1983). Arteriovenous malformation causes fits usually focal in nature in about 40% of cases (Peterson et al, 1936). Fits are said to be rare in subdural haemorarhages.

**Cerebrovascular Disease** - is an even more common cause of adult onset fits than tumour (Dodge et al, 1934). It may be responsible for 10-20% cases of adult onset epilepsy, but after the age of 50 the figure is 50% or more (Juell-Jensen, 1964a and Wardcock and Cobgrove, 1964). It is estimated that as many as 25% of those with cortical infarcts will have fits (Richardson and Dodge, 1954), although the incidence of fits in non-embolic cerebral infarcts in general is about 5% (Louis and McDowell, 1967).

**Migraine** - while loss of consciousness in an attack of migraine, often at the height of the headache, is usually syncope, there is a slightly increased incidence of epilepsy in migraine sufferers even in those who have no evidence of a cerebral lesion. A possible role of tyramine in the physiological mechanism of epilepsy and migraine was postulated by Scott et al, (1972).

**Perinatal asphyxia** - has been found to be a very common cause of epilepsy. In a study of Brown et al (1976)
out of 94 infants who suffered from asphyxia, 32 had birth injury and 40 had convulsions.

**Hereditary predisposition** — plays a considerable part in idiopathic epilepsy. EEG abnormalities are six times more common in relatives of epileptics than the controls (Lennox et al, 1948). Lennox (1947) believed that the dysrhythmia is inherited as a mendelian dominant trait although the predisposition is clearly of relatively low penetrance (Brown, 1982).

**CLINICAL CLASSIFICATION OF SEIZURES**

The terminology and classification of seizures has evolved over many years, creating a variety of interchangeable and confused descriptive terms.

Traditionally, attacks of epilepsy, whether idiopathic or symptomatic have been divided into major epilepsy (Grand mal), minor epilepsy (Petit mal), focal epilepsy (Jacksonian epilepsy), temporal lobe epilepsy (psychomotor epilepsy) and myoclonic attacks (Janz, 1969). However, this descriptive classification has become increasingly unsatisfactory for many reasons. Thus there are many forms of minor epilepsy with or without transient impairment of consciousness, which are not true petit mal; in epilepsy of focal onset, depending upon the site of origin in the brain, a variety of motor, sensory, behavioural and psychomotor manifestation may be found but if the epileptic discharge spreads rapidly to become generalised, a major attack may occur and the focal symptoms
then constitute merely the aura of the major attack. Temporal lobe epilepsy is now more generally known as complex partial epilepsy (Penry and Daly, 1975).

Many new classifications have been proposed notably by the International League Against Epilepsy (Gastaut, 1969); Sutherland and Eadie (1980) and the comprehensive clinical and electroencephalographic classification recommended by the International League Against Epilepsy, the World Federation of Neurology and the International Federation of Societies for Electroencephalography and Clinical Neurophysiology.

A simpler working classification, from Marsden and Reynolds (1982) is given as follows. They point out that it is and always will be, impossible to create a single code to cover three basically incompatible systems of classification viz one according to the clinical signs and symptoms in the attacks; one relating to the anatomical and physiological evidence as to its source; and one defining aetiology.

Classification of Epilepsy
(Marsden and Reynolds, 1982)

I. Generalized
   - Tonic-clonic
   - Tonic
   - Atonic
   - Absence
- Atypical absence
- Myoclonic

II. Partial (Focal)
A. Without impairment of consciousness (Simple partial seizures).
B. With impairment of consciousness (Complex partial seizures).
   i) With motor signs (e.g. Jacksonian variants).
   ii) With somato-sensory symptoms
       (e.g. olfactory, visual).
   iii) With autonomic features (e.g. epigastric sensations).
   iv) With psychic symptoms (e.g. fear).
   v) With automatisms (complex partial seizures only).

III. Partial seizures Secondarily Generalised

Clinical or electrical evidence of focal discharge during or after the generalised seizures.

IV. Unclassifiable

Seizures which cannot be classified because of incomplete data.

PHENOMENOLOGY

GENERALIZED SEIZURES

One of the most common type of epileptic attack is the generalized seizures. Some of these appear to be primary generalized seizures and others are the result of
secondary generalization of partial seizures.

**Tonic Clonic grand mal seizures**

The classical attack of epilepsy consists of preconvulsive symptoms, aura, convulsion and the post-convulsive phase. In pre-convulsive symptoms, some patients experience a warning signal for hours or even a day or two. These vague symptoms include irritability and depression, abnormal feelings referred to the head, giddiness and sudden myoclonic twitches. In other cases patient had no warning but becomes unconscious at once. Aura is less common in major seizures than in the seizures of focal onset.

Primary generalized seizures as described by Dichter (1967) usually start without warning, although some individuals sense a vague nonspecific sense of the impending event. The onset is heralded by sudden loss of consciousness, a tonic contraction of the muscles, a loss of postural control, and a cry produced by a forced expiration caused by contraction of the respiratory muscles. The individual falls to the floor in an episthotonic posture, often sustaining injury and remains rigid for many seconds. There may be cyanosis as respiration is inhibited. Soon a series of rhythmic contractions of all four limbs occur. The clonic phase can last for a variable period of time and ends when the muscles relax. The individual remains conscious and unarousable for a period of minutes or longer. There is usually a gradual return to consciousness and often there is a period of disorientation
during recovery. Post-ictally, headache and drowsiness are common sequelae and the individual may not return to baseline functioning for days.

The clinical features of a major fit in a child are similar to those in the adult. However, children often do not go through the postictal phase of the coma, confusion, headache and sleep, but usually recover completely within minutes (Laidlaw and Richens, 1982).

**Tonic Seizures**

Tonic seizures are a less common form of generalized seizures which consist of spasm of the limbs or torso often with deviation of the head and eyes towards one side (Dichter, 1987). They are not followed by clonic phase and are often of shorter duration, usually 10-20 seconds (Gastaut et al, 1963).

**Absence (Petit mal) Seizures**

Petit mal is now defined as brief absence of attacks, occurring almost always in childhood (6-14 years of age) and associated with characteristic EEG paroxysms of three per second, bilateral, synchronous spikes and wave discharge (Gibbs et al, 1935). It rarely appears for the child loses his consciousness, his eyes stare, and he may show minor movements such as blinking or twitching of the face and arms but he does not fall. Suddenly consciousness is regained but there is total amnesia during the brief period of the attack. The patient looks around for a
moment but then resumes his previous occupation. If engaged in conversation before the attack, he will have missed a sentence or so (Laidlaw and Pichems, 1982). Such classical petit mal attacks may occur very frequently even as often as hundred or more times daily (Lennon, 1945; Gibberd, 1966; Delby, 1969). Automatic minor motor phenomenon may resemble complex partial seizures (Penry, 1975). Other possible associated clinical features of absence seizures include myoclonus, version of the head and conjugate deviation of the eyes, decreased postural tone and autonomic changes. Approximately 50% of patients with absence seizures also experience generalized tonic-clonic seizures (Gibberd, 1966 and Lagaresi, 1973). Absence status may last for hours, with clouding of consciousness and reduced accuracy of responses (Manuel, 1983). There is usually no period of post ictal confusion. Status of petit mal does not appear to have any deleterious prognostic implications (Andermann, 1972).

The onset of absence is rare before the age of three years; it is most common between four and eleven years of age, and unusual after age of twenty years (Andermann, 1972).

Atypical Absence Seizures

The association of the EEG pattern of “Petit mal variant” (Gibbs, 1971) or “Slow spike wave” (Lennon, 1949) with certain clinical aspects led to the concept of a syndrome variously named “severe myoclonic epilepsy of
early childhood with slow spike and wave" (Sorel, 1964),
Childhood epileptic encephalopathy with diffuse slow spike
waves" (Clastaut, 1966), Lennox Syndrome (Schneider, 1970)
and "Lennox-Clastaut Syndrome (Naidermeyer, 1969; Castaut,
1979). The term Lennox-Clastaut Syndrome was proposed by
Clastaut et al (1966) to designate a form of epilepsy of
childhood with frequently repeated fits of several types
and an interictal ECG pattern of diffuse spike–waves at
a rhythm of approximately 2 Hz, previously described as
petit mal variant (Gibbs and Gibbs, 1982). The role played
by genetic factors is controversial. The incidence of
family history of convulsive disorder is low (2.5%) in
some series (Chvare and Alcardi, 1972) and very high(50%)
in other series (Deese et al, 1970). Mental retardation
is present from the onset in 20–60% of patients and in
half of the cases it is severe. The prognosis of Lennox
Clastaut Syndrome is poor. According to Castaut (1973),
80% will continue to have seizure. It is characterized by
onset of seizures early in childhood. More than one
variety of generalized seizures occur; predominantly tonic
tonic–clonic, atonic, akinetic, absence and myoclonic
(Chvare, 1972; and Blume, 1973). Mental retardation is
often present. The ECG contains generalized sharp and slow
wave complexes (Markand, 1977). The minimal age expression
of the Lennox-Clastaut syndrome is between 1 and 5 years
with a slight preponderance of males (Scile-Levissari, 1977).
During 'minor' status epilepticus i.e. the prolonged episodes of frequently repeated akinetic or myoclonic seizures, the patient may be unable to maintain the head erect. The accompanying difficulty in eating and swallowing resembles pseudobulbar palsy (Doose, 1970). Delayed psychomotor development has been found in 20–30% of patients (Blume, 1973). Additional abnormalities in the neurological or ophthalmological examination are found in 30–59% of patients (Markand, 1977). Mental deficit was especially prevalent in patients who had infantile spasms, tonic seizures or minor status epilepticus (Blume, 1973).

**Atonic Seizures**

Atonic seizures are brief losses of consciousness and postural tone not associated with tonic muscular contractions. The individual may simply drop to the floor without apparent cause. Atonic seizures usually occur in children and are often accompanied by other forms of seizures. The EEG contains polyspikes and slow waves. The "drop attack" of atonic seizure needs to be distinguished from cataplexy seen in narcolepsy (where the patient remains conscious), transient brain stem ischaemia or sudden rise in intracranial pressure (Dichter, 1987).

Sudden falls have also been described in patients with partial epilepsies especially partial complex seizures (Caffi, 1973 and Delgado, 1982). Stress had been given upon the serious nature of partial epilepsies with drop attack because they often resist drug treatment (Rogar, 1981 and Poelo, 1983).
**Myoclonic Seizures**

Myoclonic seizures are sudden brief, single or repetitive muscular contractions involving one body part or the entire body, in which seizure is accompanied by a violent fall without a loss of consciousness. Myoclonic phenomena is a very common experience and epilepsy is only one of their many causes. Many people experience myoclonic jerkings when falling asleep. Myoclonic attacks that are epileptic in nature have been regarded as fractionated or miniature attacks of grand mal (Gastaut and Fischer-Williams, 1959b). Marden et al (1979) pointed out that myoclonus is used as a descriptive clinical term which has no physiological, etiological or therapeutic implications. Myoclonic epilepsy of adolescence usually begins at about puberty (Sevons, 1977), rarely before the age of 9 years, with myoclonic jerks involving the head, arms and upper trunk.

**Infantile Spasm or Dysrhythmia**

This form of primary generalized seizure occurs in infants before one year of age. Peak age is 3 and 6 months (Sevons, 1964). It consists of brief synchronous contractions of the neck, torso and both arms (usually in flexion) lasting 2-10 seconds. Infantile spasms often occur in children with underlying neurologic disease, such as encephalopathy or tuberous sclerosis, but can rarely occur in an otherwise normal infant. The prognosis of children with this type of seizure disorder is grave and approximately 90% develop mental retardation in
additions to their seizures. The EEG is characterized by a very disorganized background, random high voltage slow waves, spikes and burst suppression (hypsarrhythmia). The spasms and hypsarrhythmia tend to disappear over the first of generalized seizures. Psychomotor development may have been delayed before the onset of the infantile spasm, but more often the retardation is noted after their onset.

The association of infantile spasms, arrest of psychomotor developments and hypsarrhythmia has been called the West's syndrome (Gastaut, 1971).

PARTIAL SEIZURES

Simple Partial Seizures

Simple partial seizures can occur with motor sensory, autonomic or psychic symptoms without impairment of consciousness.

The typical partial motor fits consists of onset of tonic spasms followed soon by repetitive twitching, usually in the angle of mouth, thumb and index finger, or great toe which then spreads in an orderly manner. The convulsive movements may remain confined to the site of onset or may spread to involve one half of the body and may terminate in a typical grand mal fit with loss of consciousness. If the left hemisphere is the source speech may be last during the attack (Laidlow and Richens, 1982). Very rarely true inhibition of movement has been described as an epileptic event (Veffron, 1961). Fits may be followed by Todd's paralysis. In patients, especially
in adults, a prolonged Todd's paralysis may be due to
tumour causing the fit and in such cases if it persists
for more than 48 hours, full investigation is required.
Another not uncommon form of motor seizure is the adver-
sive attack due to discharges arising in pre-motor areas
of the frontal lobe (Penfield and Welch, 1951). Typically
the head and eyes are forced away from the affected
hemisphere, usually with preservation of consciousness.

Somatic sensory seizures characteristically
commence in one of the preferred sites, such as thumb or
mouth and show a spread of march. Complaint is usually of
numbness or pins and needles usually motor phenomenon is
associated.

Visual fits are due to discharges in an occipital
pole and usually consist of unformed simple visual pheno-
mena such as spots, flashes of light balls on fire through-
out the visual field. Other types of visual seizures which
are more common, are associated with temporal lobe epilepsy.

Auditory, gustatory and olfactory symptoms are
usually found with temporal lobe seizure.

Visceral aura, consisting of abnormal feelings
in epigastrium may be accompanied by autonomic phenomena,
such as tachycardia, sweating, pupillary dilatation and a
fall in blood pressure (Von Buran and Ajmonomarson, 1960).
Whether this sequence of autonomic events occurring
briefly and repeatedly, but without any other evidence of
epilepsy, is a form of restricted autonomic epilepsy, is
debatable, but has been found to respond to antiepileptic drugs (Fox et al., 1973).

Involuntary micturition can occur in isolation as a possible manifestation of seizure discharge, often with loss of awareness such that patient finds himself wet without prior knowledge of events (Maurice-William, 1974).

**Complex Partial Seizures**

Complex partial seizures are episodic changes in behaviour in which an individual loses conscious contact with the environment. The onset of these seizures consists of variety of auras.

These are more commonly known as psychomotor seizures. The term psychomotor epilepsy was first used by Van Gieson (1902) and again by Gibbs, Gibbs and Lennox (1937). Lennox (1951) suggested the name "temporal lobe epilepsy". Psychomotor epilepsy denotes clinical psychomotor attacks, irrespective of the presence and location of a focus while the term temporal lobe epilepsy means epilepsy with a temporal lobe focus in the EEG respective of the clinical seizure pattern (Stevens, 1966). Gibbs and Gibbs (1964) reported 80% incidence of temporal lobe foci on EEG of his patients with clinical psychomotor epilepsy. As against this typical psychomotor seizures may be associated with frontal epileptic foci (Penfield and Kristiansen, 1951). The reported incidence of temporal lobe epilepsy within the total epileptic population varies from 23% (Chadday et al., 1966), 26% (Penfield
and Jasper, 1954), 34% (Selinski, 1974), 40% (Castañet et al, 1975), 2.7% (Bagadia et al, 1973), 17.5% (Mohan, 1974) and 25% (Gopalkrishnan et al, 1968).

Majority of the patients had the onset of seizure below the age of 20 years (Shukla et al, 1979), while Gibbs et al (1948) had reported only 9.3% of the epileptics below age of 20 years. Currie et al (1971) reported maximum incidence of seizures in the third and fourth decades. However, 62–75% of cases of Aird et al (1967), Viramani and Sawhney (1966) and that of Peddy (1971) had their first attack in first two decades. Phenomenology of complex partial seizures is quite varied. More than one type of clinical seizure pattern may be exhibited by a patient of temporal lobe epilepsy (Falconer and Taylor, 1970).

1. Auras only.
2. Absences.
3. Psychomotor attacks.
4. Falling attacks.
5. Grand mal attacks.

It is not possible here to discuss every group separately and in detail. Fear or anxiety is the most common premonitory emotional experience (Williams, 1956 and 1965) and but some times the attack may cause pleasure of considerable intensity, as was described by Dostoevsky (1935). The onset of these seizures may consist of any of the variety of aura. Aura has been found in 60% of cases (Shukla et al, 1979). The commonest were visual halluci-
nation followed by vertigo and emesis sensation. Unusual smell (as of burning rubber); a feeling that the current experience had happened before (déjávù); a sudden intense emotional feeling; a sensory illusion such as micropsia or macropsia or specially formed sensory hallucinations, could be other type of aura. Paroxysmal vomiting as a sole manifestation of temporal lobe epilepsy has also been reported (Shukla and Mishra, 1981).

Olfactory and gustatory auras once said to be diagnostic of temporal lobe seizures, are in fact rare (De Jong, 1957 and Dennerill, 1964).

A curious feature is the tendency of turn towards religion (Esquirol, 1838; Marel, 1960; Howden, 1972–73; Bowen, 1919; Slater et al, 1963 and Slater and Roth, 1969). It occurs most commonly in temporal lobe epilepsy, because in them the aura often takes the form of some religious experience (Narayana and Robertson, 1985; Slater et al, 1963 and Sedman and Hopkinson, 1966). Dowhurst and Beard (1970) reported 6 cases of temporal lobe epilepsy with "religious conversion experiences".

During complex partial seizures there may be a cessation of ongoing activity with some new motor manifestations, which usually follow aura. Though epileptic attack can cease just after aura, sometimes generalized tonic–clonic seizures may be the final event in which usually there is amnesia for the aura.

Among the clinical features of temporal lobe seizures, several authors include "somatic manifestations"; localized or generalized, tonic, clonic, tonic–
clonic or postural manifestations (Leen, 1960). In a study (Rossi, 1984) 10% of psychomotor epileptics had motor symptoms; lateral deviation of head and eye occur frequently (King and Ajmone-Marson, 1977). Hypotonia may occur causing the patient to fall down (Geier et al., 1977).

Additional motor activity may be in the form of automatisms which occur in the form of smacking of lips; running at the onset of seizure (Chen and Froster, 1973). This can be in association of gelastic epilepsy, i.e. excessive laughter (Comper et al., 1970); picking at clothes walking aimlessly. Sometimes, complex acts like motor driving or performing on musical instruments is automatic motor phenomenon; laughing or crying can occur (Offen et al., 1976). Sexual automatisms in the form of masturbation or pelvic thrusting has been reported (Spencer et al., 1983). A significantly greater number of temporal lobe epileptics do have emotional disturbances in childhood and psychiatric abnormalities in later part of life, in comparison to patients with grand mal epilepsy (Shukla et al., 1979).

Abnormal sexual behaviour associated with temporal lobe epilepsy has frequently been described in man (Gastaut and Colombo, 1954; Marchini and Cinisi, 1957 and Heiron and Saunders, 1966) and in animals (Kluver and Busé, 1939). Almost all the types of sexual perversion have been observed in association with temporal lobe epilepsy.
The occurrence of hypersexuality in association with temporal lobe epilepsy is rather rare (Taylor, 1966b). However, Blumer (1969) reported 7 cases of temporal lobe epilepsy who had distinct episodes of hypersexuality, usually following abrupt cessation of seizure activity. Only one patient out of 100 in the series of Taylor (1966b) was hypersexual. A large number of temporal lobe epileptics were found to be hyposexual (Shukla et al., 1979).

Secondary Generalization of Partial Seizures

Simple or partial complex seizures can progress to get generalized with loss of consciousness and often with convulsive motor activity. This may occur immediately or after many seconds or a minute or two. In addition, many patients with focal seizures have generalized seizures without an obvious initial focal component and are difficult to distinguish from primary generalized seizures. The presence of an aura or the observation of any focal feature at the onset of generalized seizures or the presence of postictal focal neurological deficit (Todd's paralysis) are important clues to the focal origin of seizure (Dichter, 1967).

Epileptic Reflex Epilepsy

Sometimes an attack can be excited by some form of external stimulation. This may be a sudden loud noise (acoustico-motor epilepsy) or music (musico-genic epilepsy) or a visual stimuli e.g. reading (reading epilepsy)
(Sinjal, 1957; Stoupel, 1968) or viewing television
(television epilepsy) (Brooks and Ilausch, 1971). In some
cases attacks are precipitated by speaking and writing
(language induced epilepsy) (Geschwind and Sherwin, 1967)
or by blinking when starting to speak (Tsoano, Parrino,
Manconi and Mancia, 1983).

**INTERICITAL BEHAVIOUR IN EPILEPTICS**

The association of specific behavioural changes
with epilepsy has been questioned for long time (Freud,
1950; Lennex, 1940; Geschwind et al, 1980 and Reynolds,
1983). Community surveys in unselcted populations have
confirmed an increased incidence of psychological distur-
bances in epileptics (Rutter et al, 1960 and Gudmundson,
1966). Recently use of objective measures of mental/
intellectual function, has been tried to document whether
characteristic changes in cognitive or behavioural
function occur in patients with epilepsy (Doddill, 1978
and Giordani et al, 1985).

Dennis et al (1985) found a significantly low
level of intellectual functions in patients with secondary
generalized tonic-clonic seizure in comparison to other
epileptic population. It appears that for newly diagnosed
unmedicated epilepsy patients, the level of intellectual
function compares favourably with that of general popula-
tion. Lower levels of intellectual performance have
frequently been reported for epilepsy samples (Klove and
Mathews, 1965; Johnson et al., 1974 and Reynolds, 1983). This may have resulted, in part, from inclusion of more chronic, poorly controlled and highly medicated epilepsy patients.

"Even with the normal levels of intellectual performance for the epilepsy sample and the close age and education match between the control and epilepsy groups, significant comparative differences were consistently observed between the two groups (Laidlaw and Richens, 1982).

**Epilepsy and Psychosis**

Many authors have, however, failed to find any difference between the incidence of psychosis in different types of epilepsy (Alstrom, 1950; Vielle and Henriksen, 1958; Guerrant et al., 1962; Small et al., 1962; Juul-Jensen, 1964; Stevens, 1966 and 1973). Guerrant and coworkers (1963) evaluated 32 patients with psychomotor epilepsy, 36 with grand mal and 26 control cases with chronic medical illness, not involving brain. The study was designed to test the hypothesis that functional psychiatric disturbances were more common in psychomotor epilepsy. The hypothesis was not confirmed. Standage (1973) studied 53 patients of epilepsy in a mental hospital. Of these, 8 were psychotic.
LABORATORY INVESTIGATIONS

In a patient in whom we are suspecting epilepsy our aim is to confirm its diagnosis and then to decide its cause. The initial step in investigation of the case of epilepsy is a thorough history both from the patient and a witness and general and systemic examination. A number of causes can be recognised in the clinic itself. A history of birth trauma, or anoxia combined with body asymmetry, such as small thumb or toe will point out to cerebral damage in early life. Tubercous sclerosis and Sturge-Weber syndrome will be indicated by the appropriate skin lesions and congenital or genetically determined syndromes may be suggested by a characteristic facial appearance or other signs i.e. mongolism. In some cases examination is directly concerned with detecting signs of focal brain damage or raised intracranial pressure.

INITIAL OUT PATIENT INVESTIGATIONS

Initially simple tests are required. In all patients, X-ray skull and EEG are required. Now various tests had been done such as routine blood count, VDRL test for syphilis and biochemical test for blood sugar and calcium. Even though one can rarely detect the cause of fit by these tests, however, these base line investigations should be performed in all the cases. X-ray chest is a necessary to exclude bronchogenic carcinoma (Laidlaw and Richens, 1962). Evidence of tuberculosis can also be seen by it. In some patients an X-ray of the
thigh may show the calcified cysts of cysticercosis. In idiopathic epilepsy the CSF is normal except that during or after frequent fits of an attack of status there may be a rise in pressure. A consistently raised CSF proteins and a pleocytosis should suggest that the epilepsy is symptomatic. Usually the X-ray skull is normal in the patients but at times it may show calcification in tumor, evidence of raised intracranial pressure or other lesions and bony changes of meningioma may be detected. Pinea1 shift may be the indicative of a mass lesion. Asymmetry, particularly of middle cranial fossa may point to a long standing atrophic lesion. Such informations are crucial in a few cases and a skull X-ray should be undertaken in every epileptic patient (Laidlaw and Richens, 1962).

ELECTROENCEPHALOGRAPHY

Historical aspects - In 1914 Cybulski recorded an epileptic seizure caused by cortical stimulation in a dog. All the earlier works were done on animals and it was not until 1929 that Hans Berger published the first report of the electroencephalogram of man. Pen writers were available in 1940's and made it possible to have an immediate permanent record. The other great technical advance at this time was the use of the differential amplifier which eliminated much of the interference from external sources (Matthews, 1934; 1938; Tenniere, 1938; Farr and Walter, 1943). Since 1940, there have been only little change of basic technique.
INTERICTAL ELECTROENCEPHALOGRAPHY

It is seldom that a satisfactory EEG recording is obtained during the attack of fits because of the muscle and movement artefacts that occur. It is unusual for a routine EEG recording to coincide with an actual seizure and it is necessary therefore to depend on interseizure pattern for diagnostic assistance in most cases of epilepsy.

The diagnostic role of interictal EEG in epilepsy has been questioned by many authors (Goodin, 1984). Diagnosis of epilepsy is mainly clinical (Kellaway, 1981). Spike and wave and similar epileptiform discharges are sometimes found in the EEG of individuals who are not known to have epileptic attacks of any kind, emphasizing that epilepsy must always be diagnosed on clinical grounds and not solely on EEG evidence. Zevin and Ajmone Marsan (1968) found that of 6497 non-epileptic patients examined at the National Institutes of Health, Bethesda, 142 (2.2%) had spikes or sharp waves with or without associated slow waves in their EEG. Thus the role of EEG is restricted to classify the seizure disorders, localising the epileptogenic focus and guiding prognosis (Mathews, 1964 and Critchley, 1978). In a study by Goodin (1984) in general population, 60% of epileptic patients had positive epileptic form of activity. On the other hand only 4% of non-epileptic had positive EEG. In another group who were suspected to have epilepsy, 93% positive EEGs belong to epileptic group. Absence of epileptiform activity in
EEG reduces the chances for epilepsy in the suspected cases from 50% (clinically) to 33%.

**INTERSEIZURE EEG IN GENERALIZED EPILEPSY**

Interseizure epileptiform discharges in generalized epilepsy are always widespread, bilaterally synchronous and more or less symmetrical. The degree of abnormality of interseizure record is often related to the frequency of attacks and the time lapsed after the last attack.

Abnormalities found in EEG, in generalized epilepsy, are of many varieties. They are usually non-specific but in some conditions are specific. In generalized tonic-clonic epilepsy various abnormalities are found like spike and wave, polyspikes, sharp waves, slow waves or sharp and slow wave complexes, but they must essentially be bilaterally synchronous and almost symmetrical. Absence seizure show typical EEG pattern i.e., bilateral spike and wave activity of three bursts (Ferry, 1975). These bursts show high degree of bilateral voltage symmetry, spatial distribution and synchrony.

Kellaway (1963) showed the relationship of age specific epileptiform EEG patterns to clinical seizures in children. Interictal EEG recording of patients with infantile spasm is almost always severely abnormal and typically demonstrates the abnormality known as hyperarrhythmia (Gibbs et al., 1952). This pattern rarely persists beyond the age of 4-5 years.
Various authors used different criteria to define Lennox-Gastaut syndrome. Some workers selected the EEG pattern as main criterion (Blume, David and Gomez, 1973; Wiedamayr, 1969; Barkand, 1977) and included all the clinical correlations of the diffuse slow spike wave pattern. Others base their description mainly on a clinical picture dominated by falls and massive myoclonia (Kruse, 1968; Doose et al, 1970) and use the clinically descriptive term of 'myoclonic-astatic petit mal' which has often and wrongly been considered synonymous with that of the Lennox-Gastaut Syndrome. Still others (Aicardi, 1973; Gastaut, 1973; Jeavons, 1977) used combined electroencephalographic and clinical criteria.

Drop attacks or akinetic seizures are associated with various types of interictal EEG abnormalities like diffuse slow spikes-wave discharges; with or without focal epileptic abnormalities (Pasto, 1935). EEG accompaniment of myoclonic attack often resembles the spike and wave phenomenon in so far as they consist of an association of high voltage spikes and slow waves which are bilaterally synchronous, but the episode seldom have the regularity of true spike and wave. In epileptic subjects these myoclonic discharges may be provoked by sudden noises or photic stimulation (Kiloh, 1981).
INTERSEIZURE PATTERNS IN PARTIAL EPILEPSY

The important features of interictal EEG in partial epilepsy is the presence of localised spikes and with a bipolar montage have a focal origin of which is usually seen in the form of phase reversals. They are usually monophasic but may be biphasic or triphasic. The duration of these waves is less than 50 milliseconds. In many patients the duration of discharge lies between 50 - 200 milliseconds and it is customary for these to be called as sharp waves. Sometimes the discharge originates in one hemisphere and is reflected as a mirror focus in the appropriate area of contralateral cortex; this is especially seen in frontal discharges and those originating from the medial aspects of the temporal lobe, owing to the richness of the commisural connections existing between homologous areas in the two hemispheres (Kiloh et al, 1982). Odum et al (1956) have shown that discharges are so closely synchronised that transcerebral connections cannot account for them. Morphology of the EEG activity in partial seizures may be sequential spikes and sharp waves (Jasper, 1949; Gastaut, 1953; Kiloh et al, 1972; and Klus, 1975). Although a spike or sharp wave focus provides the most frequent EEG evidence of a cortical epileptogenic lesion, the discharge may comprise spikes and slow waves of paroxysmal activity in alpha, theta, or delta ranges. Mattson and Knott (1977) pointed out that sharp waves are less reliable indicators of a cortical epileptogenic lesion than spikes. At times multiple independent foci
called as 'shifting foci' are seen. Migratory foci are particularly found in children. The initial focus may be superseded by another elsewhere.

The incidence of epileptiform abnormality is greater in epileptic children than in epileptic adults because there is tendency of a epileptic focus to move forward or tendency to diminish in frequency or disappear with increasing age, as seen by improvement in EEG (Gibbs and Gibbs, 1953).

In temporal lobe epilepsy, the medial aspect of the temporal lobe is the common site for epileptogenic foci due to its susceptibility to anoxic damage either at birth or as a result of febrile illness and because the threshold to stimulation of this region is the lowest of any area of cortex. When the site of origin of epileptiform discharge is on the convexity of one of the temporal lobe its discovery and localization are easy (Engle et al, 1975). Deep temporal lobe lesion can given rise to distant spike. In the series of temporal lobe epilepsy, reported by Jasper, Partuissot and Flamigia (1951) the focus was unilateral in 34%, bilaterally synchronous but consistently asymmetrical in 24%, bilaterally synchronous and of equal voltage in 19%, whilst in remaining 23% bilateral independent foci were present.

When a localized lesion gives rise to bilaterally synchronous and often symmetrical discharge the phenomenon is referred to as secondary bilateral synchrony or
secondary generalisation. Jasper (1952) and Fenfield et al (1954) showed that it was possible for bilaterally synchronous abnormalities to appear in the EEGs of the patients with epilepsy caused by unilateral parasaggital, orbito-frontal or anterior temporal lesions.

**PROVOCATIVE TECHNIQUES**

Clinically significant changes in the EEG may be evoked by certain procedures as hyperventilation, photic stimulation and sleep.

1. **Hyperventilation**

   Here patient is asked to take deep breaths at 20 respiratory rate for 3–4 minutes. The usual response to hyperventilation is the appearance of bilateral slow activity, due to the fall in arterial carbon dioxide. This response is potentiated by mild hypoglycaemia occurring after three hours without eating. Changes are seen more in temporal lobe epilepsy and in children.

2. **Photic Stimulation**

   This procedure activates the epileptogenic lesions in the occipital region evoking focal sharp waves or slow waves or both. In this technique repetitive flashes of light are presented at rates ranging from 15–30 per second. The finding is rare since lesions in the occipital lobe are infrequent.
3. Sleep

In drowsiness and the early stages of sleep, generalized seizure discharges are sometimes enhanced. Niedermeyer (1955; 1966) claimed that patients with generalized seizure often show massive irregular bursts of spikes which are associated with K complexes. Pharmacological provocation have little or no importance because they can provoke epileptiform activity in normal individuals. The enthusiasm of Gibbs and his coworkers (1958) for sleep as a method of provocation is not shared by all, but its value is greatest in cases of temporal lobe epilepsy. Merlis, Grossman, and Henriksen (1951) found that about half their patients with psychomotor epilepsy showed focal epileptiform discharges during sleep, as compared with about one third in the alert state, but Clear, Tsai, and Naddad (1958) pointed out that in only 7% of their patients sleep recording was necessary to obtain EEG confirmation of their clinical diagnosis of temporal lobe epilepsy. On the other hand Silverman and Morisaki (1958) considered that sleep recording was crucial in confirming the diagnosis in almost 20% of their mixed epileptic group. A more recent study by Niedermeyer and Roua (1972) found that 23(31%) of 73 patients showed epileptic discharges only during sleep, whilst in another 33(45%) of these were enhanced. As a rule the patient of suspected temporal lobe epilepsy who has a nonspecific routine EEG and who does not fall to sleep spontaneously, should have a sleep recording carried out before being subjected to
the greater ordeals of the insertion of special electrodes. Any hypnotic may be used. Sleep recordings are usually obtained by depriving the patient of sleep for 24 hours (Pratt et al, 1969). Kriebel and Schlager (1973) used this technique in patients suspected of having seizures. Of routine EEG 8.6 per cent showed focal epileptiform discharges, while of the sleep recording, 18 per cent showed such discharges. These mostly occurred as the patient was falling asleep. Scoilo Lavissari, Pratt and de La Cruz (1975) found that 138 of 294 epileptic patients whose routine records lacked supporting evidence, showed epileptiform discharges after sleep deprivation for about 24 hours. A significant increase in spike wave activity has been observed by Burr et al (1986) just after falling asleep and (less pronounced) after awakening in the morning. Malara (1984) observed that micro arousals are especially related to the production of the spike wave paroxysms.

LUMBAR PUNCTURE

This procedure gives no useful information in a usual case of epilepsy. It is required when it is suspected that the epilepsy may be due to infection, encephalitis, or stroke or other intracranial disorders.

CONTRAST METHODS

Contrast methods are commonly used for the cases of computerized transaxial tomography (CT), carotid angiography pneumoencephalography and ventriculography.
a. **CAROTID ANGIOGRAPHY**

In this method we inject an iodine containing contrast media in common carotid artery. Here we can visualize cerebral arteries and their branches.

b. **PNEUMENCEPHALOGRAPHY AND VENTRICULOGRAPHY**

Injection of air into a lumbar subarachnoid space in the patient with sitting position permits visualization in considerable detail of the size and position of the ventricles and subarachnoid space (upper spinal and cerebral) and indirectly the structures which lie between the ventricle and meninges.

Except CT scanning, other contrast methods are not used now a days as they are invasive procedures and less informative than CT scan (Bolshauer et al, 1977).

**Computerized axial Tomography CT Scan**

It is an harmless procedure which is now being used widely. It differentiates epidural, subdural and intracerebral haemorrhages and deformities of the ventricular system from mass lesions and demonstrates tumours, abscesses, granulomas when done after an intravenous injection of meglumine diatrizoate (Renografin), or other contrast medium, as well as areas of brain oedema and infarction, hydrocephalus and brain atrophy. Gastaut (1976) has summarised the CT scan findings in 1762 epileptic patients of all ages combined from seven research groups. Over all proportion of abnormalities varied from
34-51% with a mean of 46%. Amongst these lesions 56% were atrophic in character. Tumours were found in 3-11%. The relationship of CT abnormalities to various seizure types is well illustrated by the study of Gastaut and Gastaut (1976) in 401 patients. A clear difference emerges between the relatively low incidence 11% associated with primary generalised seizures and the much higher proportion 60-80% in relation to other seizure types. These authors also estimated that CT scan detects 20% more cerebral lesions than the combination of long established techniques (Skull X-ray, EEG, angiography etc). These observations have been extended by Yang et al (1979) who scanned 256 children up to age of 17 years with a mean age of 4 years. Abnormalities were seen in 33%. They were able to distinguish a low yield group (2.5 - 8%) with idiopathic generalised seizures. Simple partial seizures either solitary or recurrent, were more likely to be associated with structural disease (Yang et al, 1983; and Boodanoff, 1979). In study by Yang et al (1983) approximately half of the patients with simple partial seizures had positive scans and even a solitary focal seizure was likely to reflect a structural abnormality. The correlation between positive scans, simple partial seizures, focal neurologic signs and a focus on EEG were taken into account. If two or more features were present structural disease was detected in approximately half of the patients. Conversely when focal features were absent, the CT scan was abnormal in only 6% of the patients. Of the 52 patients with CT
abnormalities only 15 had lesions that were potentially treated with surgery and all these patients had one or more focal features. Thus fewer than 10% of the patients had their management influenced by CT.

**Radioactive Isotopes**

Such as technetium (Brain scan) are occasionally used for visualization of tumours, inflammatory masses, viral encephalitis, and some other vascular lesions. It is a simple non invasive but costlier procedure.

**Magnetic Resonance Imaging (MRI)**

Its use has permitted the visualization of cerebral lesions not evident on CT scan. The technique permits delineation of tissues without administration of contrast enhancing agents and because bone elicits no interference, it is particularly useful for visualizing structures at the brain - bone interface i.e. in the posterior cranial fossa.

**Positron Emission Tomography (PET)**

It is an experimental investigative technique available only in few centres in the world (not available in India). Although these studies show great promise in the biochemical analysis of brain functions, the cost of the instrumentation and the technology required to produce isotopes will restrict PET scanning to major medical centres.